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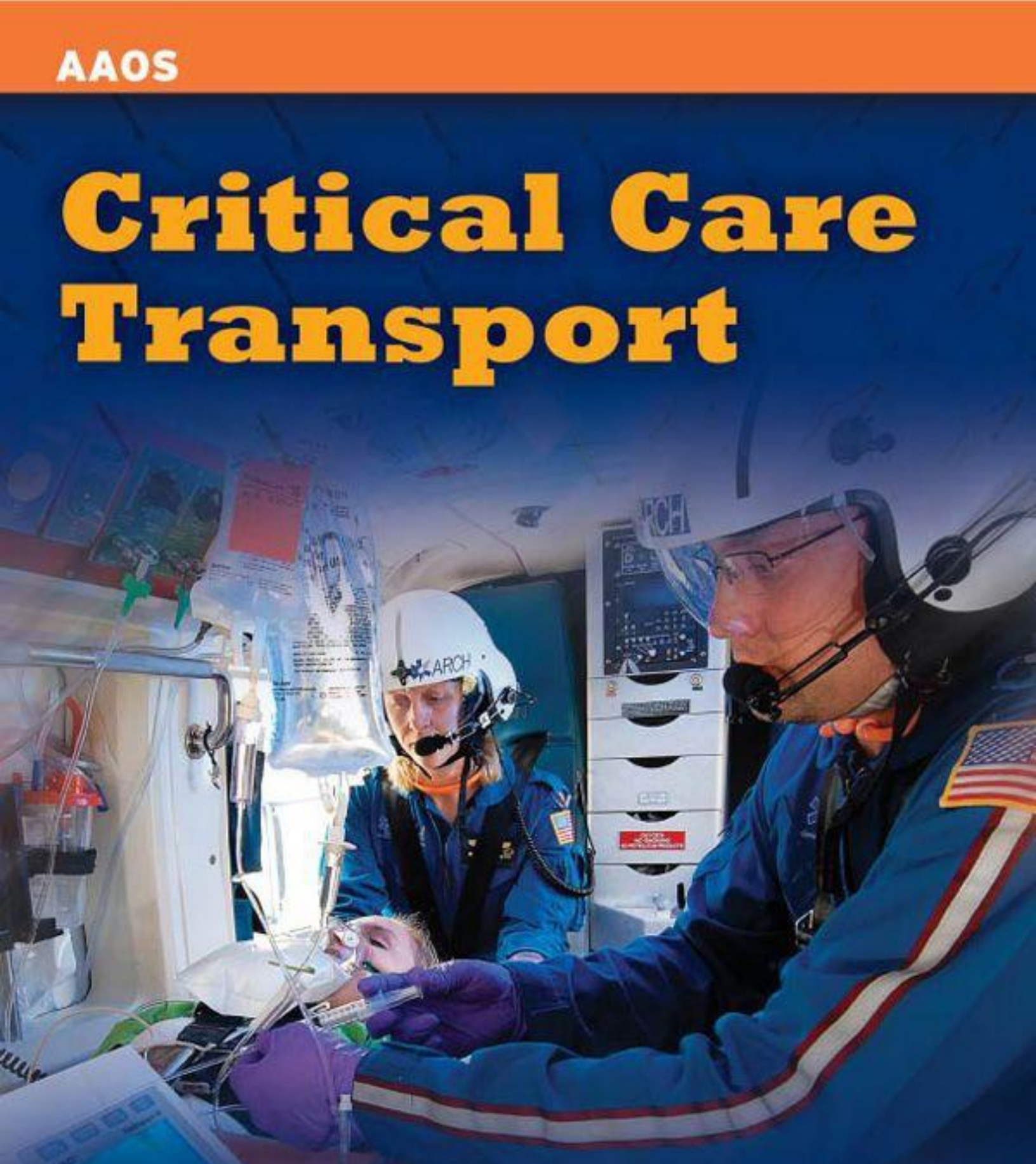
Critical Care Transport



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Critical Care Transport

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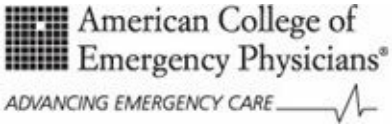

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The procedures and protocols in this book are based on the most current recommendations of responsible medical sources. The American Academy of Orthopaedic Surgeons; the American College of Emergency Physicians; University of Maryland, Baltimore County; and the publisher, however, make no guarantee as to, and assume no responsibility for, the correctness, sufficiency, or completeness of such information or recommendations. Other or additional safety measures may be required under particular circumstances.

This textbook is intended solely as a guide to the appropriate procedures to be employed when rendering emergency care to the sick and injured. It is not intended as a statement of the standards of care required in any particular situation, because circumstances and the patient‘s physical condition can vary widely from one emergency to another. Nor is it intended that this textbook shall in any way advise emergency personnel concerning legal authority to perform the activities or procedures discussed. Such local determination should be made only with the aid of legal counsel.

The patients and providers described in the case studies throughout this textbook are fictitious.

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Brief Contents

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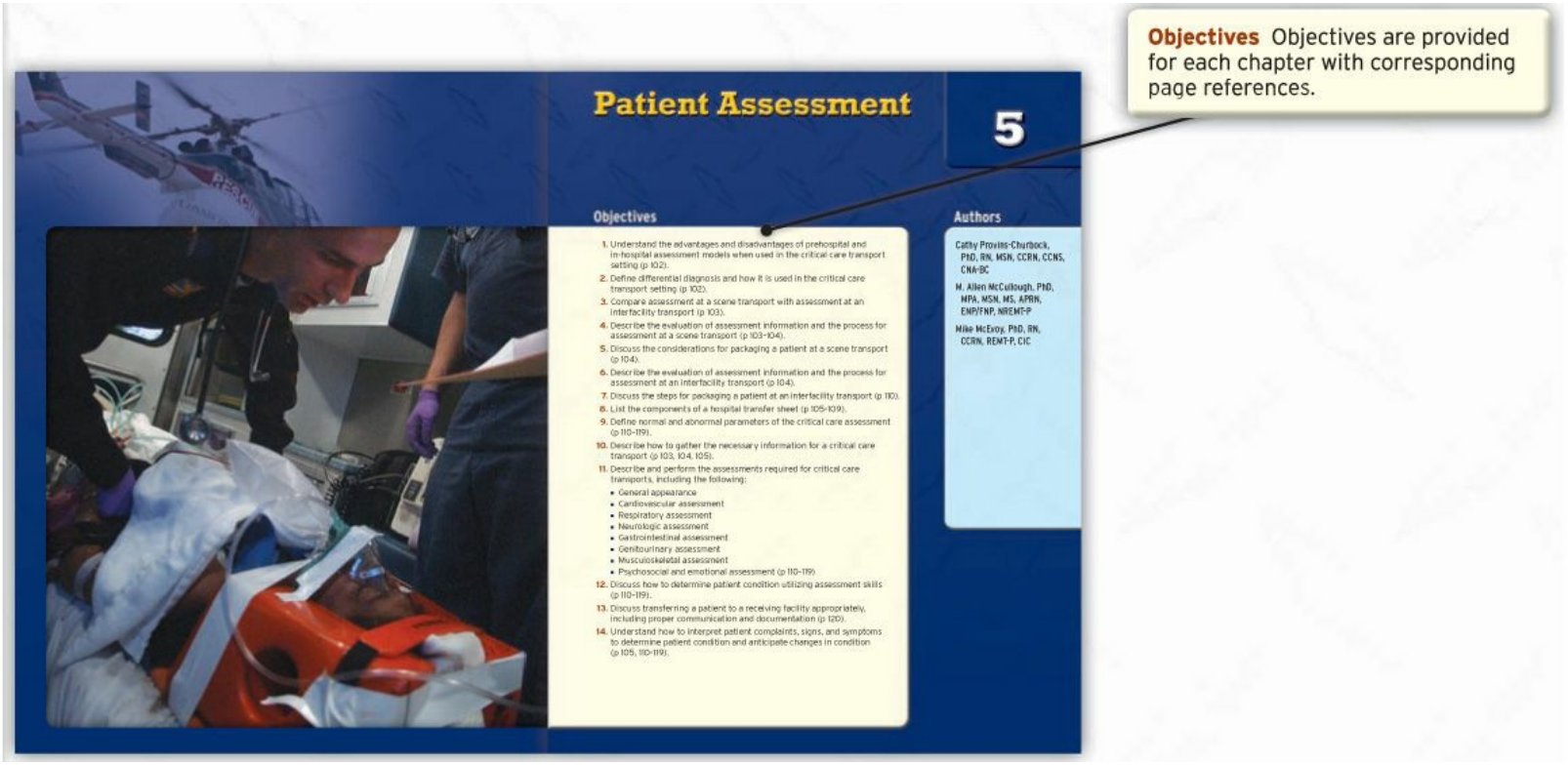
Resource Preview

Welcome to the new gold standard in critical care transport training. Published in conjunction with the American Academy of Orthopaedic Surgeons (AAOS) and the American College of Emergency Physicians (ACEP), *Critical Care Transport* offers cutting-edge content relevant to any health care provider training in critical care transport.

This book meets the curricula of major critical care training programs, including University of Maryland, Baltimore County (UMBC). It covers both ground and flight transport, and meets the objectives of critical care transport certification exams such as the Certified Flight Paramedic (FP-C) exam administered by the Board for Critical Care Transport Paramedic Certification.

Chapter Resources

A multitude of dynamic features have been incorporated to enhance learning of this advanced subject matter. The following pages show the features that help students learn—from skill drills to in-depth case studies to summary boxes.



CHAPTER 10 Trauma 241

possibility of unstable cervical spine and cord injury must always be considered. For scene calls, selective spinal immobilization criteria have become standard. If a patient has a cervical collar in place at a sending facility and the sending physician is unwilling to clear the cervical spine, the CCTP must consider full reimmobilization prior to transport. Patients with neck injury should always be considered difficult airway patients. Securing the airway with endotracheal intubation should be considered prior to most transports. Deterioration of the patient's airway in the back of a transport vehicle with limited space and personnel can be disastrous. Special airway considerations in the presence of neck injury include bleeding into the airway field, expanding hematomas, and tracheal disruption.

For neck trauma resulting in hoarseness accompanied by skin lacerations, ecchymosis, tenderness, subcutaneous emphysema, and/or stridor, an immediate otolaryngology consult is recommended. This may be the reason for transport to a larger hospital. Humidified oxygen, inhaled corticosteroids, and/or nebulized epinephrine may be ordered. Wounds may need to be dressed with occlusive dressings. If the wound is to the lower neck or clavicular area, vascular access may need to be achieved in the lower extremity as the result of compromised drainage from the arms.

Laryngotracheal Injuries

Laryngotracheal injuries represent less than 1% of all traumatic injuries. Most are in the area of the cervical trachea. Direct blunt trauma is the most common. Examples include a steering wheel to the extended throat, hanging, strangulation, and "clothesline" injury during sports such as mountain biking or snowmobiling. Penetrating laryngotracheal injuries are 10% of all penetrating neck injuries. Bubbling from a neck wound and subcutaneous air, along with dysphonia, dyspnea, stridor, visible wounds, and swelling, are all signs of laryngotracheal injury. These injuries can appear stable for a time, followed by rapid and catastrophic deterioration.

The most severe cases are laryngotracheal disruptions. It is vital that paralytics and muscle relaxants not be used. The only support for the trachea may be the supporting musculature. If the patient is paralyzed, the trachea may retract into the chest, making it impossible to ventilate the patient. If the trachea is visible through the neck wall, the endotracheal tube should simply be passed through the neck into the trachea.

Signs and Symptoms

Laryngotracheal Injury

- Bubbling from a neck wound
- Subcutaneous air
- Dysphonia
- Dyspnea
- Stridor
- Visible wounds
- Swelling

Transport Management

Laryngotracheal Injury

- Airway management with careful endotracheal intubation
- Occlusive dressing for open neck wounds
- Spinal precautions
- Transport to a trauma center

Thyroid Injuries

The thyroid gland is very vascular, and direct trauma to the front of the neck can cause hematomas of sufficient size to impinge on the airway. If surgical airway management is required, the location may need to be inferior to the usual site and inferior to the thyroid gland.

There are multiple case reports of thyrotoxicosis (thyroid storm, an excess of thyroid hormones) after trauma, including strangulation and direct blows to the anterior neck, shock (hypotension), or surgical procedures involving the thyroid. Thyroid storm has a 20% to 30% mortality rate. Thyroid storm causes a hypermetabolic crisis, including tachycardia over 140 beats/min, hyperthermia (sometimes with a temperature >103.9°F), coma with agitation, nausea, vomiting, diarrhea, and unexplained jaundice and pulmonary edema. Definitive diagnosis usually includes an elevated thyroxine level (normal level, 5.1 to 12.5 µg/dL). Treatment may include a beta-blocker such as an esmolol drip, sedation, and cooling for transport.

Signs and Symptoms

Thyrotoxicosis (Thyroid Storm)

- Tachycardia greater than 140 beats/min
- Hyperthermia (sometimes a temperature >103.9°F)
- Coma with agitation
- Nausea
- Vomiting
- Diarrhea
- Unexplained jaundice and pulmonary edema
- Elevated thyroxine level

Differential Diagnosis

Thyrotoxicosis (Thyroid Storm)

- Head injury
- Sepsis
- Toxic ingestion

Transport Management

Thyrotoxicosis (Thyroid Storm)

- Administer a beta-blocker (eg, an esmolol drip).
- Administer a sedative.
- Implement cooling measures.

Vascular Neck Injuries

Injury to the carotid, subclavian, and vertebral arteries and external and internal jugular veins can produce rapid exsanguination, hematoma formation, or embolization of air. Vascular neck injury accounts for one fourth of all penetrating

Signs and Symptoms Summarize signs and symptoms of conditions discussed in the chapter.

Differential Diagnosis List potential differential diagnoses of conditions discussed in the chapter.

Transport Management Summarize the transport-specific management of conditions discussed in the chapter.

Special Populations Discuss the specific needs and transport considerations of special populations, including pediatric patients, geriatric patients, and special needs patients.

1) nautical mile = 1.5 miles). For this reason, many services choose to send patients by fixed-wing aircraft, if available, for flights exceeding 100 nautical miles. Some air medical helicopter programs do not charge for their services and are instead funded by taxes or donations. Other air medical services offer a membership program that allows people to pay an annual subscriber fee for their household; this fee covers the cost of any air medical transport during that period.

Special Populations

Any high-risk complication of pregnancy, childbirth, or miscarriage qualifies as criteria for air medical transport.

Determining Air Medical Transport Suitability

When you are considering whether to use air medical transport, the requesting EMS provider or physician must avoid three critical errors:

- Requesting air medical transport for a patient when a more suitable means of transport is clearly indicated
- Requesting air medical transport when the patient could arrive at the receiving facility more quickly by ground transport
- Requesting air medical transport for a patient when cardiac arrest is ongoing or imminent

In most situations, air medical transport compares unfavorably to ground transport on the basis of safety, cost, and availability. On average, air medical transport costs at least four to five times more than ground transport over the same distance. In terms of availability, one air medical aircraft may serve a population of 500,000, whereas 20 or more ambulances may be available to serve the same population. Given this disparity, providers who are requesting air medical transport need to determine whether they will be needlessly tying up resources that could be better utilized elsewhere.

One other consideration to take into account when determining ground vs air transportation is the level of care that can be given. In some states, the highest level of ground provider is a paramedic. When considering flight programs, registered nurses are usually the highest level of provider, thus allowing the higher level of care such as balloon pumps, certain critical medications, and IV infusions of nonparamedic approved medications.

Thirty nautical miles (34.5 miles) or 30-minute transport is considered to be the minimum for which air medical transport is quicker than ground transport. (The industry-wide average for miles transported per flight is 61 miles.) However, factors such as traffic, construction, and weather have to be considered when making this determination. Some patients who require extended extrication time or have limited accessibility may benefit from air medical transport. Other patients may benefit from the advanced caregiving skills of the flight team, including their ability to perform rapid-sequence intubation, place chest tubes, or administer blood.

Other Justifications for Air Transport

A helicopter may sometimes be requested because the ground EMS unit is overwhelmed by the severity of the patient's injuries and needs the expertise of the flight crew. Air medical programs are frequently called to scenes where the ground EMS units have waited on the helicopter to perform advanced procedures such as rapid-sequence intubation. This situation is not strictly limited to ground EMS units, but is frequently observed in smaller hospitals as well. The medical flight team from the helicopter is often well respected in the medical community and is viewed as a "safety net" that can be relied upon when the provider on the ground requests assistance. As a member of the flight crew, you should be fast, proficient, and skilled in your area of expertise. By being proficient and skilled, you can ensure that your team is maintaining a limited on-scene time, which helps reduce the overall flight time to higher levels of care.

Transport of Patients in Cardiac Arrest

It seems counterintuitive to not utilize air medical transport for a patient who is in cardiac arrest or on the precipice of arrest. Cardiac arrest represents one of the most serious medical situations and would, in theory, stand to benefit the most by air medical transport. Nevertheless, most air medical providers do not routinely transport patients in active cardiac arrest for the following reasons.

First, it is against federal air regulations for passengers or crew onboard an aircraft to not be restrained in seatbelts during takeoffs and landings or as requested by the pilot. Providing effective CPR in the aircraft often requires the medical crew to be unbuckled.

Second, CPR is often futile, with fewer than 5% to 8% of all patients who receive CPR being resuscitated. Use of air medical transport for a patient in cardiac arrest is, therefore, generally viewed as a poor use of resources.

Last, to provide effective resuscitation, CPR generally requires a minimum of three providers: one provider performing chest compressions, one managing the airway, and one providing pharmacologic therapy. Rotor-wing aircraft typically carry only two medical providers, thus limiting the effectiveness of resuscitation. Although a patient in cardiac arrest should not be placed in the aircraft and flown, the CCTP must realize that occasionally a patient may arrest in flight and should be prepared to deal with this situation.

Triage of Air Medical Transport Patients

Intuitively, the use of air medical transport makes sense. It expedites transport of critical patients to appropriate facilities. However, numerous studies have concluded that air medical transport tends to be overused. This section discusses considerations when triaging critical care patients.

Overtriage in the Field

A retrospective review of pediatric trauma patients transported by helicopter reveals a trend toward overtriage, in which a patient is considered to be in more serious condition than he or she actually is. For example, a study in central Ohio found that nearly 70% of pediatric trauma patients transported by air were discharged from the emergency department. Similarly, a study at Children's National Medical Center in Washington, DC, found that 85% of pediatric trauma patients sent

Vocabulary Terms are easily identified and defined within the text. A comprehensive vocabulary list follows each chapter.

to the hospital by air were actually overtriaged and did not require air medical transport. A study that reviewed pediatric trauma patients flown to hospitals in Los Angeles, CA, found that 33% were overtriaged and were discharged home from the emergency department. However, many of these cases involved appropriate use of air transport: 37% of these patients had multiple injuries, 14% were intubated in the emergency department, 18% were admitted to the intensive care unit, and 4% were taken directly to the operating room. To curtail the trend toward overtriage, some are advocating for better triage training for prehospital personnel.

An estimated 10% or lower of all patients (pediatric and adult) sent to hospitals require multiple interventions. By contrast, more than 90% of the patients transported by rotor-wing or fixed-wing aircraft require multiple interventions. The patient acuteness (ie, the seriousness of the patient's condition) is much higher in the air medical industry because of the triage by experienced providers prior to the initiation of air medical transport.

Undertriage in the Field

Although many believe air medical transport is overused, there is some benefit to overtriage. **Undertriage** in the field, in which a patient is considered to be in less serious condition than he or she actually is, is a problem as well.

Some trauma physicians state that an overtriage rate of 25% is acceptable to ensure that those patients who most desperately need care are not overlooked. A recent literature review, however, indicates that this number can be as low as 10% and still encompass most outliers. Most trauma surgeons agree that a discharge rate from the emergency department of less than 10% is an indication of undertriage and suggests that patients who would most benefit from quick transport and trauma team activation are not being appropriately triaged.

Air Medical Use Criteria

Table 3-2 lists guidelines for determining whether use of air medical transport from one facility to another is appropriate. When you are making this determination, it is important to identify those patients who will not benefit from air medical transport. Table 3-3 identifies some of the absolute and relative contraindications to air medical transport.

Current **helicopter emergency medical service (HEMS)** criteria use both mechanism of injury and physiologic parameters to determine which patients might benefit most from transport in helicopters. Strictly basing the need for air medical transport on the mechanism of injury is no longer a clinically accepted practice because it has resulted in needless trauma team activations and emergent transports of stable patients. In contrast, new models that use both mechanism of injury and physiologic parameters to triage patients have been found to be effective at including appropriate patients and excluding patients who do not require trauma activation.

HEMS providers should have frequent discussions with the local trauma team about which criteria are used to triage patients. In addition, a formal system to track the "time to discharge" on all patients flown should be implemented. Air medical services whose overtriage rates exceed accepted limits should create opportunities to educate local providers about triage criteria.

Controversies

Controversy exists concerning specialty referral centers refusing transfers from outlying emergency departments unless their particular air medical service is used—even when ground transport would be perfectly acceptable. This practice needs to be addressed in the industry.

TABLE 3-3 Patient Characteristics That Warrant Air Medical Transport

Burns ≥ 10% total body surface area (second degree or greater)
Burns resulting from an explosion with severe respiratory distress or altered level of consciousness (patient not alert)
Burns with sustained unconsciousness
CO inhalation or HazMat exposures with sustained unconsciousness
Postresuscitation from near drowning
Diving accident with suspected neck injury
SCUBA accidents
Electrocution or lightning strike with sustained unconsciousness
Long fall (from heights exceeding 6') with sustained unconsciousness
Stab, gunshot wound, or other penetrating trauma to the torso or head with sustained unconsciousness
Childbirth or miscarriage with high-risk obstetric complication: • Viable premature infant (≤ 20 weeks' gestation) • Viable multiple births (≤ 20 weeks' gestation) • Maternal bleeding disorder • Maternal use of blood thinners
Traffic or transportation crashes with patient pinned and sustained unconsciousness
Traffic or transportation crashes with sustained unconsciousness and high mechanism: • All-terrain vehicles • Automobile vs bicycle or motorcycle • Ejection • Personal watercraft • Rollover crash • Vehicle off bridge/height
Need for advanced life support or specialized care not available by ground services
Time-critical illness or injury: • Stroke or acute coronary syndrome with potential for interventional care • Amputated limb with potential for reattachment

Controversies

Organ donation is a major concern in a critical care unit, but should not be relevant to critical care transport. CCTPs care for critically ill patients as though they are expected to survive. The patient's status as an organ donor should have no impact on care.

Controversies Highlight issues that may be under debate in the critical care community.

Skill Drills Provide written step-by-step explanations and visual summaries of important skills and procedures.

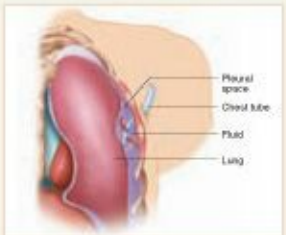


Figure 10-21 A chest tube is inserted through the side of the chest into the pleural space.

- Kelly clamps (curved and straight)
- Sterile occlusive dressing
- Suturing material
- 2% Chlorhexidine
- A local anesthetic if the patient is awake
- A fluid collection device with a one-way valve that allows air, fluid, or pus to flow out of the chest. If not available, an indwelling catheter bag with a one-way valve may be used
- Tape
- Mechanical suction device (not always used)

Indications include:

- Pneumothorax
- Hemopneumothorax
- Empyema

Complications include:

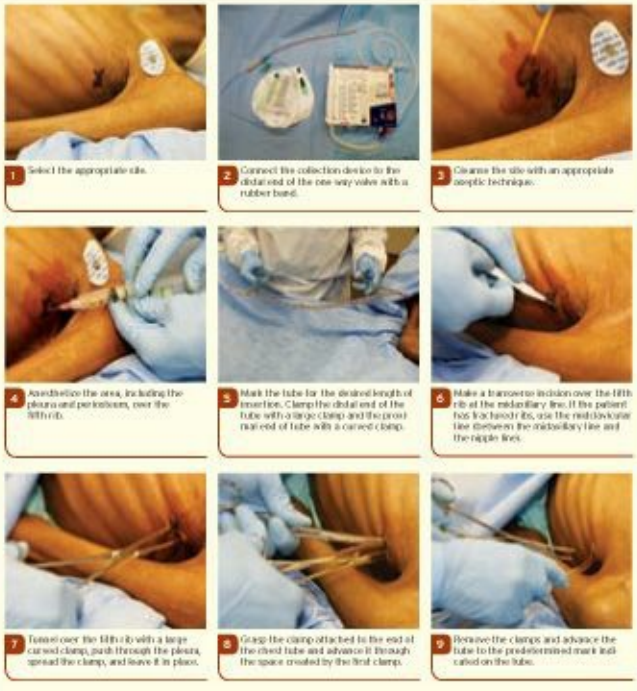
- Recurrent pneumothorax
- Accidental removal
- Broken collection chamber
- Parenchymal injury
- Laceration of intercostal vessels
- Creation of a hemothorax or bleeding
- Misplacement below the diaphragm
- Infection

Skill Drill 10-2 shows the clinical procedure for inserting a chest tube, which is described below:

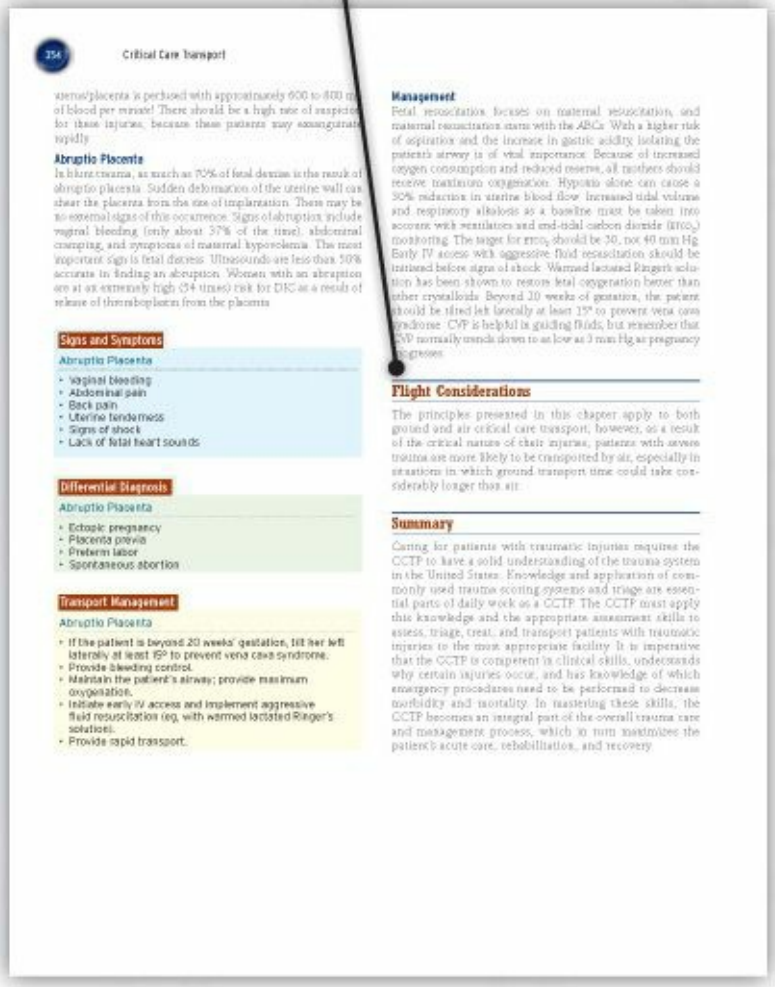
1. Select the appropriate site: midaxillary over the fifth rib (Step 1).
2. Connect the indwelling catheter bag or the collection device to the distal end of the one-way valve with a rubber band (Step 2). Be certain that the arrow on the valve is facing away from the patient.
3. Cleanse the site with an appropriate aseptic technique (Step 3).
4. Anesthetize the area, including the pleura and periosteum, over the fifth rib (Step 4), if the patient is conscious and time permits. If possible, place the patient in a 30° reverse Trendelenburg (head up) position so blood drains to the low end of the lung.
5. Mark the tube for the desired length of insertion (Step 5).
6. Clamp the distal end of the tube with a large clamp (such as a Kelly clamp) and the proximal end of the tube with a curved clamp.
7. Make a transverse incision over the fifth rib in the midaxillary line (Step 6). If the patient has fractured ribs, use the midclavicular line (between the midaxillary line and the nipple line).
8. Tunnel over the fifth rib with a large curved clamp, push through the pleura, spread the clamp, and leave it in place (Step 7).
9. Grasp the clamp attached to the end of the chest tube and advance it through the space created by the first clamp, directing it posteriorly and downward toward the diaphragm (Step 8).
10. Remove the clamps and advance the tube to the predetermined mark indicated on the tube, or least 5 cm beyond the last hole (Step 9).
11. Connect the collection device (Step 10).
12. Remove the distal clamp (Step 11).
13. Suture the tube in place and close the wound (Step 12).
14. Note the depth of the tube at the skin (marked on the tube) and mark with a felt-tip pen.
15. Cover the insertion site with an occlusive dressing and reinforce all connections with tape (Step 13).
16. Upon arrival at the receiving facility, obtain a chest radiograph to confirm placement.
17. Document the procedure, including the size of the chest tube inserted, the amount of return of air/fluid after insertion of the tube, and any changes in patient condition (including oxygen saturation).

Skill Drill 10-2

Chest Tube Insertion



Flight Considerations Summarize flight considerations for emergencies discussed in the chapter.



uterus/placenta is perfused with approximately 600 to 800 mL of blood per minute! There should be a high rate of suspicion for these injuries, because these patients may exsanguinate rapidly.

Abruptio Placenta

In blunt trauma, as much as 70% of fetal demise is the result of abruptio placenta. Sudden deformation of the uterine wall can shear the placenta from the site of implantation. There may be no external signs of this occurrence. Signs of abruption include vaginal bleeding (only about 37% of the time), abdominal cramping, and symptoms of maternal hypovolemia. The most important sign is fetal distress. Ultrasounds are less than 50% accurate in finding an abruption. Women with an abruption are at an extremely high (54 times) risk for DIC as a result of release of thromboplastin from the placenta.

Signs and Symptoms

Abruptio Placenta

- Vaginal bleeding
- Abdominal pain
- Back pain
- Uterine tenderness
- Signs of shock
- Lack of fetal heart sounds

Differential Diagnosis

Abruptio Placenta

- Ectopic pregnancy
- Placenta previa
- Preterm labor
- Spontaneous abortion

Transport Management

Abruptio Placenta

- If the patient is beyond 20 weeks' gestation, tilt her left laterally at least 15° to prevent vena cava syndrome.
- Provide bleeding control.
- Maintain the patient's airway; provide maximum oxygenation.
- Initiate early IV access and implement aggressive fluid resuscitation (eg, with warmed lactated Ringer's solution).
- Provide rapid transport.

Management

Fetal resuscitation focuses on maternal resuscitation, and maternal resuscitation starts with the ABCs. With a higher risk of aspiration and the increase in gastric acidity isolating the patient's airway is of vital importance. Because of increased oxygen consumption and reduced reserve, all mothers should receive maximum oxygenation. Hypoxia alone can cause a 30% reduction in uterine blood flow. Increased tidal volume and respiratory alkalosis as a baseline must be taken into account with ventilators and end-tidal carbon dioxide ($ETCO_2$) monitoring. The target for $ETCO_2$ should be 30, not 40 mm Hg. Early IV access with aggressive fluid resuscitation should be initiated before signs of shock. Warmed lactated Ringer's solution has been shown to restore fetal oxygenation better than other crystalloids. Beyond 20 weeks of gestation, the patient should be tilted left laterally at least 15° to prevent vena cava syndrome. CVP is helpful in guiding fluids, but remember that CVP normally trends down to as low as 3 mm Hg as pregnancy progresses.

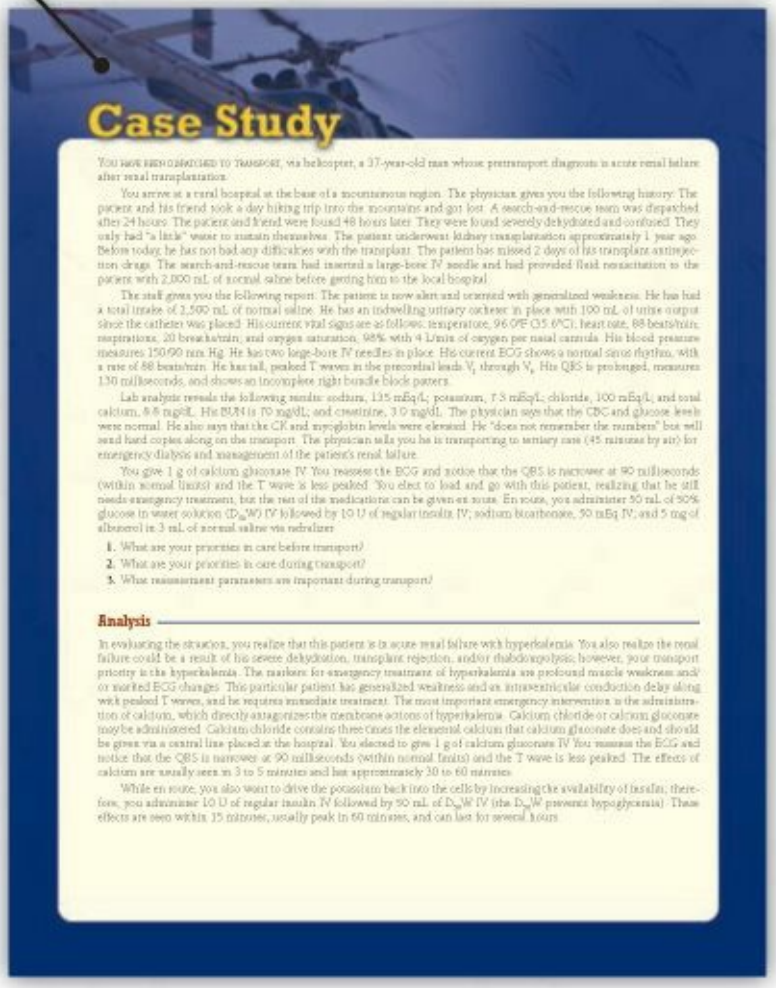
Flight Considerations

The principles presented in this chapter apply to both ground and air critical care transport; however, as a result of the critical nature of their injuries, patients with severe trauma are more likely to be transported by air, especially in situations in which ground transport time could take considerably longer than air.

Summary

Caring for patients with traumatic injuries requires the CCTP to have a solid understanding of the trauma system in the United States. Knowledge and application of commonly used trauma scoring systems and triage are essential parts of daily work as a CCTP. The CCTP must apply this knowledge and the appropriate assessment skills to assess, triage, treat, and transport patients with traumatic injuries to the most appropriate facility. It is imperative that the CCTP is competent in clinical skills, understands why certain injuries occur, and has knowledge of which emergency procedures need to be performed to decrease morbidity and mortality. In mastering these skills, the CCTP becomes an integral part of the overall trauma care and management process, which in turn maximizes the patient's acute care, rehabilitation, and recovery.

Case Study An in-depth, comprehensive scenario at the end of each chapter allows students to synthesize and apply what they've learned. Concludes with an analysis that discusses learning points.



Case Study

YOU HAVE BEEN DISPATCHED TO TRANSPORT, via helicopter, a 37-year-old man whose pretransport diagnosis is acute renal failure after renal transplantation.

You arrive at a rural hospital at the base of a mountainous region. The physician gives you the following history: The patient and his friend took a day hiking trip into the mountains and got lost. A search-and-rescue team was dispatched after 24 hours. The patient and friend were found 48 hours later. They were found severely dehydrated and confused. They only had "a little" water to sustain themselves. The patient underwent kidney transplantation approximately 1 year ago. Before today, he has not had any difficulties with the transplant. The patient has missed 2 days of his transplant antirejection drugs. The search-and-rescue team had inserted a large-bore IV needle and had provided fluid resuscitation to the patient with 2,000 mL of normal saline before getting him to the local hospital.

The staff gives you the following report. The patient is now alert and oriented with generalized weakness. He has had a total intake of 2,500 mL of normal saline. He has an indwelling urinary catheter in place with 100 mL of urine output since the catheter was placed. His current vital signs are as follows: temperature, 96.0°F (35.6°C); heart rate, 88 beats/min; respirations, 20 breaths/min; and oxygen saturation, 98% with 4 L/min of oxygen per nasal cannula. His blood pressure measures 150/90 mm Hg. He has two large-bore IV needles in place. His current ECG shows a normal sinus rhythm, with a rate of 88 beats/min. He has tall, peaked T waves in the precordial leads V_2 through V_4. His QRS is prolonged, measures 130 milliseconds, and shows an incomplete right bundle block pattern.

Lab analysis reveals the following results: sodium, 135 mEq/L; potassium, 7.5 mEq/L; chloride, 100 mEq/L; and total calcium, 8.8 mg/dL. His BUN is 70 mg/dL; and creatinine, 3.0 mg/dL. The physician says that the CBC and glucose levels were normal. He also says that the CK and myoglobin levels were elevated. He "does not remember the numbers" but will send hard copies along on the transport. The physician tells you he is transporting to tertiary care (45 minutes by air) for emergency dialysis and management of the patient's renal failure.

You give 1 g of calcium gluconate IV. You reassess the ECG and notice that the QRS is narrower at 90 milliseconds (within normal limits) and the T wave is less peaked. You elect to load and go with this patient, realizing that he still needs emergency treatment, but the rest of the medications can be given en route. En route, you administer 50 mL of 50% glucose in water solution ($D_{50}W$) IV followed by 10 U of regular insulin IV; sodium bicarbonate, 50 mEq IV; and 5 mg of albuterol in 3 mL of normal saline via nebulizer.

1. What are your priorities in care before transport?
2. What are your priorities in care during transport?
3. What reassessment parameters are important during transport?

Analysis

In evaluating the situation, you realize that this patient is in acute renal failure with hyperkalemia. You also realize the renal failure could be a result of his severe dehydration, transplant rejection, and/or rhabdomyolysis; however, your transport priority is the hyperkalemia. The markers for emergency treatment of hyperkalemia are profound muscle weakness and/or marked ECG changes. This particular patient has generalized weakness and an intraventricular conduction delay along with peaked T waves, and he requires immediate treatment. The most important emergency intervention is the administration of calcium, which directly antagonizes the membrane actions of hyperkalemia. Calcium chloride or calcium gluconate may be administered. Calcium chloride contains three times the elemental calcium that calcium gluconate does and should be given via a central line placed at the hospital. You elected to give 1 g of calcium gluconate IV. You reassess the ECG and notice that the QRS is narrower at 90 milliseconds (within normal limits) and the T wave is less peaked. The effects of calcium are usually seen in 3 to 5 minutes and last approximately 30 to 60 minutes.

While en route, you also want to drive the potassium back into the cells by increasing the availability of insulin; therefore, you administer 10 U of regular insulin IV followed by 50 mL of $D_{50}W$ IV (the $D_{50}W$ prevents hypoglycemia). These effects are seen within 15 minutes, usually peak in 60 minutes, and can last for several hours.

Ready for Review Summarize chapter content in a comprehensive bulleted list.

Prep Kit

Ready for Review

- All cells require energy a continuous supply of oxygen, and nutrients. From those nutrients, mitochondria manufacture energy in the form of adenosine triphosphate (ATP). This process is called cellular respiration.
- Cellular respiration has three parts: glycolysis, the citric acid cycle, and the electron transport chain (oxidative phosphorylation). Unlike the other two parts of cellular respiration, which require oxygen, glycolysis also can occur in an anaerobic environment. But this solo attempt to support cellular respiration causes a reduction in ATP and an increase in lactic acid, which can result in cell damage, impaired function of tissue, and a cascade of injurious effects.
- To get oxygen to the body's cells, the respiratory system must allow oxygen and carbon dioxide exchange across the alveolar-capillary membrane. Once this transfer occurs, arterial blood transports oxygen to the tissues (the oxygen is dissolved in plasma [3%] or is bound to hemoglobin [97%]).
- Various factors can affect the delivery of oxygen-rich blood to the tissues, including blood volume, viscosity and arterial elasticity. However, the primary determinants of transport are blood pressure (it must be stable) and cardiac output (it must be adequate).
- Under normal conditions, only 25% of the oxygen delivered to the tissues of the body is actually extracted. This allows the body a buffer zone for periods of low oxygen delivery, during which the cells must extract more oxygen. Therefore, management of shock is geared toward maintaining oxygen consumption by maintaining adequate oxygen supply, providing hemoglobin through administration of blood products, and optimizing cardiac output. If there is a breakdown in one or more of these components, homeostasis is lost.
- Shock is a progressive, whole-body response to an inadequate supply of oxygen within cells, tissues, and organs, from one or multiple causes. It is commonly classified in four stages, and the mortality rate increases with progression.
  - During initial or early shock, blood flow into the microcirculatory beds decreases and lactic acid levels begin to rise. However, compensatory mechanisms, such as an increase in oxygen consumption, are able to keep vital signs at baseline.
  - During the compensatory stage of shock, neural, hormonal, and chemical mechanisms attempt to compensate for the now severely reduced delivery of oxygen. They, in effect, cover up low circulatory volumes, poor stroke volume, and falling cardiac output, especially in children and healthy young adults. Common signs and symptoms are as subtle as mild tachypnea and tachycardia. The MAP may decrease 10 to 15 mm Hg from baseline. Cardiac output also may drop slightly.
  - During the progressive or decompensating stage of shock, multifaceted systemic responses to cell breakdown occur, affecting nearly every organ of the body. Compensatory mechanisms have begun to fail. Patients have more pronounced signs of shock, including altered mental status, tachycardia, and a drop in MAP of more than 30 mm Hg from baseline. Laboratory analysis shows a progression of acidosis, hyperkalemia, and climbing lactate levels. This is a life-threatening emergency that requires immediate treatment.
  - During the refractory or irreversible stage of shock, compensatory mechanisms have failed. At this stage, the patient is unresponsive to verbal stimuli. Blood pressure will be inadequate and the heart rate will be increased. The respiratory rate is increased and respirations are shallow. Skin will be cold, cyanotic, and/or mottled and peripheral pulses are weak and thready to absent. Urinary output will decrease and bowel sounds will be absent. Anaerobic metabolism progresses to permanent organ dysfunction and treatment no longer can reverse the main effects. Multiple organ failure ensues and the risk of mortality is at its highest.
- MODS is diagnosed when two or more organs stop functioning. The organs affected early in MODS are the brain, kidneys, liver, adrenal glands, and heart. Sepsis is the leading cause of MODS.
- Shock traditionally is classified by cause. More than one type of shock may be present at the same time.
- Cardiogenic shock results when the ventricles are unable to pump blood forward, ultimately decreasing cardiac output. Manifestations vary depending on the underlying cause, but common signs and symptoms are low blood pressure; altered mental status; cool, pale, diaphoretic skin; decreased urine output; weak and thready pulse; distant or abnormal S_1 or S_2 heart sounds; fluid accumulation in the lungs or limbs; and tachypnea. Management focuses on enhancing cardiac output while decreasing left ventricular work load.
- Hypovolemic shock is present when there is too little circulating blood volume within the vascular system, resulting in hypotension. Manifestations include tachycardia; hypotension; signs of poor tissue perfusion; altered mental status; and cold, mottled, and pulseless extremities. Management revolves around treating the underlying condition, administering oxygen, and initiating volume replacement.
- Distributive shock is a term used to describe several types of shock involving loss of vasomotor tone or increased vascular permeability: neurogenic shock, anaphylactic shock, and septic shock.
- Neurogenic shock results from conditions that impede the ability of the sympathetic nervous system to control the constriction and dilation of vessel walls, such as trauma to the brain or spinal cord. The signs and symptoms associated with neurogenic shock can be very different from those

Vital Vocabulary Provide key terms and definitions from the chapter.

- Hemolytic transfusion reactions are caused by ABO or Rh incompatibility, intradonor incompatibility, improper cross-matching, or improper blood storage.
- Immediate hemolytic transfusion reactions may occur during transfusion or within 3 to 7 days following transfusion. Red blood cells are destroyed, and hemoglobin and red blood cell remnants are released into the bloodstream. Symptoms include chest pain, facial flushing, shortness of breath, chills, fever, hypotension, flank pain, hemoglobinuria, oliguria, bloody oozing at the infusion site, burning along the vein receiving the blood, shock, signs of renal failure, and DIC. Treatment of immediate hemolytic transfusion reactions is directed toward prevention and supportive management.
- Delayed hemolytic transfusion reactions do not occur until 3 to 7 days after transfusion. Signs and symptoms are usually mild and include mild fever, chills, and moderate jaundice, with treatment focused on the prevention and the treatment of severe complications should they arise.
- Plasma protein incompatibility is caused by immunoglobulin A incompatibility. Clinical presentations include abdominal pain, diarrhea, shortness of breath, chills, fever, flushing, and hypotension. Management includes oxygen administration, fluids, epinephrine, and corticosteroids as indicated.
- Hypocalcemia may occur when blood products containing citrate are infused too quickly and bind calcium, causing calcium deficiency and leading to arrhythmias, hypotension, muscle cramping, nausea, vomiting, seizure activity, and/or a tingling sensation in the fingers. Slowing or stopping the transfusion may be indicated. Management considerations include the administration of calcium gluconate IV infused slowly.
- High potassium levels in stored plasma may result in potassium intoxication during blood administration. Clinical manifestations include diarrhea, intestinal colic, flaccidity, muscle twitching, oliguria, signs of renal failure, bradycardia, ECG changes with visible tall peaked T waves, and/or cardiac arrest. Management considerations include a diagnostic 12-lead ECG tracing; Kayexalate administration; administration of an albuterol nebulizer; administration of furosemide; and the infusion of 50% glucose, insulin, sodium bicarbonate, or calcium.
- Hypothermia may result from a rapid infusion of large amounts of cold blood products. Hypothermia symptoms include chills, shivering, hypotension, arrhythmias, bradycardia, and/or cardiac arrest. Treatment may include stopping the transfusion, warming the patient, obtaining a diagnostic 12-lead ECG, and warming the blood if the transfusion is restarted.

Vital Vocabulary

ABO-incompatible transfusion reactions A type of transfusion reaction in which the patient possesses antigens to a blood type and receives that blood type.

adaptive immune system The secondary mechanism that protects the host by reacting with and eliminating specific antigens after requiring more time than the innate immune system to mobilize its defense against unknown pathogens.

aerobic metabolism A form of energy production in which mitochondria utilize glucose, amino acids, and fatty acids combined with oxygen and ADP to produce ATP, carbon dioxide, water, and heat.

albumin A blood product containing the specific protein found in the blood, and prepared by the fractionation of pooled plasma; used for volume replacement in certain conditions.

anaerobic metabolism A less efficient form of energy production in which an alternative pathway converts glucose to pyruvic acid with the simultaneous production of ATP and that also results in the production of lactate.

anaphylactic shock A severe hypersensitivity reaction that involves bronchoconstriction and cardiovascular collapse.

anaphylactoid reaction A non-IgE-mediated response that causes the rupture of mast cells and basophils, which then release histamine and other defense mediators.

antigen Any substance that can create an immune response in the body.

arterial oxygen content (Ca_{O_2}) The total amount of oxygen in the arterial blood.

cardiac index A hemodynamic value that adjusts a patient's cardiac output to take into account his or her total body surface area.

cardiac output (CO) The amount of blood pumped by the heart per minute, calculated by multiplying the stroke volume by the heart rate per minute.

cardiogenic shock A condition caused by loss of 40% or more of the functioning myocardium; the heart is no longer able to circulate sufficient blood to maintain adequate oxygen delivery.

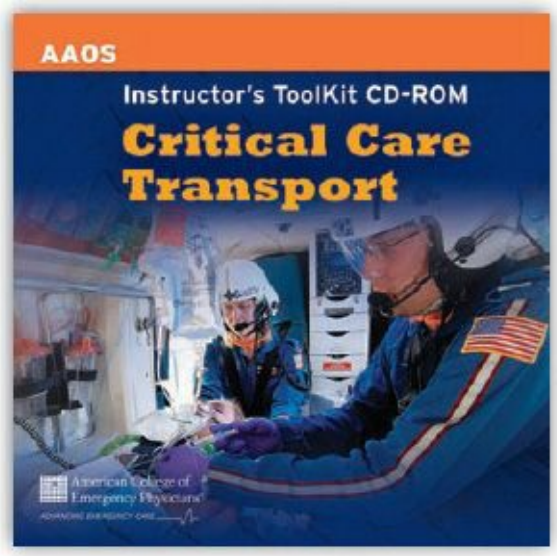
cryoprecipitate A blood product created from plasma and in which clotting factors, especially factor VIII, are concentrated; used to treat patients with coagulation disorders.

cytokines Chemical messengers that enhance cell growth, promote cell secretion, direct cellular traffic, stimulate macrophage function, and destroy antigens; interleukins are a type of cytokine.

delayed hemolytic transfusion reactions A type of transfusion reaction that does not occur until 3 to 7 days after transfusion.

disseminated intravascular coagulation (DIC) A complex condition arising from different causes that activate coagulation mechanisms, resulting in obstructed blood flow as a result

Instructor and Student Resources



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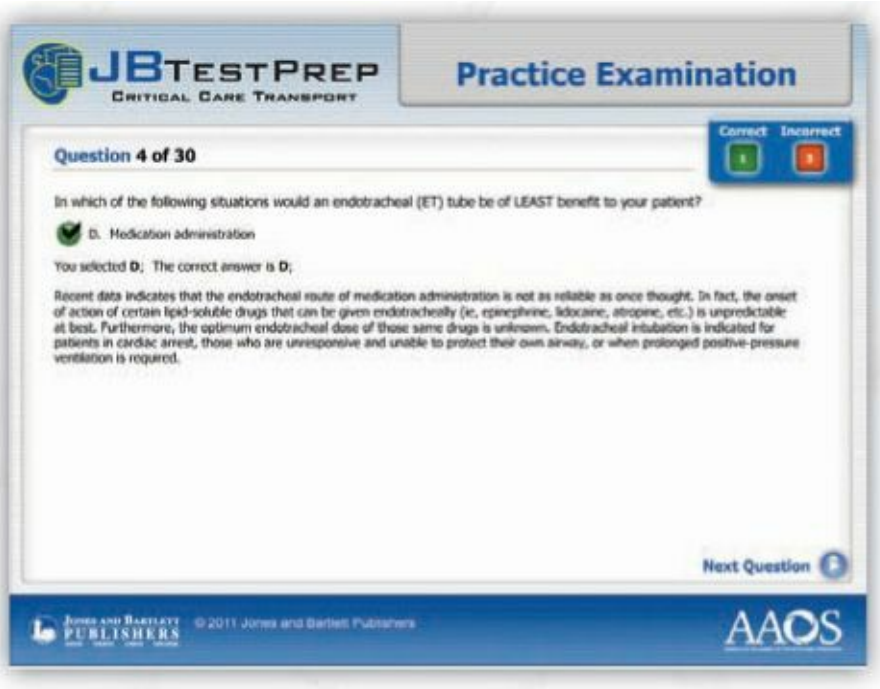

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Introduction to Critical Care Transport

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Objectives

1. Define the term *critical care* (p 4).
2. Understand the history of critical care transports, both air and ground (p 4).
3. Describe critical care transport team composition (p 8).
4. List the qualifications of members of a critical care transport team (p 8).
5. Identify the modes of transportation used during critical care transports, including mobile (ground) units, rotor-wing aircraft, and fixed-wing aircraft (p 4–7).
6. Discuss the differences between the modes of transportation (p 9, 10).
7. Understand the advantages and disadvantages for each mode of transportation (p 11).
8. Understand the role of dispatch in determining the mode of transportation (p 10).
9. Understand the role of medical control in the critical care transport environment (p 12).
10. Identify stresses specific to the critical care transport professional and their signs (p 13).
11. Discuss state and national standards for critical care transport (p 13).
12. Discuss reimbursement criteria and their relevance to critical care transport (p 13).
13. Understand the importance of interpersonal communications with the patient and family members (p 14).
14. Explain the process of quality assurance and improvement, and understand the importance of maintaining skills and knowledge (p 15).
15. Discuss the role of critical care transport professionals in emergency medical services (p 4).
16. Recognize the impact of specialty/critical care on the health care system (p 4).

Introduction

Critical care transport is at the forefront of the ever-changing world of modern health care. Driven by cost containment, insurers, clinical advancements, new regulations, and a continual shift toward specialty centers, the field of critical care transport will continue to grow exponentially. Transports once managed exclusively by physicians and nurses now include EMTs and paramedics. This expansion of EMS providers into critical care transport brings with it a pressing need for formal education and clinical preparation in the multifaceted and complex area of critical care transport. This education will aid all providers in bridging the gap from their respective educational foundations to the level of **critical care transport professional (CCTP)**.

Critical care is complex, detailed health care that is provided to patients experiencing acute, life-

threatening conditions. CCTPs must have the ability to deal with crucial situations rapidly and with precision using and interpreting data provided by various advanced machines and devices for treating and monitoring patients.

Methods for transporting the **critical care patient** have evolved from horse-drawn carts in the 17th century, to the use of hot-air balloons, to the use of ground, rotor-wing, and fixed-wing vehicles. With more efficient transport methods came rapid provision of medical treatment and increased the survivability of critical injuries and illnesses.

This chapter focuses on the evolution of critical care transports, the members of the health care community involved in patient care, their educational requirements, the various transportation modes used (including their differences, advantages, and disadvantages), the role of air medicine, and the effect of critical care transports on the health care system.

The History of Critical Care Transport

Ground Transport

The area of critical care transport is growing rapidly, and with it comes the challenge of initial and continued training for the staff manning these units. This training involves current and expanded treatment modalities, the use of specialized equipment, and the practice of advanced procedures. During these times of change, CCTPs must maintain the focus of their primary mission—providing care to critically ill and injured patients. Critical care requires the ability to deal with crucial situations rapidly and with precision. Until recently, this type of patient care was limited to intensive or critical care units within a hospital setting. Now, there is an emphasis on critical care treatment in the prehospital setting and during interfacility transports, as well as within the hospital itself.

Over the years, the need for a rapid transport system to move critically injured or ill patients to a definitive care facility has become clear. As will be discussed, early methods of transportation were initially developed out of necessity to reduce the incredibly high rates of **morbidity** and **mortality** among soldiers. Later, these modes of transportation would also be used to move critically ill and injured patients to more definitive care facilities. Because these modes of transportation vary widely, it is advantageous to examine the progression of each separately.

The first record of moving injured patients was in the 11th century during the Crusades by the Knights of St John. The Knights of St John treated and transported injured soldiers after learning about first aid procedures from Arab and Greek physicians. This was the birth of the first emergency medical providers. At the same time, any soldier who carried an injured comrade to receive medical treatment would receive a small monetary reward.

Ground transportation was first used in 1792 to evacuate injured soldiers during the French Revolution. A surgeon in the French army, Dominique Jean Larrey, noticed many of his countrymen lay wounded on the battlefield for as long as 36 hours until they were evacuated by fourgons, or multi-horse wagons **Figure 1-1**. This delay in transport resulted in the death and disability of many soldiers, which Larrey found unacceptable.

Larrey designed and produced several two- and four-wheeled carts called the *ambulance volante* (flying ambulance) **Figure 1-2**, which were fast and maneuverable. These carts were used to transport surgeons and equipment to the battlefield to give first aid and then return the wounded to nearby field hospitals. Larrey's flying ambulance was the first recorded use of an evacuation system to help improve the survival rates of soldiers. He is also credited with creating the first official army medical corps using stretchers, hand carts, and wagons as a means of transporting injured patients.

America's first ambulance service was instituted in 1862 during the Civil War at the Battle of Antietam, by surgeon Jonathan Letterman. Director of the Army of the Potomac, Letterman developed a

plan for an integrated medical treatment and evacuation system with its own vehicles, facilities, and personnel **Figure 1-3**. This system became known as the Ambulance Corps and was the basis of Army medical doctrine. On June 23, 1917, the US Army Ambulance Service was established as a descendent of the initial Ambulance Corps. During this time, the use of train ambulances, steamboat hospitals, and car or trolley ambulances was popular in some cities.

Figure 1-1 The first vehicles used for transport of casualties were horse-drawn.

Figure 1-2 The *ambulance volante* (flying ambulance), shown here, was a two- or four-wheeled cart that transported surgeons and equipment to the battlefield.

In 1869, America's first city ambulance service was established in New York City by Bellevue Hospital **Figure 1-4**. The hospital's Center Street Branch dispatched ambulances via telegraph and was staffed with physicians or surgeons from the hospital. Incredibly, the ambulance responded to more than 1,800 calls throughout the city of New York that first year. As the call volume increased, it became more difficult to staff the ambulances without decreasing the number of physicians in the hospital; in response to this problem, the hospital began placing orderlies or kitchen or janitorial staff on the ambulance. Because these employees had little or no medical training, responded without equipment, and frequently arrived at the scene hours after being dispatched, the mortality rate of seriously ill and injured patients increased dramatically. The continued population growth and the Industrial Revolution fueled the need for better training and easier access to rapid prehospital emergency medical care.

Figure 1-3 The first integrated treatment and evacuation system was established by surgeon Jonathan Letterman in 1862 during the Civil War at the Battle of Antietam.

Figure 1-4 One of New York City's early ambulances.

In 1899, the first motorized ambulance was developed in Chicago, IL, and donated to the Michael Reese Hospital by a group of businessmen. The unit weighed 1,600 pounds and could travel at speeds up to 16 miles per hour.

In the early 1900s, ambulance crews had evolved to include a driver and a person responsible for patient care, but generally the crews only picked up and delivered the patient to a base hospital.

The next milestone for prehospital care came in 1966 in Dublin, Ireland, with the provision of prehospital coronary care by physicians. Research by Francis Pantridge, MD, indicated that most patients experiencing treatable ventricular fibrillation had this fatal arrhythmia outside the hospital. His thought was to have portable, lightweight, battery-driven defibrillators carried as part of the rapid response system he had developed. His foresight was the driving force for the current use of defibrillators by paramedics on mobile coronary care units and automated external defibrillators (also known as AEDs) used by the lay public.

Prehospital defibrillation was not used in the United States until 1969, shortly after the nation's first paramedic program was instituted by the Miami, FL, Fire Department under the direction of Eugene Nagel, MD. Physicians in Los Angeles, CA; Seattle, WA; and Columbus, OH, followed Nagel's lead and began paramedic programs in their states in the hope that prehospital emergency care would reduce preventable deaths. Today, prehospital and interfacility transports are accomplished not only by ground, but also by air.

Air Transport

After witnessing balloon flight demonstrations by the Montgolfier brothers in 1784, physicians began to consider the benefits that flight could bring to their patients. However, it was not until 1870 that the first documented case of air medical transport took place during the Prussian Siege of Paris, when 160 wounded soldiers and civilians were transported from the battlefield by hot-air balloons. At the time, this method was considered a radical means of transport and was met with harsh criticism; however, improvements were made. During the period between 1890 and 1910, M. de Mooy, chief of the Dutch Medical Service, pursued the idea of using litters suspended from balloons to transport patients.

On December 17, 1903, Orville Wright climbed into the plane he and his brother built for the second attempt at flying a motorized plane. The site was Kill Devil Hills, NC, and the plane stayed airborne for 12 seconds and flew for 120′ before landing under the power of its pilot, making history as the first pilot-operated airplane.

In 1909, Captain George Groaman of the US Army Medical Corps built a plane specifically designed to carry patients; unfortunately, it crashed during testing and never received government approval. The first successful use of a motorized aircraft for transporting patients occurred 8 years later when a plane named the French Dorand AR II served as an air ambulance, transporting patients. During the next several decades, the air medical transport industry grew, predominantly as part of the military. In 1914, Igor Sikorsky invented the helicopter, which would later be used by the military to transport wounded soldiers from the battlefield to definitive medical care.

After World War I, the US Army took a new interest in the development and successful use of air ambulances. The chief surgeon of the Army proposed modifications to the de Havilland aircraft, making it capable of transporting a pilot, a medical officer, and two patients. This became the mind-set for the evacuation of injured soldiers during wartime. Further advancements continued to be made, and in the 1930s, air ambulances were specifically designed with multiple engines, heated cabins, and short runway capacity. Within the next decade, the push to create helicopters capable of transporting patients continued **Figure 1-5**.

Figure 1-5 The de Havilland DH89B Dominie loading a mock casualty onto an aircraft in 1945.

On April 23, 1944, several wounded airmen were transported via helicopter in Burma during World War II. This is thought to be the first time wounded soldiers were transported via this means, marking a new era of air medical transport. After the war, the military determined that air evacuation of wounded patients from battlefields would be a major goal. The first official military rescue evacuation was made on August 4, 1950, 1 month after the start of the Korean War. Wounded soldiers were transported to MASH (mobile army surgical hospital) units on cots fitted on the skids of Bell-47 and Sikorksy S-51 helicopters **Figure 1-6**. A drop in the mortality rate was directly attributed to the use of helicopters to rapidly evacuate wounded soldiers from combat and deliver them to surgical care **Table 1-1**.

The Vietnam War brought a significant change in the removal of wounded patients from the front lines. The majority of battlefield rescues, stabilizations, and evacuations were carried out by a much larger helicopter, the Bell UH-1 (Huey), code named *Dnstnff* **Figure 1-7**. This helicopter was capable of carrying more patients and personnel, and patients could be carried inside the aircraft where they could receive medical care en route to the receiving facility. This war saw the transport of more than 800,000 patients to specialty care facilities. The marriage of rapid air transport to specialty care facilities proved successful and kindled interest in the civilian arena.

TABLE 1-1 Mortality Rates in Military Conflicts

Military Conflict	Time From Injury to Surgical Care	Mortality Rate
World War I	12 to 18 hr	8.8%
World War II	6 to 12 hr	5.8%
Korea (with helicopter)	2 to 4 hr	2.4%
Vietnam (with helicopter)	1 to 1.4 hr	1.7%

Adapted from: Holmes E. *Soundings: aeromedical history.* Available at: http://members.cox.net/eholmes333/soun-pg4.html. Accessed January 28, 2008.

Figure 1-6 Soldiers wounded during the Korean War were sometimes evacuated on litters strapped to the skids of aircraft.

Figure 1-7 The Bell UH-1 helicopter, code named *Dustoff*, evacuated patients during the Vietnam War.

In 1958, Bill Mathews, a northern California businessman, was the first to organize a civilian air medical service. He lived in the small town of Etna and was met with skepticism from the town's 700 residents. Fortunately, Granville Ashcroft, MD, the area's only physician, began using the helicopter to transport patients. Soon the town druggist also started using the helicopter to deliver medications during emergencies.

Likewise, during the 1960s, Europeans instituted helicopter programs that were dedicated to patient care and transport. During that same period, the United States saw the emergence of several civilian- and government-funded helicopter projects. These projects were funded to study the feasibility of using helicopters to transport patients in the civilian arena. In 1966, the National Institutes of Health and National Academy of Sciences published "Accidental Death and Disability: The Neglected Disease of Modern Society," paving the way for the initiation and growth of civilian air medical transportation programs and recommending the initiation of a system designed to transport patients via air ambulances based on the successful military model.

In 1969, the state of Maryland received a grant to purchase a Bell JetRanger helicopter and instituted the nation's first public service medevac program **Figure 1-8**. The four helicopters, staffed with paramedics, were part of the Maryland State Police and were strategically stationed throughout the state for quick responses to emergencies. When the helicopters were not being used for medical emergencies, they could be used for law enforcement missions.

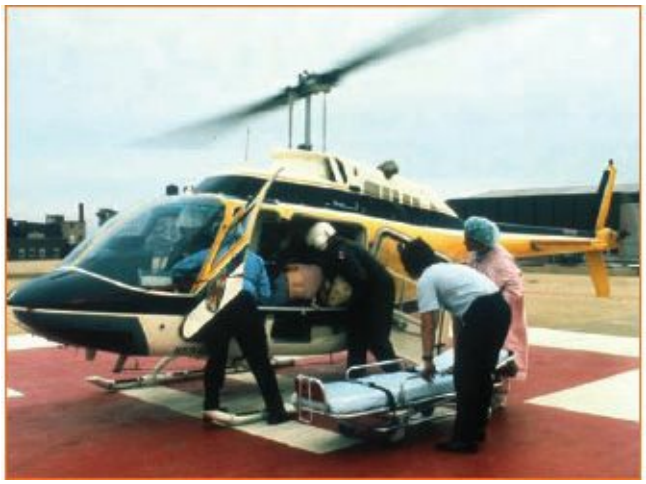


Figure 1-8 The Bell JetRanger helicopter was used for the first public service medevac program.

In 1972, the first hospital-based air medical transport program was established at St Anthony's Hospital, Denver, CO. Following continued skepticism about the civilian use of helicopters for medical transports, the National Highway and Transportation Safety Administration (NHTSA) released a study titled "Helicopters in Emergency Medical Systems: Experience to Date." This study stated that the use of helicopters in urban settings was not realistic and had little or no advantage over ground-based EMS systems. In 1981, a second report was released by the NHTSA titled "Air Ambulance Guidelines," in which Willis Winert, MD, indicated that air ambulances were improperly equipped to handle critically ill or injured patients and were nothing more than flying taxis with removable seats. This statement prompted Federal Aviation Association (FAA) regulators to demand better equipment and specially trained crews for air ambulances.

Since the development of air medical programs, the helicopter has been the obvious choice as an air ambulance owing to its ability to land and take off without an airport. Although it was met with much criticism regarding the effectiveness for transporting critically ill and injured patients, the development of

such a system has occurred in great leaps and bounds. With the help of special technologies, providers trained at the intensive care unit level can now deliver care in the transport environment.

Today, there are more than 650 air medical programs in the United States, each with specially trained flight crews. Because of the intensive training they receive and their high level of experience, flight teams are allowed by most medical practices to perform more sophisticated procedures than their prehospital and hospital counterparts.

Figure 1-9 outlines the history of ground and air transport.

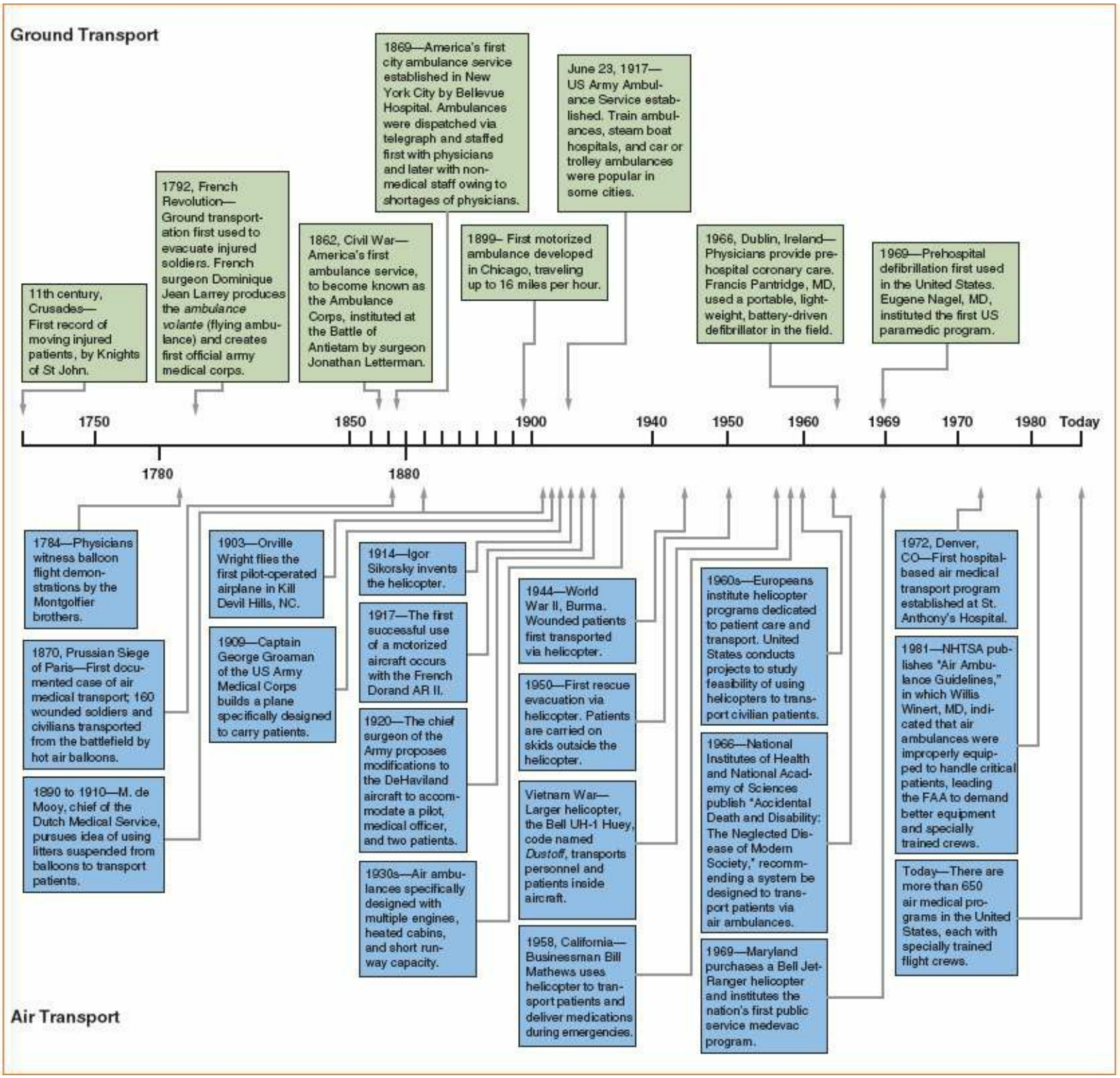


Figure 1-9 Timeline of the history of ground and air transport.

Critical Care Team Composition and Qualifications

The type of crew configurations varies as much as the types of ambulances and helicopters used throughout the United States. Whether in the air or on the ground, the configuration of the critical care transport team depends on the staffing policy of the organization. The combination of physicians, nurses, paramedics, and EMTs on critical care transport teams varies **Figure 1-10**.

The most commonly used combinations of health care professionals are as follows:

- Nurse and paramedic
- Nurse and respiratory therapist
- Nurse and physician

It is important to understand that this is not a comprehensive list, but rather the most commonly seen configurations. Other *less* common configurations include the following:

- Paramedic and physician
- Nurse, respiratory therapist, and physician

Figure 1-10 The critical care team composition varies but often includes a physician, a nurse, and a paramedic.

Additional crew members, including **perfusionists**, neonatalogists, or pediatric intensivists, may be required for certain patients in flight.

The three-team-member configuration is used but is less common because of the increased costs of adding an additional staff member. Most patients requiring transport do not have a condition with the acuteness that would necessitate a third team member.

As much as crew configurations vary, so do the recommended educational qualifications for each of the team members. CCTPs must be able to provide care at the same level and in many cases, at a greater level, than their non–critical care transport counterparts. In addition, CCTPs must be educated and skilled in the use of advanced practice procedures, such as surgical airways, blood product administration, pericardiocentesis, escharotomies, intracranial pressure monitoring, intra-aortic balloon pump therapy, central line and chest tube monitoring, monitoring arterial pressure lines, adult intraosseous infusion, and the use of ventilators and possibly portable ultrasound, which may fall outside of their formal medical education.

Emergency Medical Technicians

An EMT is trained at the basic life support level and frequently fills the role of ambulance driver. This position requires a recommended 150-hour program based on the EMT National EMS Education

Standards. EMTs' employers and the states in which they work may mandate additional requirements. An emergency vehicle operator's course (EVOC) is highly recommended. Finally, an EMT driver is also expected to maintain current certification and proficiency in CPR in order to assist the crew when necessary.

Paramedics

Paramedics must complete a course based on the Paramedic National EMS Education Standards encompassing 1,200 hours of didactic and clinical time, in addition to proving competency in all areas of study. They must also have an average of 5 years' experience in a busy 9-1-1 EMS system. Specialty certifications and verifications must also include courses in basic, advanced, and pediatric life support; basic and prehospital trauma life support; and a sanctioned critical care transport class.

Registered Nurses

Registered nurses must have attended an accredited nursing program, passed a state licensing examination, and have experience in one or a combination of the following areas: critical care, emergency care, and prehospital care. It is also recommended that they have specialty certifications and verifications in numerous areas, including flight medicine; emergency medicine; mobile intensive care; critical care; and advanced, pediatric, and neonatal life support. Finally, it is recommended that they have completed a variety of nationally recognized trauma care courses.

Respiratory Therapists, Perfusionists, and Physicians

Respiratory therapists, perfusionists, and physicians can function as regular members of the critical care transport team or as temporary members as needed. Respiratory therapists and perfusionists must attend and graduate from an accredited program and pass a national accreditation examination before practicing. They may also be subject to the qualifications and certifications required by the state in which they practice. The qualifications and certifications for physicians vary widely and are mandated on a state-by-state basis.

Modes of Transportation and Their Differences

Modes of transportation vary widely from agency to agency and by region of the country. The determination of the optimal transportation mode is dictated by patient acuteness, distance between sending and receiving facilities, weather conditions, and topography of the region. The standard types of **mobile intensive care units** used for critical care transports include mobile or ground units, rotor-wing aircraft, and/or fixed-wing aircraft. Commonalities among these transportation modes include standard equipment **Table 1-2**, the ability to accommodate the need for specialty equipment **Table 1-3**, and advanced medical training among their providers.

Mobile Units

Mobile (ground) units, also known as ambulances, are generally used to transport critically ill or injured patients up to distances of 50 miles from the patient's location to the receiving facility. A mobile unit may also be used when a patient has contraindications for air transport. These vehicles are available in many body types and sizes to suit the needs of the specific provider.

Rotor-Wing Aircraft

Rotor-wing aircraft, also known as helicopters, are used to transport critically ill and injured patients in rural settings and can travel distances of up to 150 miles. A wide variety of helicopters are available for use, including the BK 117, Bell Model 220/230, Sikorsky S-76, AgustaWestland AW109, and the Dauphin **Figure 1-11**. These helicopters can be single or twin engine and have the capability of loading patients from the side or the rear of the aircraft. As with ambulances, the size and type of helicopter varies by program needs and the area serviced.

TABLE 1-2 Standard Onboard Medical Equipment*

Fixed and portable suction units
Invasive monitoring devices
Arterial line kits and pressure monitoring capability
Pulmonary artery catheters
End-tidal carbon dioxide measurement devices
Oxygen
Pulse oximetry
IV infusion pumps
Portable Doppler machines
Electrocardiographic monitor and defibrillator
Transcutaneous pacemaker device
Autosyringe pumps
Central venous line kits
Chest tubes
Needle and surgical cricothyrotomy supplies
Ventilator
Intubation and airway equipment
Fluid warming device
Point of care (POC) testing for blood gases, glucose, hematocrit, and chemistries
Various medications
Indwelling urinary catheter kits
Ostomy management equipment
Peripheral IV supplies

*Varies based on state law and accreditation organizations.

TABLE 1-3 Specialty Equipment
Neonatal isolette
Portable intra-aortic balloon pump
Ventricular assist device
Extracorporeal membrane oxygenation support device

Fixed-Wing Aircraft

Planes or **fixed-wing aircraft** are capable of transporting critically ill or injured patients a distance of 150 miles or more. These aircraft models can range from the Cessna to Learjet and are often used in combination with mobile critical care units, which transport patients to the aircraft site **Figure 1-12**.

Dispatch

Having an organized and competent staff of emergency dispatchers is an integral part of the critical care transport team. Depending on the situation, the dispatch center may be located within a hospital or at a remote, independent site. Regardless of the scenario, the initial triage process starts with the call taker. A standard handwritten form is typically completed by the call taker, as information about the patient and his or her condition is obtained from the referring facility. Examples of pertinent information that should be obtained from the referring facility are as follows:

Figure 1-11 Various types of helicopters include some of those shown. **A.** BK 117. **B.** Bell Model 206. **C.** Sikorsky S-76. **D.** AW109. **E.** Dauphin. Part C: © Sikorsky Aircraft Corporation 2009. All rights reserved. Used with permission of Sikorsky Aircraft Corporation.

Figure 1-12 Fixed-wing aircraft are used for longer transports. **A.** Cessna. **B.** Challenger 604A.

- Patient age
- Patient weight
- Diagnosis
- Number and type of infusions
- Most recent vital signs
- Level of consciousness
- Airway status (patent or intubated)
- Presence of mechanical ventilation
- Presence of cardiac-assist devices

Although this list does not include all information needed about the patient, it does provide dispatch center personnel adequate information for proper decisions. Once this information has been obtained, a clinical supervisor should be involved to decide on the crew configuration and the mode of transportation. In many cases, the attending physician and staff at the referring facility suggest the particular mode of transportation that they believe is necessary. Many critical care transport services honor their request, whereas others make their own determination based on the patient report provided.

Another factor that needs to be considered by dispatch center personnel is the type of call. A critical care transport team should be dispatched and used for patients with any of the following situations:

- Mechanical ventilation
- Vascular access devices
- Vasoactive medications
- Colloid infusions (ie, blood, plasma, or platelets)
- Cardiac-assist devices (ie, intra-aortic balloon pump, extracorporeal membrane oxygenation, or ventricular-assist device)
- Instability requiring frequent interventions such as sedation, paralytics, and/or analgesia

If a situation arises in which a service is asked to transport a patient who requires one of the aforementioned cardiac-assist devices, a certified perfusionist may need to be part of the team to manage and monitor the devices. CCTPs remain responsible for all other aspects of patient care during transport.

Transporting by Ground vs Air

Each mode of transporting critically ill and injured patients has its own set of advantages and disadvantages. **Table 1-4** lists some of the advantages and disadvantages of each mode.

The decision to transport a patient by ground ambulance or helicopter is a decision that must be made as rapidly as possible. In most cases, the patient needs expedient transport to the tertiary care center to receive the care not available in the present location. Helicopters enable more rapid transport than ground vehicles because they can bypass vehicular traffic, intersections, and stop lights, which delay the total transport time. Although a helicopter travels much faster than a ground ambulance, it may not be the most practical solution for transporting a patient to the tertiary care center. Helicopters are usually strategically located throughout a geographic area. Depending on the location of the referring hospital, the helicopter may need lengthy travel time to reach the patient. In a situation like this and if a critical care ground ambulance is significantly closer, the most reasonable solution may be ground transportation.

The acuteness of the patient's condition is one of the major determinants of whether ground or air transportation is needed. The determination is typically made by the referring physician, who is ultimately responsible for patients until they are transported out of the referring facility. Time-sensitive conditions that may require air transport include:

- Acute myocardial infarction
- Acute cerebrovascular accident
- Severe traumatic injury
- Acute intracranial hemorrhage
- The need for emergency surgery (ie, abdominal aneurysm, limb reattachment)

TABLE 1-4 Advantages and Disadvantages of Transportation Modes

Transportation Mode	Advantages	Disadvantages
Mobile (ground) unit	• Large interior • Quieter than aircraft • No special housing needed • Less expensive to operate and maintain • Less specialized training needed to operate vehicle (emergency vehicle operator course) • Multiple provider capabilities • Less weather dependent • Fewer height and weight limitations • Fewer adverse effects on patient and crew	• Usually limited to single-patient transport • Slower than aircraft • Limited travel distance
Rotor wing (helicopter)	• Speed (100–180 mph) • Specialized personnel and technology needed or provided • Few altitude ramifications below 2,000′ • Can serve a larger portion of the community	• Restricted by weather conditions • Interior space limitations • Expensive to operate and maintain • Special landing requirements • Need for FAA communications • Adverse effects on crew

	• Two-patient capabilities	members and patients • Expensive to patient • Requires special housing • Limited space for providers and equipment • Weight limitation
Fixed wing (plane)	• Speed • Multiple patient capabilities • Capable of traveling great distances • Specialized personnel and technology needed or provided • No weight limitation • Instrument-assisted flying	• Adverse altitude considerations • Adverse effects on patient and crew • Need for FAA communications • Expensive to patient • Expensive to operate and maintain • Can only serve a small portion of the community • Requires special housing • Special landing requirements
Abbreviation: FAA, Federal Aviation Administration.		

The list is not all-inclusive, and many other conditions and situations warrant air medical transport. Each case must be individually evaluated, and the determination should be made after all data have been reviewed.

The thought process for determining the mode of transportation is relatively simple; however, certain unpredictable occurrences might preclude use of the first choice. For example, a clinical coordinator could decide to transport a patient by helicopter or fixed-wing service, but weather issues may prohibit the flight. An alternative method must be identified so the patient will arrive at the accepting facility within a reasonable time. Sending a ground unit may be more harmful to the patient due to the extended return time to the accepting facility. In most such cases, the decision is made to wait for the weather to improve and launch the aircraft as soon as is practical. When the distance from the referring facility to the accepting facility is not that great, CCTPs may choose to complete the mission by ground.

Medical Control

There are varying levels of medical control, or medical direction, within any given critical care transport system. The first type is protocols, which allow CCTPs to initiate or maintain certain therapies and treatment without contacting a command physician. The protocols in a critical care transport setting are typically much more aggressive and give CCTPs the opportunity to perform these tasks without contacting online medical control. The second type is online medical control in which CCTPs take part in a real-time interaction with a medical control physician. This task is typically accomplished through telephone or radio communications **Figure 1-13**. The degree of medical guidance provided relies solely on the type of assistance needed. An example in which a CCTP might need to consult with online medical control is

during the transport of a ventilator-dependent patient who experiences hypercarbia. If the local protocol does not allow CCTPs to titrate ventilator settings, online medical control must be contacted immediately to obtain orders for corrective action. This type of guidance must continuously be available to CCTPs, regardless of the given situation.

Figure 1-13 Online medical control involves working directly with a medical control physician via phone or radio.

Interfacility Transport

Part of the responsibility of EMS includes interhospital transfers. The continuum of care during transfer is expected to be maintained at the level at which it originated. **Specialty care transport (SCT)** is the term used by the Centers for Medicare and Medicaid Services (CMS; formerly the Health Care Finance Administration, or HCFA) to define the ground transportation and medically necessary supplies and services for critically ill or injured patients. SCT is a level of interfacility service established to provide these services, which are beyond the scope of practice for providers not trained at the CCTP level. SCT is considered one step above the level of advanced life support transfers because it involves patients in more critical condition, requires the use of specialized equipment, and necessitates additional training or education for registered nurses and paramedics.

Along with using the services of the SCT companies, many facilities are preparing transfer teams to assist in these transports in an effort to provide continuous bed-to-bed care. As the acuteness of the patient's condition increases, the critical care transport team is often supplemented with staff from the sending or receiving facility. This supplemental assistance allows incorporation of health care services with EMS services. The interfacility transport is dependent on collaborative rapport among all team members, in addition to staff and physicians at the referring and receiving facilities.

State and National Standards

Standards vary from state to state; however, most critical care transport programs follow national standards. Some states, such as Maryland, require any air medical service that operates in the state to be certified by the Commission on Accreditation of Medical Transport Services (CAMTS). However, more often than not, a state organization governs the licensure of air medical services and their practitioners. For example, in Pennsylvania, the Department of Health licenses air medical transport services and prehospital care providers. An individual CCTP's scope of practice also varies according to his or her level of certification and the state in which he or she practices. For example, registered nurses may administer any medication ordered by a physician, whereas paramedics may administer only medications

approved and listed on their state's prehospital drug list. The most accurate information on governing organizations and scope of practice is available from the state agency responsible for EMS accreditation and licensure.

Reimbursement Criteria

HCFA was developed in 1977 with the sole purpose of overseeing the Medicare and Medicaid programs. Now, as CMS, it serves 67 billion people and spends more than $230 billion each year on health care services. Its primary responsibility in the critical care transport field is to ensure that patients are taken to the closest appropriate facility. For example, consider a patient who has been diagnosed with an acute myocardial infarction at a local facility without interventional cardiology capabilities. Arrangements are made for transfer of the patient to a hospital with such capabilities, which is 50 miles away. A helicopter is called to transport the patient to this facility for balloon angioplasty; the patient spends 2 days as an inpatient. During billing review by CMS staff, it is determined that the helicopter passed over a hospital 20 miles closer and offered the same cardiac capabilities. This is significant because most air medical transport services charge $50 to $70 per loaded mile. In this situation, Medicare would reimburse for a total of 30 miles instead of 50, which totals $1,000 or more that would not be reimbursed.

Well-Being

The same tenets of well-being in EMS apply to well-being for the CCTP. Proper nutrition, weight control, and a regular exercise program are important.

Shift work can affect a person by disrupting his or her circadian rhythm. These effects and the effects of fatigue are discussed in Chapter 4.

Stress

Working in the fast-paced environment of critical care transport poses unique stressors on the body. *Stress* can be defined as the wear and tear bodies experience during adjustment to the activities of daily life and the environment. Stress is a normal feeling that affects people differently. It is capable of causing positive and negative feelings. Positive stress can heighten awareness of a situation or provide new perspectives, whereas negative stress can cause negative feelings such as depression or anger.

Like other health care providers, CCTPs are susceptible to burnout. Burnout is a consequence of chronic, unrelieved stress. While these stresses may be legitimate, how a person reacts to them can help alleviate the stress. Avoiding perfectionist beliefs can help reduce stress. Symptoms of impending burnout include:

- Chronic fatigue and irritability
- Cynical, negative attitudes
- Lack of desire to report to work
- Emotional instability
- Changes in sleep patterns, or waking without feeling refreshed
- Feelings of being overwhelmed, or being helpless or hopeless
- Loss of interest in hobbies
- Decreased ability to concentrate
- Declining health—frequent colds, stomach upsets, or muscle aches and pains

- Constant muscle tightness
- Overeating, smoking, or abusing drugs or alcohol

Because CCTPs work in proximity to controlled substances, substance abuse can easily become a way of dealing with negative stress when overwhelmed. Be alert to signs of substance abuse among colleagues. Signs and symptoms of substance abuse are wide and varied. Some classic signs include:

- Sudden change in behavior or becoming secretive
- Frequent absences from work or difficulty carrying out usual responsibilities
- Impulsive behaviors and lack of self-control
- Physical symptoms such as dilated or constricted pupils, needle marks, and red eyes

CCTPs must be cautious and not jump to conclusions if colleagues have one or more of the aforementioned signs. CCTPs should report suspected substance abuse to their supervisor, who will follow the program's policies and procedures for managing the situation.

Finally, the CCTP will encounter critical incidents (those that overwhelm the CCTP's ability to cope, either at the scene or later). It is standard practice for EMS systems in the United States and abroad to deploy specially trained teams to conduct critical incident stress debriefings (CISDs) with emergency personnel who have been involved in particularly traumatic calls or other painful incidents. A CISD is usually held within 24 to 72 hours of the incident. The sorts of incidents that are apt to require debriefing include:

- Serious injury or death of a fellow worker in the line of duty
- Suicide of a colleague
- Multiple-casualty incidents
- Serious injury or death of a child
- Intense media attention to an incident

Safety Gear

Appropriate safety gear and clothing are necessities for every member of the critical care transport crew. CCTPs should ensure that the appropriate safety gear (PPE, N95 mask, etc.) and clothing are properly and consistently worn **Figure 1-14**. At a minimum, safety gear should meet the following criteria:

- Clothing should fit properly and be flame retardant. Clothing should include a uniform or flight suit, outerwear such as a coat or vest, and undergarments. Clothing should never be tight or loose fitting.
- Clothing should be layered for proper ventilation and heat regulations. Long-sleeved shirts and pants limit water loss and exposure.
- Head and eye protection should fit snugly and meet or exceed Occupational Safety and Health Administration specifications.
- Leather boots should be worn to protect feet against the elements and fire. Flame-retardant gloves should be worn to protect the hands against the elements and fire.
- Uniforms should have reflective materials or striping to increase nighttime visibility of CCTPs at crash scenes.

Figure 1-14 Proper safety gear and clothing, including snug-fitting head and eye protection, leather boots, and flameretardant gloves. Layers should be worn underneath the uniform and for additional protection. If involved in scene transports, the CCTP uniform should also include reflective material or striping.

Interpersonal Communication

Communicating properly and effectively with patients and families is of utmost importance. Most acute illnesses are not predictable or expected. When they occur, all parties involved experience tremendous fear and anxiety. No matter how acutely ill a patient is, each member of the critical care transport team must make every effort to communicate intentions to the patient and family. Communication starts with an introduction of each team member. These simple introductions help comfort patients and families. When family members desire, they should be in the room when the team assesses the patient.

When preparing to depart with the patient, family members should be given time to speak briefly with the patient, and questions they have should be answered. Requests for a brief prayer by members of the clergy or others should be honored. Although every second counts when transporting critically ill or injured patients, the brief moments for family involvement have effects that words cannot describe.

Death and Dying

Because of the severity of the conditions of the patients with whom CCTPs interact, death and dying are a regular part of the job. Not only will CCTPs interact with seriously ill and injured patients, but they will also interact with the patient's family members who are in various stages of the grieving process. CCTPs should be familiar with Elisabeth Kübler-Ross' five stages of grieving, summarized in **Table 1-5**. CCTPs must realize that these stages will not necessarily occur in the order listed. Also, not all people experience all stages, although they will experience at least two.

TABLE 1-5 Five Stages of Grieving by Kübler-Ross

Stage	Description
Denial	The person cannot believe what is happening.
Anger	The person asks, "Why me?" or thinks, "It's not fair."
Bargaining	The person wishes to live for a certain amount of time, for example to see a child marry.
Depression	The person is sad and feels that nothing matters.
Acceptance	The person comes to peace with the issue and accepts the outcome.

When communicating with critical care patients and their families, CCTPs should follow the same principles as in any other professional health care setting: Listening provides comfort to patients and families, shows empathy, and provides helpful data for effective care. Telling patients and families that the best possible care will be provided offers realistic reassurance and promotes confidence.

Quality Assurance and Improvement

Quality assurance and improvement (QA&I) programs are an essential part of the overall health care system in the United States. The primary function of a QA&I program is to generate data to be used to improve the quality of service provided. A QA&I program is an integral component of a successful critical care transport program. CCTPs are regularly charged with providing advanced care to the "sickest of the sick," often in uncontrolled, unpredictable environments. An effective QA&I program serves as a check and balance system to protect stakeholders.

Once financial and human resources are devoted to a QA&I program, the first step is to define quality. Many critical care transport programs use their mission statement to define what quality means to their stakeholders. Once defined, measurable indicators of quality must be established and baseline data must be obtained.

The second step is to collect and analyze data. Data can be obtained from various sources. Most critical care transport programs obtain data, at a minimum, from patient charts, dispatch records, and customer feedback surveys. Once data are obtained, they must be analyzed for meaning and application to the program's goals, objectives, and indicators of quality.

The third step in the QA&I process is focused on improving quality, which requires analysis of the data and development of an understanding of underlying issues. For example, if an audit of patient charts reveals that several CCTPs are incorrectly documenting the use of transport ventilators, and analysis of the reason for incorrect documentation reveals a lack of knowledge, a continuing professional education course on effective documentation for ventilator-dependent patients can be conducted. The QA&I program should direct the continuing professional education for transport program staff

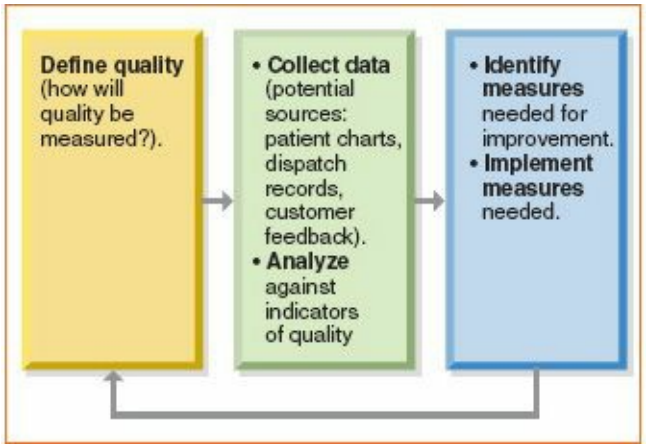


Figure 1-15 The three components of the quality improvement process: defining quality, collecting and analyzing data, and determining and implementing measures to improve quality.

Continuing Education

Critical care medicine is a dynamic, ever-changing science that requires CCTPs to be lifelong learners. Most critical care transport programs require CCTPs to complete frequent continuing professional education classes, which exceed the requirements to maintain the state or national certification and/or licensure necessary for employment. Most critical care transport services hold a variety of mandatory educational and competency training days each year. Topics include review of uncommonly used advanced procedures, skill and competency validation, case studies, and cutting-edge clinical information.

Summary

Much attention has been given to specialty care and critical care transports since the establishment of reimbursement fees by Medicare. As the health care system continues to evolve, so must CCTPs and the agencies providing transports.

Despite the extensive amount of training necessary to become a member of a critical care transport team, clinical care is only one of the many aspects to evaluate when considering how stakeholders perceive quality. Excellence in clinical care is not a "nice to have" but an expectation. CCTPs must remember that a "routine trauma case" to CCTPs is most likely a life-changing event for patients and families. With this in mind, CCTPs must look and act in a professional manner at all times. In addition, CCTPs must use effective communication skills in interactions with patients, families, and other health care providers.

Case Study

AFTER 1 WEEK OF GENERAL PROGRAM ORIENTATION, your first day of duty as a flight paramedic finally arrives. After the morning safety briefing, your preceptor takes you to the helipad to review the aircraft safety and procedures that you learned at the general orientation program. As you make your way back to the base, you hear the klaxon alerting system and the radio say, "Medevac 1 stat page scene run town of Martins Ferry, Belmont County, Ohio." You check with your preceptor and the pilot to see if the flight can be accepted. Once the determination is made to accept the flight, you pack the blood and make your way back to the helipad to complete your preflight checklist.

In flight, your communications center advises that you are responding for a 28-year-old patient who was in an all-terrain vehicle (ATV) crash in a remote area. Ground EMS reports that the patient was an

unhelmeted operator of an ATV that hit a tree head on.

On landing at a remote landing zone, you are transported by a fire department unit to the crash scene. You find your patient lying supine on the ground with obvious head, abdominal, and leg injuries. He responds only minimally to pain and has decorticate posturing. His vital signs are as follows: Glasgow Coma Scale (GCS) score, 7; blood pressure, 190/100 mm Hg; pulse rate, 68 beats/min; and respirations, 8 breaths/min, assisted by ground providers with a bag-mask device. Attempts at oral intubation were reportedly unsuccessful owing to clenched teeth. You and your preceptor quickly determine that the first thing needed is to obtain definitive control of the airway. Your preceptor directs you to establish an intravenous (IV) line of normal saline solution and begin administering drugs according to the rapid-sequence intubation (RSI) protocol. Once the IV line has been established and the drugs prepared, you check your calculations with your preceptor and then administer the appropriate doses of lidocaine, etomidate, and succinylcholine. Your preceptor swiftly places an endotracheal tube and begins manual ventilation while you attach a capnography monitor and confirm tube placement as per protocol. You then administer the appropriate doses of vecuronium and fentanyl. Your preceptor tells you that he will oversee immobilization of the patient and transportation to the aircraft while you contact medical control for ventilator settings for the 25-minute transport to the trauma center. You contact medical control via cellular telephone and are lucky enough to get the program's medical director, command physician 1, and report:

> *"This is Medevac 1. We are currently loading a 28-year-old, 90-kg, male patient, who was the unhelmeted operator of an ATV that hit a tree head on. On arrival, the patient had a GCS score of 7. We used RSI and intubated him orally. Our flight time is 25 minutes, and we need ventilator settings."*

Physician 1 asks several questions about the mechanism of injury and then tells you to set the ventilator at a tidal volume of 900 mL, rate of 14 breaths/min, and a fraction of inspired oxygen (FIO_2) of 100% with +5 of positive end-expiratory pressure (PEEP). You repeat the orders and thank the physician and approach the aircraft, where your preceptor just loaded the patient for transport. The transport ventilator is set up and the patient is "connected" according to the ordered settings.

In flight, you monitor your patient's vital signs, including end-tidal carbon dioxide levels, and administer additional vecuronium and fentanyl as necessary to effectively maintain paralysis and sedation. On arrival at the trauma center, you do a "hot" off-load (unloading the patient without shutting down the aircraft) and transport the patient to the trauma bay, where you give your report to the trauma team. You and your preceptor clean your equipment and get the aircraft back in service. Once you return to base, you complete the patient's medical chart and notify the referring EMS agency. As you and your preceptor are discussing the call and completing your postflight orientation report, the program medical director calls and says he needs to talk about your medical consult report.

1. What are the components of a daily safety briefing?
2. What additional pertinent information should you request from dispatch?
3. What additional information should you have provided to online medical control?

Analysis

The safety briefing should include the daily plan, crew member duties, equipment and aircraft issues, weather expectations, and emergency situations. Safety is an integral part of each shift. Team members must be familiar with the inherent occupational safety risks and procedures in order to protect patients and themselves. Critical care transport teams should hold daily safety briefings at the beginning of each scheduled shift.

Although sometimes information may be limited from crews at a hectic scene, if possible, CCTPs should ascertain the patient's level of consciousness, airway status, and most recent vital signs. Effective interpersonal communication skills are essential to the success of CCTPs. In this case, greater details from the dispatch center would have allowed the CCTPs to be better prepared to provide the appropriate level of care and ensured that all necessary equipment was taken to the crash scene. Specifically, as much information as possible should be obtained from ground crews about the patient's level of consciousness and airway to allow for preparation of RSI equipment and drugs. If the need for RSI and the patient's weight are available en route, drugs can be prepared in the correct doses to allow expeditious airway control on arrival at the scene.

In the field, CCTPs are the physician's eyes and ears and must provide a detailed report. In this case, the medical control physician should have been provided with the mechanism of injury, current vital signs, capnography reading, medications administered, assessment findings, and any other pertinent information about the medical history. Detailed reports to the medical control physician allow for the receiving facility to have necessary equipment and personnel available to care for the patient on arrival.

Prep Kit

Ready for Review

- Critical care transport is the provision of medical care to a critically ill or injured patient during ground or air transport.
- CCTPs include EMTs, paramedics, nurses, physicians, and specialty health care personnel such as respiratory therapists and perfusionists.
- CCTPs must be able to provide care at the same or sometimes greater level than their non–critical care counterparts.
- CCTPs must be educated and skilled in the use of advanced practice procedures that may fall outside of their formal medical education.
- Ground and air transport have evolved along with changes in transportation and technology.
- Crew configurations vary as much as the types of ambulances and helicopters. The configuration depends on the staffing policy of the organization. The most common configurations are nurse and paramedic, nurse and respiratory therapist, and nurse and physician.
- Additional crew members, including perfusionists and neonatal or pediatric intensivists, may be required for certain patients.
- Modes of transportation may vary widely by agency and by region of the country. Possible modes of transport include mobile or ground units, rotor-wing aircraft, and fixed-wing aircraft.
- The mode of transport to be used is dictated by patient acuteness, distance between sending and receiving facilities, weather conditions, and topography of the region.
- Mobile or ground units (ambulances) are generally used to transport critically ill and injured patients up to distances of 50 miles from the patient's location to the receiving facility.
- Rotor-wing aircraft (helicopters) are used to transport critically ill and injured patients in rural settings and can travel distances of up to 150 miles.
- Fixed-wing aircraft (airplanes) are capable of transporting critically ill and injured patients a distance of 150 miles or more.

- Dispatch personnel obtain pertinent information about patients and their condition from the referring facility. A clinical supervisor then helps determine crew configuration and mode of transportation.
- A critical care transport team should be dispatched and used for patients with any of the following: mechanical ventilation, vasoactive medications, colloid infusions, cardiac-assist devices, and unstable conditions requiring frequent interventions such as sedation, paralytics, and/or analgesia.
- Although a helicopter travels much faster than an ambulance, in some cases, a ground ambulance may be located significantly closer to the patient and, therefore, is the preferred mode of transport.
- Patients with the following time-sensitive conditions are typically transported by air: acute myocardial infarction, acute cerebrovascular accident, severe trauma, acute intracranial hemorrhage, and the need for emergency surgery.
- Weather issues may prohibit a flight from taking place. Usually, a decision is made to wait for the weather to improve and launch the aircraft as soon as practical.
- Air transport programs are typically hospital based with many hospitals sharing the responsibility and costs of that service. Air transport programs can also be provided by public service agencies or private services.
- The basic concept of a critical care transport service is to rapidly transport CCTPs to a patient, stabilize the patient's condition, and transport the patient to a tertiary care center as rapidly as possible.
- Patients should be evaluated and assessed for the effects of altitude and other forces that can adversely alter their conditions en route.
- Medical control within critical care transport systems includes protocols and online medical control. Protocols in a critical care transport setting are typically much more aggressive than in other aspects of prehospital care.
- Standards vary from state to state; however, most critical care transport programs follow national standards. Usually a state organization governs the licensure of air medical services and their practitioners.
- An individual CCTP's scope of practice varies according to his or her level of certification and the state in which he or she practices.
- The CMS ensures that patients are taken to the closest appropriate facility. This organization performs billing reviews and determines whether the patient was taken to the closest facility with the necessary capabilities. Medicare and Medicaid make reimbursement decisions based on the CMS assessment.
- Appropriate safety gear and clothing are necessities for every member of the critical care transport crew and include layered clothing with reflective materials and flame resistance, head and eye protection, and leather boots.
- Interpersonal communications are as important in critical care transport as in other health care settings. CCTPs should introduce themselves to patients and family members and maintain professionalism and a caring demeanor.
- Quality assurance/improvement programs are an essential part of the overall health care system. Most critical care transport programs obtain data from patient charts, dispatch records, and process customer feedback surveys.

Vital Vocabulary

critical care Constant, complex, detailed health care as provided in various acute life-threatening conditions; the ability to deal with crucial situations rapidly and with precision using various advanced machines and devices for treating and monitoring the patient's condition.

critical care patient Any patient who experiences an actual or potential life-threatening illness or injury that requires continual monitoring and care by a specially trained physician, registered nurse, or paramedic.

critical care transport The transport of a patient from an emergency department, critical care unit, or incident scene during which the patient receives the same level of care as was provided in the hospital or originating facility.

critical care transport professional (CCTP) A health care professional who has successfully completed a recognized critical care program and meets the minimum qualifications set forth by the employing transport program.

fixed-wing aircraft Airplanes; these are capable of transporting critically ill and injured patients a distance of 150 miles or greater.

mobile intensive care units Ambulances or helicopters that are used only for maintaining specialized or intensive care treatment; used primarily for interfacility transports.

morbidity An illness or an abnormal condition or quality; the rate at which an illness occurs in a particular area or population.

mortality The condition of being subject to death; the number of deaths per unit of population in any specific region, age group, disease, or other classification.

perfusionists Highly trained technicians who are intimately familiar with the operation of intra-aortic balloon pumps and adult and pediatric extracorporeal membrane oxygenation machines and who may assist during any medical situation, including critical care transports, in which it is necessary to support or temporarily replace a patient's circulatory or respiratory function.

rotor-wing aircraft Helicopters; these are used to transport critically ill and injured patients in rural settings and can travel distances of up to 150 miles.

specialty care transport (SCT) The term used by the Centers for Medicare and Medicaid Services to define ground transportation and medically necessary supplies and services for critically ill and injured patients.

Bibliography

Association of Air Medical Services. Frequently asked questions about AAMS and air ambulance/medevac. Available at: http://www.aams.org/Content/NavigationMenu/AboutAAMS/FrequentlyAskedQuestions/default.htm. Accessed January 8, 2008.

The Automotive Chronicles. Available at: http://www.automotivechronicles.com/articles/2005/apr/02/index.php. Accessed January 7, 2008.

Bellis M. Ambulance history. Available at: http://inventors.about.com/library/inventors/blambulance.htm. Accessed November 19, 2007.

Olive-Drab.com. Aeromedical evacuation in World War I. Available at: http://www.olive-drab.com/od_medical_evac_fixedwing_ww1.php. Accessed January 8, 2008.

Emergency Nurses Association/Air & Surface Transport Nurses Association. Joint position statement: staffing of critical care air medical transport services. 2001. Available at: http://www.astna.org/PDF/Staffing-Position.pdf. Accessed March 11, 2009.

EMS history, part I. Available at: http://www.nyc.gov/html/fdny/html/ems_week/ems_history1.html. Accessed March 10, 2003.

The history of the air ambulance. Available at: http://www.bellhelicopter.com/print/encyclopedia/applications/ems/airMedBook/section2/html. Accessed February 13, 2003.

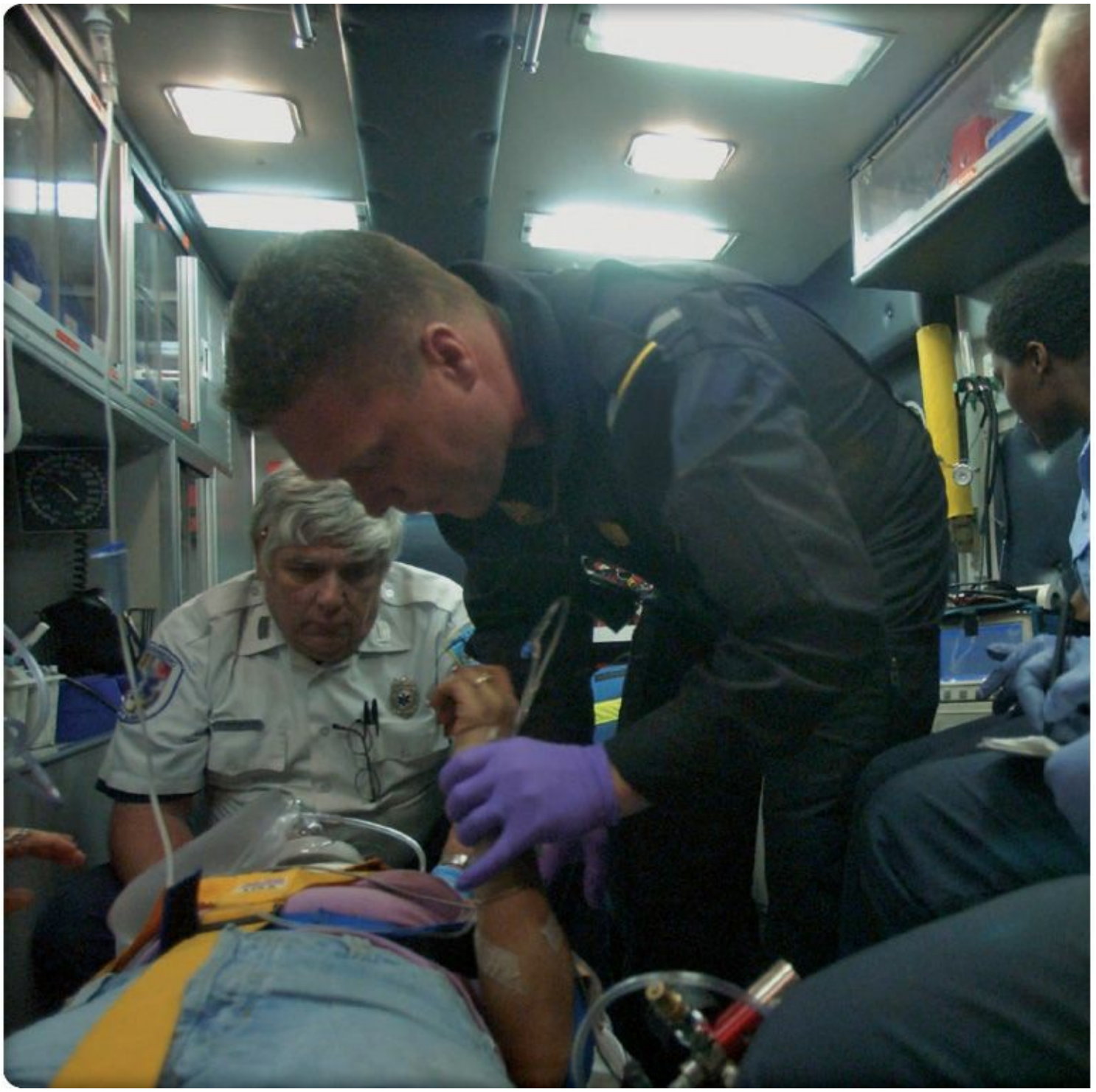
History of air medical transport systems. Available at: http://www.bellhelicopter.com/print/encyclopedia/applications/ems/airMedBook/section3.html. Accessed February 13, 2003.

The history of EMS: milestones in EMS. Available at: http://warhammer.mcc.virginia.edu/cars/milestones.html. Accessed November 19, 2007.

Holmes E. Soundings: aeromedical history. Available at: http://members.cox.net/eholmes333/soun-pg4.html. Accessed January 8, 2008.

Hudak C, Gallo B, Morton P. *Critical Care Nursing: A Holistic Approach*. 7th ed. Philadelphia, PA: Lippincott; 1998.

Varon J, Wenker O, Fromm R. Aeromedical transport: facts and fiction. *Internet J Emerg Intensive Care Med.* 1997;1. Accessed January 28, 2008.



Medical-Legal Issues

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Objectives

1. Discuss the Emergency Medical Treatment and Active Labor Act (EMTALA), its implications for critical care transport, and the potential consequences of violating the law (p 22).
2. Describe how the transport provider's certification level and scope of practice affect the steps of planning and executing a critical care transport (p 25).
3. Recognize and discuss patient rights and the legal risks and liabilities they pose in critical care transport (p 26).
4. Describe the legal principles of consent, negligence, and abandonment (p 26–28).
5. Discuss the major steps and pertinent issues in accepting a patient transfer (p 30).
6. Discuss the major steps and pertinent issues in assessing and preparing for transfer of a patient (p 30).
7. State the responsibilities of CCTPs during transport (p 31).
8. State the role of other health care providers who accompany the patient during transport (p 32).
9. Discuss the ways in which communications capabilities and agreements about medical direction affect medical decision making in the transport environment (p 30).
10. Discuss the major steps and pertinent issues in transferring care to the receiving facility (p 33).
11. Identify areas of potential liability for CCTPs and the transport agency (p 26).
12. State how risks can be minimized in the critical care transport environment (p 32).
13. Discuss the general concepts established by current EMTALA case law (p 22).

Introduction

Legal considerations are a constant presence in health care. One way for caregivers to reduce their level of concern about legal action is to give diligent attention to the various elements contributing to the quality of care they provide: skills maintenance, continuing education, protocol familiarity and compliance, and interpersonal communications, among others.

In addition, care providers have to devote a certain amount of attention to remaining familiar and complying with the laws and regulations that govern their practice. These include federal laws that apply across all American jurisdictions and state and local requirements that differ considerably from one area to another.

This chapter reviews general legal principles and specific laws that affect CCTPs. It concludes with discussions of specific cases that have helped clarify federal law on the subject.

The Emergency Medical Treatment and Active Labor Act

Interfacility transport is the main responsibility of a critical care transport service, whereas scene

transports are less common. The number of interfacility transports has risen dramatically, as demonstrated by the increase in the number of critical care transport services established throughout the United States and the increased interest in critical care transport training courses. It has become a crucial and specialized component of EMS. Interfacility transport comprises an ever-increasing portion of the calls for many EMS agencies and results in a great deal of income. With the evolution of specialized interfacility transport come additional areas of legal liability, most of which relate to negligence (discussed later in this chapter).

Many interfacility transport agencies have evolved as services provided by hospitals, although a substantial and ever-increasing number of such transports are handled by non–hospital-based services. Transporting critically ill or injured patients is a complex process and requires the cooperation of all entities involved. Hospitals that participate in Medicare reimbursement programs are subject to the **Emergency Medical Treatment and Active Labor Act (EMTALA)**, so personnel who perform interfacility transports need to be familiar with EMTALA and how it applies to them. The addition of EMTALA, enacted as part of the Consolidated Omnibus Budget Reconciliation Act (COBRA) in 1986, was initially designed to prevent financially motivated transfers of patients in unstable condition. EMTALA is also known as the "antidumping act" because its primary focus is to add safeguards to prevent facilities from selecting the most favorable patients (capable of paying for their services) and transferring patients who are unable to pay. Although EMTALA is intended to regulate hospitals and the manner in which they conduct transfers, EMS providers involved in these transfers must be familiar with the law.

EMTALA requires all medical facilities that receive Medicare funding to choose "qualified personnel and transportation equipment" for each patient transferred in unstable condition. But because COBRA neither defines qualified personnel nor transportation equipment, it was left up to others to do so in an effort to determine guidelines for such transfers.

To comply with EMTALA, a hospital has certain obligations to the patient that must be met. These include the following:

- Provide an appropriate medical screening examination within the capabilities of the hospital to any individual who presents to the hospital, using all available resources to determine whether an emergency condition exists. It is important to understand that a screening examination is different from triage and must involve several basic elements that include a triage record; initial vital signs; oral history; physical examination; use of appropriate diagnostic resources to determine if an emergency condition exists; use of on-call physicians, if necessary; and discharge vital signs. All of the foregoing must be documented. The screening examination should be conducted by a physician or, if properly designated by the hospital, a mid-level provider such as a physician's assistant or nurse practitioner.
- Provide such treatment as may be required to stabilize the condition if an emergency condition is found or transfer the patient to another medical facility with specialized capabilities not available at the first hospital in accordance with other sections of the law. For purposes of EMTALA, an emergency condition is deemed to exist if the following exists:
  - The patient has acute symptoms that could reasonably be expected to result in serious impairment or serious dysfunction of any organ or part, or
  - In the case of a pregnant woman having contractions, there is inadequate time to safely transfer the patient to another hospital.

  It is important to note that an interfacility transfer requires the consent of the patient or a legal representative. In some cases, when the patient is incapable of providing consent and no legal representative is available, a transfer may occur without consent. (A more detailed discussion of

the transfer requirements is given later in this chapter.)

A hospital is subject to EMTALA when the patient comes onto hospital property. The scope and further refinement of EMTALA are ongoing as cases are tried in courts all over the United States; for example, one case's judgment indicated that EMTALA is not triggered until the patient presents to the hospital's emergency department. This legal evolution has had substantial impact on EMS agencies that provide interfacility transport. For example, there are specific portions of EMTALA that require "qualified personnel" and "appropriate equipment" for each transport. More recent findings indicate that hospital property includes ambulances owned by the hospital, even if the ambulance is not on hospital grounds.

The use of helicopters and the implications of EMTALA have been the subject of much interest in recent years. The *State Operations Manual*, published by the US government's Centers for Medicaid and Medicare Services, describes the circumstances under which helicopter use will and will not trigger the provisions of EMTALA:

- If a hospital-owned helipad is used by a local EMS as a pickup point for the transport of a patient to another hospital, the provisions of EMTALA will not apply.
- If the flight or EMS crew requests the intervention (examination or treatment) of the hospital's physician, the provisions of EMTALA will most likely apply.

Consequences of Violating EMTALA

First, it is necessary to understand that EMTALA is clearly intended to regulate and apply to hospital and physician conduct and was never intended to apply to the EMS community. The exception to this rule is when an ambulance or helicopter is owned by a hospital. For purposes of EMTALA, a patient is considered to have "come to a hospital" if the ambulance is owned by the hospital and the patient is in the ambulance for purposes of examination and treatment.

If a hospital or physician is found to have violated the provisions of EMTALA, several possible penalties may be imposed. These include the following:

- A hospital may be fined between $25,000 and $50,000 per violation.
- A physician may be fined up to $50,000.
- A hospital or physician may be terminated from the Medicare and Medicaid programs.
- A patient may commence civil litigation against a hospital or physician for injuries arising out of an EMTALA violation.

The fact that a non–hospital-based ambulance service may not be subject to the provisions does not mean that it cannot be held accountable for injuries arising out of an EMTALA transfer. The patient may file a claim for negligence, as described more fully later in this chapter.

EMTALA Case Law

This summary of current EMTALA case law includes implications for interfacility transport. Because more and more cases are being decided, interfacility implications may become more far-reaching. As mentioned, the intention and purpose of EMTALA were to prevent hospitals from "dumping" patients for inability to pay. Because EMTALA is a federal statute, cases are most appropriately filed in federal court, which differs from a medical malpractice action, a form of negligence filed in state court.

There are many EMTALA cases that have bearing on critical care transport. Specific judgments made in particular EMTALA cases include the following:

- EMTALA is not a substitute for malpractice action.
- Emergency stabilization is not long-term.
- The plaintiff has the burden of proof.
- A patient can be transferred, even if in unstable condition, if the benefits of transfer outweigh the risks.
- Stabilization relates to available capabilities.
- The patient must present to the emergency department (ED) to trigger EMTALA.
- The patient must have an emergency condition.
- Transfer by ambulance is not required.
- Radio medical control does not invoke EMTALA.
- The physician has an obligation to "certify" transfer.
- Active labor requires treatment.

These examples of lessons learned from EMTALA cases can give CCTPs an idea of the implications of EMTALA and how this law can play out in the critical care transport setting. The following are examples of the specific judgments in the preceding list.

Transfer Benefits vs Risks

Barris v. County of Los Angeles, 70 Cal. Rptr. 2d 281 (1997). (Note: This case also limits the damages that a plaintiff can collect for personal injury to that available under state law.)

On May 6, 1993, an 18-month-old girl was taken by ambulance to the ED of King/Drew Medical Center. The patient was a member of the Kaiser Foundation Health Plan but was not taken to a Kaiser participating hospital. Instead, she was taken to King/Drew, the nearest emergency medical facility. She was lethargic and had vomiting, diarrhea, difficulty breathing, and a temperature of 106.6°F. The physician in the pediatric ED of King/Drew noted signs and symptoms of sepsis. He contacted a Kaiser physician, who told the King/Drew physician that Kaiser would treat the patient.

Kaiser has a program called Emergency Prospective Review Program that facilitates patient transfer when a member is brought to a non-Kaiser facility for emergency care. At 7:00 PM, Dr Dang, the King/Drew physician, spoke with Dr Thompson, the Kaiser physician, to arrange transfer. Dang expressed concern that certain blood tests should be performed at King/Drew to rule out sepsis but was instructed by Thompson not to do the tests. At 8:00 PM, the patient had a seizure and became lethargic and nonresponsive. At 9:00 PM, she was transferred to Kaiser. Within 15 minutes, she arrested and died.

The plaintiff (the patient's mother) sued Dang and Thompson, the county, and Kaiser for professional negligence and EMTALA violations. Her EMTALA claim was failure to provide appropriate screening and stabilization of patient condition before transfer. The jury returned a verdict for the plaintiff of $1.35 million. The superior court ruled to cap the EMTALA portion of the claim and the professional negligence action. It reduced the award to $250,000, the maximum allowed under state law. The plaintiff appealed, but the court of appeals and the state supreme court affirmed the decision. However, it should be noted that hospitals and physicians could receive a separate penalty under EMTALA of up to $50,000 for each violation.

This case demonstrates that it is not necessarily inappropriate to transfer a patient whose condition has not been stabilized. EMTALA states that a patient can be transferred, even if in unstable condition, if the benefits of transfer outweigh the risks.

"Stabilization" Relates to Available Capabilities

Cherukuri v. Shalala (DHHS), 175 F.3d 466 (6th Cir. 1999).

On September 15, 1991, five patients were brought by ambulance to the ED at Appalachian Regional Hospital (ARH), South Williamson, KY. Two had serious internal injuries and head injuries. The hospital had no trauma center or equipment for monitoring anesthesia during brain surgery and had a long-standing policy of not performing neurosurgery on brain injuries. ARH had always transferred patients with these types of injuries to larger hospitals with the expertise to deal with them.

Dr Cherukuri, a general surgeon, was on call. He arrived and treated the five patients for several hours. On stabilizing their blood pressure and other vital signs, he transferred the two seriously injured victims to a hospital in Huntington, WV. One patient died during the transfer, and the other survived.

The Department of Health and Human Services (DHHS) fined Cherukuri $50,000 for each of the two transfers. This is the largest penalty imposed against a physician in the history of EMTALA. They claimed Cherukuri could have performed surgery and "stabilized" the conditions of the patients because an anesthesiologist was willing to "put the patients to sleep." Cherukuri appealed the decision; DHHS denied the review. He then appealed it to the Sixth Circuit Court of Appeals.

The circuit court dismissed the fine and all EMTALA charges against Cherukuri. In doing so, the court of appeals looked to the Act, which states that a patient whose condition has not been stabilized may be transferred (1) only on "a certification that based upon the information available at the time of transfer, the medical benefits reasonably expected from the provision of appropriate medical treatment at another medical facility outweigh the increased risk to the individual … from effecting the transfer" and (2) only if "the receiving facility… has agreed to accept the transfer of the individual and to provide appropriate medical treatment."

The court found that Cherukuri did not violate the stabilization provision of EMTALA. Stabilization requires an objective standard of reasonableness based on the situation at hand and "requires merely that a hospital stabilize patients within the staff and facilities at the hospital." The patient must be evaluated and, at a minimum, provided with whatever medical support services and/or transfer arrangements that are consistent with the capability of the institution and the well-being of the patient.

Radio Medical Control Does Not Invoke EMTALA

Johnson v. University of Chicago Hospitals, 982 F.2d 230 (5th Cir. 1994).

During February 1990, the defendant hospital (UCH) served as the resource facility for the South Chicago Mobile Intensive Care System. UCH's responsibilities included providing telemetry communications to ambulance paramedics and this, in turn, required UCH to direct paramedics transporting emergency patients to the appropriate hospital in the system. On February 2, the plaintiff, a 1-month-old girl, stopped breathing. Her mother called 9-1-1. Chicago Fire Department paramedics arrived and began treating the infant. Because she remained in cardiac arrest, the paramedics contacted UCH. Although they informed UCH that they were only 5 blocks from UCH, they were instructed to go to a second hospital because UCH was on "partial bypass." She was treated at the second hospital but was subsequently transferred to a third hospital with a pediatric intensive care unit. She died sometime after her transfer to the third hospital.

The mother filed suit against UCH alleging four counts, including violation of EMTALA. The lower court dismissed this count because the plaintiff failed to allege that the infant actually *came to* the emergency department at UCH. The plaintiff appealed, and this court also found that the plaintiff never came to UCH or its emergency department. Nothing in the language of EMTALA—or in the legislative history that has been cited—suggests that an extension of EMTALA would create a duty in a telemetry operator that would be actionable under the present facts. Accordingly, EMTALA liability is not invoked.

Physician Obligation to "Certify" Transfer

Burditt v. U.S. Department of Health and Human Services, 934 F.2d 1362 (5th Cir. 1991).

A mother who was near term with her sixth child arrived in the emergency department of DeTar Hospital at 4:00 PM. She was experiencing 1-minute, moderate contractions every 3 minutes, and her membranes had ruptured. Two nurses examined her and found a dangerously high blood pressure. The mother had received no prenatal care and had no means of payment. One nurse notified Dr Burditt, who was next on the rotating call list. Burditt stated that he "didn't want to take care of this lady" and asked the nurse to prepare the mother for transfer to another hospital, 170 miles away. The two examining nurses notified the nursing supervisor and the hospital administrator of their belief that it was not safe to transport the mother. Burditt called back and was told by an examining nurse that he was legally required to examine her and personally arrange for transfer.

This same nurse asked for an order to administer magnesium sulfate ($MgSO_4$) to prevent seizures. He told her that she could, only if the mother could be transported by ambulance afterward. At 4:50 PM, Burditt arrived and confirmed her blood pressure to be 210/130 mm Hg. He knew there was a strong possibility that the mother's blood pressure would precipitate complications that might kill the mother and the baby and that infants of hypertensive mothers are at higher-than-normal risk of intrauterine growth retardation. He estimated the baby to weigh less than 6 lb—less than normal weight. Still, he obtained acceptance at the second hospital, started $MgSO_4$ treatment, and instructed an examining nurse to arrange the transfer. She attempted to show him the EMTALA regulations, but he refused to read them. The nurse insisted that he sign a Physician Certificate Authorizing Transfer, which he did. He stated that "until DeTar Hospital pays my malpractice insurance, I will pick and choose those patients that I want to treat."

Burditt did not reexamine the mother, although he was there to witness her being wheeled to the ambulance. The mother delivered a healthy baby in the ambulance approximately 40 miles into the 170-mile trip and was taken to a nearby hospital for oxytocin administration to reduce bleeding. While there, the transport nurse telephoned Burditt to inform him of the birth. He instructed her to continue transport. Despite the physician's orders, the accompanying nurse returned to DeTar Hospital, where Burditt refused to see the mother. He directed that the mother be discharged if she was in stable condition and not bleeding excessively. A DeTar official pressed Burditt to allow another doctor to examine the mother. The mother stayed at DeTar for 3 days and left in good health.

DeTar Hospital had executed a Medicare provider agreement and, so, was obligated to treat the mother in accordance with EMTALA. In mid-1988, the Inspector General of the US DHHS demanded a $25,000 civil penalty from Burditt for violations of EMTALA. On appeal, an administrative law judge reduced the fine to $20,000. The Department of Appeals Board upheld the fine. Burditt appealed this decision. Burditt contended that he stabilized the mother's emergency medical condition by administering $MgSO_4$.

The court held that he could not have stabilized the mother's condition unless he provided treatment that medical experts agree would prevent the threatening and severe consequences of the mother's hypertension during transit. Burditt also contended that there was adequate time to safely transfer the mother to the second hospital before she delivered—that she was not in active labor. The court held, based on expert testimony, that this was not so.

Burditt testified that he was completely ignorant of the EMTALA requirement for the Physician Certificate Authorizing Transfer certifying that the benefits outweigh the risks of transfer. He said he signed the certificate just so he could transfer her. The court upheld the board's decision that the certification was so unacceptable that it is unlikely that Burditt actually made the required certification. Finally, Burditt contended that he sent the mother with the appropriate equipment and qualified personnel. The court said that the obstetric nurse and two EMTs were qualified to deliver the baby in the absence of complications. But it is undisputed that they were unqualified to perform a cesarean section or treat the other complications that could have developed as a result of the hypertension.

Responsibilities of CCTPs

The refinement of EMTALA has far-reaching implications that make CCTPs responsible to minimize risks associated with interfacility transport. This chapter examines the role of CCTPs in that setting, the performance of appropriate transfers, and ways to minimize legal risks.

Legal Practice Constraints for CCTPs

Specializing in interfacility transport can be challenging and rewarding. It can be challenging because you most often transport critically ill and injured patients and rewarding because of the satisfaction gained by ensuring that the patient receives excellent care. The downside of any profession, especially professions related to health care, is that the potential for litigation looms over each decision. The provision of emergency care, no matter what the setting, is governed by civil law—as opposed to criminal law **Table 2-1**—and consists of laws (statutes) and regulations originating from a number of legal sources **Table 2-2**.

TABLE 2-1 Law Types

Type	Basis
Civil	The law of civil or private rights as opposed to criminal law; two major areas—torts (civil wrongs) and contract law
Criminal	The body of law that defines offenses against society at large

Laws are usually general and include things such as the **scope of practice** and definitions of EMS provider "classes." Regulations, which are more specific, govern the types of procedures that can be performed in certain circumstances, the training required for initial licensure or certification, and the requirements for relicensure or recertification.

CCTPs can be sued by anyone, even if they perform appropriately. However, the burden of proof is on the person bringing the lawsuit (the plaintiff). Defending yourself in litigation can be expensive. Therefore, be certain to review your employer's malpractice insurance policy to ensure that the coverage extends to interfacility transports. Most states require employers to provide certain minimum amounts of malpractice insurance coverage for employees and volunteers of the organization. Most likely, your employer will provide malpractice insurance to cover CCTP actions *within the scope of employment;* these policies will not cover emergencies that you respond to on your own time. However, these policies usually are written to protect the interests of the employer (because they pay the insurance premiums). In all probability, this employer-provided insurance is more than sufficient to cover your actions within your work.

On the other hand, if you have your own insurance, it will frequently provide an attorney to represent you and your interests. A problem may arise if your insurance company and your employer's insurance company both provide attorneys and the two disagree about how to defend the case. This could create a situation in which you and your employer try to put the focus of liability on each other.

Whatever the decision, to obtain your own insurance or rely on the employer's, educate yourself on your state's requirements for malpractice insurance, your employer's policies about malpractice, and your risks.

Functioning as a CCTP requires special skills and knowledge. CCTPs also have significant legal responsibility to patients, the medical director, and the public. The scope of practice for CCTPs is limited

to skills taught in the program that resulted in advanced-level licensure or certification and any additional skills defined by state law or regulation. Additional skills may be incorporated into the scope of practice through protocols and standing orders. Exceeding the scope of practice (performing skills that you may know how to do but are not permitted by law or regulation), even if it would help the patient, could result in litigation and loss of licensure or certification.

TABLE 2-2 Law Sources

Source	Description
Constitutional	Governmental (federal or state) law based on the constitution
Statutes	Codifications established by government (Congress and the state legislatures)
Regulations	Rules or orders having the force of law issued by an executive authority of government; agencies created by state government often empowered to enact regulations (such as state EMS offices)
Common law	Law derived from judicial decisions rather than from statutes or the constitution

Figure 2-1 Transport practice takes place under the supervision of a medical director.

CCTPs need **medical direction** to practice. Medical direction occurs two different ways. **Online medical control** occurs by telephone or radio, and **off-line medical control** is in the form of approved standing orders or protocols. The ultimate responsibility for the action or inaction of a CCTP rests with the medical director **Figure 2-1**.

Finally, CCTPs have significant legal and ethical responsibility to the public to function competently. Regular skill performance, ranging from maintenance following the initial licensure or certification period to expert mastery of the skill, helps meet this responsibility. Attending continuing education and refresher programs helps ensure maintenance of adequate skill levels. Participation in case review sessions, to identify methods of improving patient assessment, treatment, and team communications, is another important component of practice.

The Patient's Rights

Several general areas of liability can arise in the provision of transport, most of which stem from violation of the patient's basic rights. These violations include assault, battery, false imprisonment, and

negligence.

The Right to Establish Advance Directives or Do Not Resuscitate Orders

To withhold resuscitative efforts, you must have a written order from a physician. If there is a question about the status of a **do not resuscitate (DNR) order**, always err on the side of providing resuscitation rather than withholding it. State laws regarding DNR orders vary substantially. Be familiar with the laws in your state.

The Right to Self-Determination

The right to refuse treatment, a basic right of all patients, is based on a fundamental concept: every person of adult age and sound mind has a right to determine what is done or not done to his or her body. Providing care without this **consent** puts CCTPs at risk of liability for **assault** or **battery**, even if the care is necessary to save the patient's life.

Figure 2-2 Explaining the need for treatment and the risks of refusal may persuade the patient to consent.

If the patient refuses care, you must explain the possible consequences. Patients who refuse treatment may be afraid or in emotional distress. Attempting to calm them with rational explanations and reassurance may lead to consent. If the patient persists in refusing treatment, follow local protocol, which will include thorough documentation and, possibly, contacting **medical control**.

To minimize legal risk, explain to the patient what may be wrong, the treatment that is indicated and why, and the consequences that may result without the treatment. Urge the patient to obtain medical care with a personal physician. When possible, encourage a family member to stay with the patient until the patient agrees to seek care or the condition resolves **Figure 2-2**. Attempt to obtain signatures on the EMS record from the patient and witnesses verifying that the patient is refusing care or transport. Thorough documentation should include the patient's history, your physical findings (including indications of the patient's mental **competence**), the stated reasons for refusing treatment, and any explanations or advice you have given to the patient or family members.

The patient also has the right to terminate treatment at any time—before or after transport is initiated. To refuse treatment, the patient must be mentally competent and of legal age. Refusal can be expressed verbally or nonverbally. Examples of nonverbal refusal are pulling away, making gestures, and using the hands to bar your access to equipment or the patient's body. However, you should still ask if the patient is actually refusing care.

Transporting a patient against his or her will can lead to **tort** actions for **false imprisonment**, assault, and/or battery. In some circumstances, criminal charges could result, although this situation is

rare. Give special consideration to patients who may be impaired by the influence of alcohol or drugs, may exhibit suicidal tendencies, may have signs of a head injury, or are not behaving normally according to family members because they may not be able (competent) to make rational decisions. In these situations, seek assistance from hospital personnel and online medical direction. In general, restraint and transport against a patient's will constitute a rare circumstance that could occur after a patient had been determined not competent to make health care decisions. If usual resources cannot or will not assist, carefully consider the potential liability for false imprisonment, balanced against the medical necessity of treatment and the risk that the patient may harm himself or others.

Authority to consent for or refuse treatment for a minor (even against the minor's wishes) rests with the custodial parent or legal guardian. If the parent or legal guardian refuses care, then proceed as you would with an adult patient who refuses care. Several states have enacted laws that mandate reporting any case in which a parent or legal guardian refuses emergency care for a minor. In that case, a court may order care to be provided, require verification that it has been given, or order an investigation to determine whether a child is still in need of treatment.

The Right to Consent

Consent must be obtained before proceeding with treatment or transport. Consent can be express or implied, and the rules for minors vary in cases of emancipation.

Informed (Express) Consent

To consent to treatment, the patient must be of legal age and sound mind. In some states, consent is not informed unless the patient is given the following information: (1) the procedure to be performed, (2) risks of the procedure, (3) any alternatives to the procedure, (4) risks of the alternatives, and (5) likely outcomes or risks if the procedure is not performed.

In general, consent must be obtained for any medical treatment, including transport. For consent to be effective, it must be informed. However, in some situations, a CCTP may provide treatment and transport under the doctrine of implied consent (discussed next). The legal responsibility for obtaining **informed (express) consent** most often rests with the physician. Most procedures associated with transport do not require an informed consent discussion with the physician, but the consent must be obtained, or health care personnel providing treatment are at risk of civil and/or criminal actions. The act of transferring a patient in unstable condition to a specialty center may require (by EMTALA) a discussion between the physician and the patient and written documentation of the risk–benefit analysis of the transfer or the patient's request for transfer.

Implied Consent

Implied consent is an exception to the rule of informed consent. Implied consent is the assumption by law that a patient who is unconscious or for some reason unable to express consent would want to receive lifesaving treatment **Figure 2-3**. Implied consent has been extended to include children who have a life-threatening condition, when the parent or legal guardian is not available. Some states also consider implied consent to apply to situations in which the patient is mentally ill, emotionally disturbed, or developmentally disabled. To implement the doctrine of implied consent, the patient must have a true emergency condition—a significant risk of death or the possibility of serious injury or illness.

Figure 2-3 The unconscious patient is considered to give implied consent to treatment and transport.

Special Populations

Although legislation varies in different areas, in most states, a pregnant teenager is "emancipated" during her pregnancy and can make all legal and medical decisions for herself and her unborn baby. However, the minute she delivers the baby, she once again assumes "minor" status, and her parents or legal guardians make all of her medical and legal decisions. She does, however, remain the legal guardian for her baby and can make all medical and legal decisions for her child (even though she cannot make them for herself).

Emancipation

One additional consideration must be given to a nuance in some states' laws—obtaining consent for treatment from emancipated minors. A minor child is emancipated by decree in a court of law. **Emancipation** can be sought by the child or the parent. The child gives up the right to maintenance and support from the parent, and the parent (or guardian) relinquishes the right to control the child. Minors are often emancipated because they get married. The same may hold true for a minor child who becomes pregnant, although pregnancy in and of itself does not constitute emancipation. State law may allow an emancipated minor to give consent for treatment or to refuse it. Emancipation may also occur "by operation of law." This means that certain events or actions can automatically result in emancipation without petitioning the court. For example, in some states, the marriage of minors automatically emancipates them. Furthermore, in some states, service in the armed forces also automatically emancipates minors.

The Right to Receive Care Without Interruption

The patient has the right to receive uninterrupted care. A CCTP could be held liable for **abandonment** if care is interrupted. Some examples of abandonment include the following:

- Initiating care and then leaving the patient or turning the patient over to someone who does not have sufficient training to provide care in the current situation.
- Leaving the patient unattended for a brief period, if the condition worsens or the patient sustains additional injury.
- Failing to transfer information to the receiving facility regarding the patient's history, treatment, or current condition, if the failure results in further injury or harm.

- Failing to respond to or complete an ambulance call.
- Failing to immediately report one's inability to complete an emergency response because of equipment failure or one's own health status.

The Right to Appropriate Care

The patient has a right to appropriate care. "Appropriate" means that you are expected to have the same training, skills, and ability as other CCTPs and are expected to exercise reasonable care and diligence in applying the skills and knowledge in caring for patients. **Negligence** results when a CCTP fails to meet this standard of care. To prove negligence, the plaintiff must prove all four elements of the claim:

1. CCTPs have a **duty to act** when they or their employer have incurred a legal obligation to provide patient care. This duty can be established formally, when the transport service has contracted to provide emergency care to the citizens of a given geographic region. It can also be established when a CCTP initiates treatment and when the sending facility calls for a transport vehicle and the dispatch center confirms that it will be sent. States have different rules about other actions or situations that establish duty. One example is whether an off-duty CCTP (or other health care professional) has an obligation to treat someone. The majority rule is that an off-duty CCTP has no duty to stop and treat someone in need; however, a CCTP who begins to provide aid is obligated to ensure appropriate care without interruption.
2. A **breach of duty to act** can be proven by offering evidence that a CCTP did not conform to the standard of care by providing inappropriate care, failing to act, or acting beyond the scope of practice. A CCTP must act as a reasonable, prudent CCTP would in the same or similar circumstances.
3. **Damage** must result from the breach of duty. All states recognize physical injury resulting from a breach of duty as damage. However, there are differences in the way states treat psychological injuries. Many states do not recognize psychological injuries or recognize them only in limited circumstances.
4. The **proximate (direct) cause** of the injury must have resulted from a CCTP's actions or failure to act. For example, imagine that a cardiac patient who is being transported sustains injuries in an ambulance crash. If the driver of the ambulance is found to be at fault, the plaintiff will most likely be able to prove that the crash was the proximate cause of the injuries.

Some situations may exist in which there is no legal duty to act but in which an ethical duty is imposed. In some states, EMS personnel are not obligated to stop and provide assistance at the scene of a motor vehicle crash. However, there is a more compelling argument about the ethical duty to do so if the EMS personnel are in an ambulance and observe a crash.

The Right to Confidentiality and the HIPAA Privacy Rule

The patient has a right to expect that any information obtained in preparation for, during, or after transport will be kept confidential. Legal protection for this expectation was given with the implementation of the **Health Insurance Portability and Accountability Act (HIPAA)** of 1996. CCTPs must be especially familiar with the Privacy Rule portion. The HIPAA Privacy Rule obligates health care personnel to ensure that confidentiality of patient information is maintained. HIPAA is a federal law that was implemented by Congress and stemmed from concerns about the following:

- Electronic transmission of information
- Information crossing state lines with different laws

- Reports of violations of privacy in certain industries
- The volume of information available

The primary goals of HIPAA are to create consistent standards for protection of patient privacy and to increase patient rights and inform them of their rights while allowing state laws to be more protective. As a general rule, HIPAA applies to "covered entities," which include health care providers who transmit information electronically, health plans, and health care clearinghouses. It requires enhanced security and privacy protection for **protected health information (PHI)**, establishment of patient rights and use of a privacy notice, implementation of policies and procedures, discipline for breaches and violations, and training for all personnel of the covered entity.

For EMS providers, the most important aspects of HIPAA are related to disclosures of PHI. In most states, the state law is more stringent than federal law. Federal law allows state law to control when the requirements are the same or more stringent. Therefore, most cases that would violate HIPAA are brought under state law. In many cases, state civil and criminal sanctions are more severe than those under federal law.

There are potential legal consequences for violating HIPAA, including civil penalties (fines) of up to $250,000, criminal penalties, and personal liability. A covered entity is responsible for its own personnel. When its employees or agents violate HIPAA, the covered entity must take appropriate disciplinary action such as providing additional training, formal warning, suspension, or even termination. In addition, violation can result in the patient filing a complaint to the Office of Civil Rights and subsequent investigation.

The Privacy Rule protects the confidentiality of PHI. PHI includes individually identifiable information (such as name, social security number, and date of birth), health information (such as laboratory results and medical history), and demographic information (such as address and telephone number). It is important to note that HIPAA protects PHI in *any* form whether it is written, verbal, or electronic. A criminal violation occurs when a provider knowingly discloses PHI for a purpose not permitted under HIPAA and with the intent to use the PHI for personal gain. Note the "knowingly" standard for criminal prosecutions. This means that inadvertent disclosure will not bring criminal sanctions; however, civil sanctions may still result.

There are situations when it is permissible to disclose PHI. Disclosure can be made without written authorization of the patient or legal representative for purposes of treatment, payment, and other health care operations. Disclosure for treatment purposes includes situations in which PHI is shared in the process of providing health care, coordinating health care among providers, referring a patient from one provider to another, and coordinating a patient's care or other services with third parties. Disclosure of PHI is also permitted (without specific written authorization) when dealing with payment issues, including determining coverage of health benefit claims, billing, claims processing, reviewing health care services (such as medical necessity, coverage, and appropriateness), and when a covered entity is undertaking utilization review activities. Finally, a covered entity may disclose PHI in the course of performing other health care operations, such as quality assessment and improvement, evaluating performance of its health care providers, and performing general administrative functions such as conducting audits.

Other than for treatment, payment, and other health care operations, a covered entity may disclose PHI when the patient or the patient's legal representative provides authorization, in response to an *appropriately executed* court document compelling release of the PHI (such as a court order) or when required by law (such as to report child abuse). Depending on state law, a patient's legal representative may include a custodial parent of a child, a family member or next of kin, a legal guardian, or a person with medical power of attorney. There are other situations in which PHI may be disclosed but may be

impacted by variances in state law, such as when it is required by state law (eg, elder abuse or a wound resulting from violence), is pursuant to a state health oversight function, involves activities related to organ donation, and is for the purpose of averting threats to public health or safety.

HIPAA Case Law

The following is an example of a civil case related to EMS that violated HIPAA and that was brought under state law.

Pachowitz v. LeDoux, 2003 WL 21221823 (Wis. App., May 28, 2003) decided under Wis. Stat. § 895.50.

On April 21, 2000, four EMS personnel, including volunteer EMT Katherina LeDoux, responded to a 9-1-1 call at the Pachowitz residence for a possible overdose. The patient, Julie Pachowitz, was transported to Waukesha Memorial Hospital at her husband's request. LeDoux had not met Pachowitz before.

Approximately 2 weeks earlier, LeDoux had learned from her friend, Sally Slocomb, that Pachowitz, a work colleague, had a medical condition. After the emergency call, LeDoux, believing that Slocomb and Pachowitz were close friends, spoke to Slocomb about having transported Pachowitz. LeDoux testified that she called Slocomb because she was concerned about Pachowitz and thought Slocomb could help Pachowitz. Slocomb then drove to West Allis Memorial Hospital where she and Pachowitz worked. There, she discussed Pachowitz's emergency call with staff.

Pachowitz later filed suit against LeDoux and was awarded $3,000 in compensatory damages and $30,460 in attorney fees.

The Patient's Fundamental Rights

Under the HIPAA Privacy Rule, patients have five fundamental rights that are briefly but separately addressed:

1. Patients have the right to inspect and copy their medical records. Patients have the right to inspect their medical records, although the covered entity can require sufficient notice to make it available and, in many situations that vary with state law, charge patients for copies of their record.
2. Patients have a right to request restrictions to what is disclosed when the covered entity releases PHI. There are situations in which the covered entity does not necessarily need to honor this request but should consider doing so. If the covered entity grants this request, there are other requirements for compliance with the HIPAA Privacy Rule.
3. Patients have a right to request that amendments be made to their medical records; however, there are specific requirements for this process and rules that apply when the request is granted.
4. Patients have a right to request confidential communications. Patients can, for example, request that information regarding their care be sent to an alternative location other than their home address.
5. Patients have a right to request an accounting of disclosures. This request requires the covered entity to track disclosures of PHI that are made and unrelated to treatment, payment, or other health care operations.

A covered entity has specific responsibilities to comply with the HIPAA Privacy Rule. These include the following:

- Ensuring that the patient or legal representative is offered a copy of the organization's Notice of Privacy Practices at the first patient encounter. The notice must be offered once. Some EMS agencies have adopted a practice of simply mailing a copy of the notice to patients following transport if they are unable to indicate their desire to receive the notice owing to illness or injury.

- Training staff (and students affiliated with the organization) in the Privacy Rule.
- Developing and implementing policies and procedures to ensure compliance.
- Monitoring compliance of its employees and agents.
- Responding to concerns or complaints.
- Providing appropriate discipline for violations.
- Retaining HIPAA-related records for at least 6 years.

There are several practical aspects for ensuring compliance with the HIPAA Privacy Rule. For example, CCTPs should ensure that telephone communications are not overheard, especially when discussing PHI. With fax communications, CCTPs should confirm the appropriate fax number before sending a document, include a fax cover sheet that clearly states the name of the intended recipient, and confirm that the fax is received by the appropriate person.

In honoring a patient's right to confidentiality, CCTPs should ensure that the patient or a legal representative signs a written authorization before any information can be released. If confidential information is released without authorization, the CCTP could be sued if the release results in injury to the patient's character, name, or reputation by false or malicious writings (libel) or by spoken words (slander), although laws regarding possible legal actions for breach of confidentiality vary from state to state. To minimize the risk of breaching confidentiality, CCTPs should relay communications about a patient's condition only to appropriate personnel and not release patient-identifying information over the radio.

Transport Preparation

Care History and Pretransport Condition

To prepare for transport, find out the nature of the transport (the patient's diagnosis and condition). Some agencies have established protocols that address the types of situations in which patients can be transported and the level of care provider needed in each situation. Once a patient has been accepted for transport, work efficiently to minimize the time spent and procedures performed at the sending facility. Before initiating transport, ensure the following:

1. The patient has a patent and secured airway.
2. The patient has patent and secured IV access, when indicated.
3. Appropriate measures are instituted to treat hemodynamic instability.
4. Chemical or physical restraint is used for confused or agitated patients who pose a threat to themselves or transfer personnel.
5. Appropriate monitoring is used as indicated, including cardiac rhythm, blood pressure, pulse oximetry, and end-tidal carbon dioxide.

Trauma patients should have appropriate spinal immobilization as indicated, including a long backboard, a cervical collar, and a cervical spine immobilization device.

Communication With the Health Care Team

When preparing for a transport, you should make every effort to communicate effectively, professionally, and succinctly with other members of the health care team. In some rare cases, conflict may arise between a CCTP and the sending physician regarding the interventions needed before transport. If this happens, state your concerns clearly and professionally. If there is still disagreement, contact the receiving

facility's physician to attempt a cooperative resolution. If the situation cannot be resolved, contact the critical care transport agency's medical director for assistance. The ultimate step may be to refuse to transport, but this step should be carefully considered. Although it may involve substantial additional legal risk, one other alternative is to begin transport and then consult with online medical direction about the most appropriate interventions.

Some requests for transport originate in a physician's office or clinic. The physician in this setting may not be familiar with local protocols. In this situation, thoroughly communicate capabilities and limitations to the sending physician. A plan of care must be developed that is within your scope of practice. If the physician is unwilling to do this, politely insist that the sending physician accompany the patient to the receiving facility. If this is not acceptable to the sending physician, proceed as outlined in the preceding paragraph or contact medical control for assistance in resolving the conflict. Consider having the sending physician speak directly with medical control to develop a plan of care for transport.

A patient's ability or inability to pay for the transport can also present a difficult situation for the transport agency. If the agency insists on payment without regard to clinical condition, awkward situations can arise but can be mitigated if the agency policy to refuse transport for people unable to pay is *clearly communicated in advance* to the sending facility. If a CCTP learns of an inability to pay only after arriving at the sending facility, refusing the transport can substantially increase the transport agency's liability risk, especially if adverse outcomes result from the delay in securing an alternative means of transport. Also, the agency may experience problems with its reputation in the community. Communicating clearly and concisely before agreeing to transport can help avoid this situation.

Transfer Preparation

In preparation for transport, obtain the pertinent patient information, including the following:

- The reason for transfer
- The name of the receiving hospital and/or physician
- Whether a BLS or an ALS provider must accompany the patient, in addition to the CCTP, and whether the sending facility plans to provide an escort
- Treatment provided at the sending facility
- Medical treatment and drug orders for the duration of the transfer
- Complications to anticipate

In addition, obtaining the name of the receiving physician before transport will serve to avoid unnecessary delay, embarrassment, and potential conflict at the receiving facility. There must be physician-to-physician contact before the transport is initiated.

Review the transport orders for treatment *before* leaving the sending facility. Ensure that the orders do not exceed your scope of practice. If they do, talk with the sending physician to modify the plan of care. If the plan cannot be modified, the sending facility must provide additional personnel to accompany you and facilitate the provision of needed care.

Patient Information

EMTALA requires that certain patient information be obtained from the sending facility and provided to the receiving facility. Although you can take reports from ancillary personnel (such as the nurse assigned to arrange transfer), you should also speak with the sending physician about the information needed.

Under EMTALA, the sending physician must send copies of medical records with the patient to the receiving facility. Failure to do so constitutes an EMTALA violation and can result in fines that include the cost of performing the tests required to obtain the information that was not provided. The sending

physician must provide the following documentation:

1. Pertinent records of the patient's emergency condition
2. The patient's signs or symptoms
3. A current diagnosis and any treatment provided
4. The results of any tests that were conducted (including laboratory tests and radiographs)
5. Written consent of the patient or a person with legal authority agreeing to the transfer
6. Verification from the sending physician that the benefits of transferring the patient outweigh the risks associated with the transfer

In addition, the sending physician must provide samples of pertinent laboratory specimens (such as peritoneal lavage fluid, blood cultures, or cerebrospinal fluid). Patient report at the time of transfer should address the current condition: subjective symptoms, especially the chief complaint; objective signs such as lung sounds and vital signs; an assessment of the patient's physical status, including a preliminary diagnosis; treatment provided at the sending facility; and a plan of care to be provided during transport. Finally, necessary demographic information includes name, age, sex, race, address, billing information, next of kin, and telephone number.

A special case occurs when transferring a patient in unstable condition. Federal law does not regulate the transfer of patients once their condition has been stabilized. Conversely, EMTALA allows transfer of patients in unstable condition only when certain conditions are met. The transfer of a patient in unstable condition for medical reasons (specialty services available at the receiving hospital but not at the sending hospital) requires the patient's written request for transfer following informed consent *or* certification by the physician, based on the information available at the time of transfer, that the medical benefits of transfer to the receiving facility outweigh the risks of transport. Obtaining the patient's written request for transfer may significantly protect CCTPs and the agency against liability. The previously listed requirements for the receiving facility and documentation also apply. Another important specific case involves the transfer of a pregnant woman with contractions. EMTALA specifies that stabilization is defined as delivery of the baby. Thus, any pregnant woman with contractions is considered in unstable condition for transfer purposes, and the aforementioned documentation requirements apply.

Patient Assessment

Before initiating transport, you should perform a rapid baseline physical examination to determine any changes in the patient's condition and whether the transfer warrants additional personnel or resources **Figure 2-4**. This assessment consists of a brief review of the ABCs and the cardiovascular, neurologic, gastrointestinal, and genitourinary systems. (Chapter 5 discusses patient assessment in more detail.) For example, ensure that lung sounds are present equally bilaterally in an intubated patient before and during transport. If the physical examination reveals a problem, seek a solution as outlined in the *Communication With the Health Care Team* section presented earlier in this chapter.

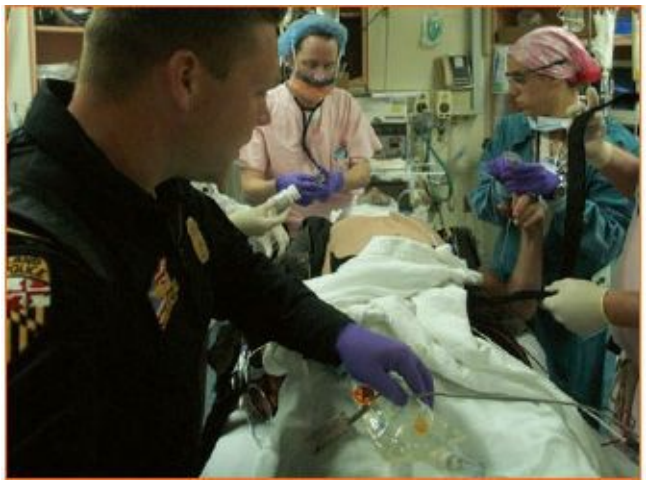

Figure 2-4 A brief physical examination before transport may reveal changes in the patient's condition or the need for further personnel.

Transport Responsibilities

Medical Direction

During transport, CCTPs usually operate with off-line medical direction using established written protocols. If treatment beyond protocol is indicated or consultation is needed, establish contact with medical control and proceed under online medical direction. Ensure the availability of online medical direction before transport. Also confirm that the sending and receiving physicians understand who will provide medical direction and that this may necessitate alteration in the established plan of care. Distance ranges of communications equipment may require shared responsibility between the sending and receiving physicians or even with the transport agency's medical director.

In determining who is most appropriate to provide medical direction, a primary criterion is communication capabilities. If only one physician can maintain direct communication with the vehicle during the transport, that physician should perform medical direction. If more than one physician has communication capabilities, it is appropriate to use one agreed on by the sending *and* receiving physicians. Protocols should include appropriate actions for cases in which communication with the established medical director is not possible. When continuous communications are not available, transport orders should enable you to respond appropriately to medical crises and changing patient status. Standing orders or protocols may be developed to meet these needs.

CCTPs may need to remind the sending physician that prehospital protocols for EMS systems do not apply automatically in the interfacility transport environment. Prehospital protocols may be limited to use in specified environments by personnel who are described under state rules for the EMS system; hospital personnel who accompany the patient usually do not fall under these protocols and often are not familiar with them. When sending physicians write transport orders, they should designate the protocols by name in the transport orders, write pertinent sections into the orders, or attach a printed copy.

Personnel Qualifications

EMTALA requires that the sending physician designate the qualifications of personnel accompanying the patient during transport. EMTALA does not specify what constitutes "qualified personnel," and government regulations have not been developed to define "qualified." The Centers for Medicare and Medicaid Services is responsible for enforcing EMTALA sanctions that involve suspension or termination of Medicare participation. The Office of the Inspector General of the DHHS is responsible for assessing civil monetary penalties for EMTALA violations. These agencies have enforced EMTALA

in a way that affects transport personnel. Personnel who accompany the patient must be medically trained and certified, as required by law, at a level that will allow performance of whatever procedure *might* be necessary to support the patient during transfer, including medical procedures required not only for the patient's current condition, but also for any *reasonably foreseeable* complications.

To determine the level of personnel necessary to care for the patient and manage specialized equipment during transport, the physician should consider that personnel aboard local transport vehicles (ground and air) may be trained in a variety of skill levels, ranging from EMT to paramedic. Some systems also staff transport vehicles with nurses and physicians. For these ambulance personnel, state statute or regulation clearly and narrowly defines the scope of practice. In addition, the personnel will be functioning under medical direction that provides protocols, including standing orders, for prehospital providers within their system. As part of the advance preparation and policy development process, physicians should learn the certification levels of EMS personnel used to staff public and private transport vehicles in the community. It is also important that all necessary and appropriate equipment, medications, and supplies that are within the scope of practice of the personnel onboard and that may be required by the patient be available in sufficient quantities. This is the responsibility of the CCTP.

If the patient's current condition and all reasonably foreseeable complications can be managed within the licensure, training, and capability of the crew on the transfer vehicle, the patient may be transferred without the provision of ancillary personnel. When faced with the need to provide care beyond your scope of practice, request additional personnel.

Ancillary personnel may provide specialized skills (such as a balloon pump technician or a credentialed registered nurse) or may assume overall responsibility for care (such as a neonatologist for a neonatal intensive care patient). Be aware that when ancillary personnel assume ultimate responsibility for the care of the patient, you should provide support to them as needed. You have less liability when care is assumed by another provider with more advanced training and relatively more risk when the ancillary crew is technicians, but in both situations, the risk of liability will undoubtedly be shared because a plaintiff tends to name as many defendants in a lawsuit as possible (to maximize collection efforts). Although CCTPs can be held liable, the primary responsibility for patient appropriateness and stability for transfer rests with the sending physician and facility.

If the sending facility provides ancillary personnel and special equipment, it is that facility's responsibility to maintain the special equipment and provide for its return.

Actions During Transport

To minimize risk of liability during a transport, CCTPs must assess the patient, discuss potential complications with the sending physician, and ensure that the sending physician understands the capabilities and limitations of the transport team. During transport, CCTPs provide appropriate care within the scope of practice and inform medical control or the receiving facility of changes in the patient's condition or any extenuating circumstances **Figure 2-5**. During transport, CCTPs do the following, as indicated:

1. Provide continued support of respiratory and circulatory systems
2. Provide continued fluid volume replacement (and blood, if trained in transfusions)
3. Use appropriate medications as ordered by the medical physician or authorized by written protocol; any medications not normally carried must be obtained before the transfer, and their use specified in transfer orders, including whether they should be administered by a registered nurse or paramedic, if they are not included in protocols.
4. Monitor vital signs

5. Maintain communications
6. Maintain accurate records of the transport

Transport Completion

On arrival at the receiving facility, ensure continuity of care by giving a report to the designated receiving care provider. The report should include the following: (1) the patient's name, age, and sex; (2) the names of sending physician and facility; (3) the reason for transfer; (4) a brief review of systems; (5) the patient's history; (6) medications administered; (7) fluid intake; and (8) a summary of the patient's condition during and tolerance of the transport in a clear, concise, and logical format. Transfer specimens, records, and laboratory and radiography results to the receiving provider, and make yourself available to answer questions after the transfer of care.

Figure 2-5 During transport, monitor the patient's condition, give care as needed, and maintain thorough documentation.

Documentation

All health care personnel are taught the importance of documentation. Nowhere is accurate and complete documentation more important than when dealing with a patient who is seriously ill or in critical condition. When a patient's status changes rapidly and without warning, numerous interventions are carried out and transfer orders may have to be modified during transport. It is often a challenge to keep up with documentation during a transfer that requires your constant attention to the patient.

Each service uses its own forms and reports to document patient care, but regardless of the forms used, it is essential that a completed run report paint an accurate and detailed picture of everything that occurred during the trip. Providers should keep in mind that they may be asked to defend the run report in a court of law.

In the critical care transport setting in which transfer often involves a team effort, it is important for team members to understand who has the responsibility for each aspect of documentation. When dealing with an acutely ill patient, it is easy to assume that the other team member may have documented an intervention or a set of vital signs only to later learn that the documentation was not included in either report. Team members should work together at the conclusion of a run to ensure that documentation is accurate and complete.

It is particularly important to record certain essential elements of every transfer. These include the

following:

- The times of each set of vital signs and each assessment finding
- The details and times of each intervention (such as drug doses and routes of administration)
- Patient responses to all interventions
- Untoward or adverse reactions
- Communications with medical control
- Deviation from transfer orders or standing orders
- Diversion to another hospital
- Refusal of treatment by the patient

If it is necessary to amend a run report following its submission to the receiving facility, make sure that the amendment is done in accordance with local protocol.

Summary

The first line of protection against legal problems lies in maintaining a professional and compassionate demeanor. Treat patients, family members, and personnel at the sending and receiving facilities with respect, and cooperate fully with other agencies to ensure continuity of care. Make it clear that you care about the patient's well-being and are doing everything possible to serve it.

The second is the discipline that produces medical excellence. Important factors include preparing thoroughly for transports, ensuring the presence of adequate and functional equipment, maintaining skills and certifications, knowing and complying with protocols, staying within the standard of care for your level of training, and documenting the transport thoroughly.

Knowing and complying with laws and regulations regarding critical care transport constitute the final step. Local laws vary, but general legal principles such as consent and negligence apply in all jurisdictions. Likewise, all transport providers are subject to, and should clearly understand, EMTALA.

Case Study

YOU AND YOUR CRITICAL CARE TRANSPORT TEAM have been requested to provide an interfacility transport from a rural hospital. The patient is a 28-year-old woman who is 29 weeks' pregnant. She presented to the local emergency department around 10:30 PM complaining of labor-type pain every 10 minutes. Emergency department staff placed the patient on a fetal monitor and confirmed their suspicions of preterm labor. The fetal monitor showed normal variability with no decelerations. The patient received a 0.25-mg subcutaneous injection of terbutaline.

When you and your critical care transport team arrive, you find the patient in the emergency department. She has responded to the terbutaline and her uterine contractions have slowed to 15 minutes apart and are less intense. The physician reports to you and your team that the fetal monitor has shown normal variability with no decelerations. You assess the patient and prepare her for transport, when you realize that the transport fetal monitor was accidentally left in the ambulance. Since the transport time was only 20 minutes, you decide not to go to the ambulance to retrieve the fetal monitor and plan to place the patient on the fetal monitor once you reach the ambulance.

During the short transfer time to the ambulance, the patient begins experiencing an increase in the contraction frequency. You load the patient into the ambulance and begin your transport. The patient's contractions have increased to every 5 minutes. Your critical care protocols state to administer magnesium sulfate, with the initial loading dose of 4 to 6 g IV over 20 minutes, followed by a

maintenance drip of 1 to 4 g/h. While you are mixing the loading dose, the patient is screaming and crying hysterically in pain. Your partner is attempting to calm her while you prepare the drip.

You place the magnesium sulfate on the transport drip. The transport pump beeps, indicating that there is air in the line. You disconnect the drip, flush the line, and reattach it to the pump. The pump beeps again, indicating air in the line. You again disconnect the line in attempts to flush it. The patient continues to scream in pain and her contractions continue to increase in frequency. You once again flush and reattach the line; the pump gives the green light indicating that it is functioning. Your ETA to the hospital is 5 minutes. You and your team reassess the patient and determine that the contractions are slowing in frequency. As you and your team arrive at the receiving hospital, you realize that you had forgotten to attach the fetal monitor during the commotion. You quickly attach the fetal monitor without reviewing the strip. You unload the patient from the ambulance and transfer her to the obstetrics (OB) unit. You report to the staff that the patient was administered magnesium sulfate because of an increase in her contractions.

You and your team disconnect the patient from your equipment and transfer the patient to the hospital bed. The OB nurse attaches the patient to the hospital fetal monitor and becomes concerned with the fetal monitoring strip. She immediately calls for a physician. The fetal strip was showing late deceleration. The nurse and physician asked to see the previous strip during the transport. You report that you had forgotten to place the patient on the monitor.

The patient is rushed to the operating room for immediate surgery. The infant was delivered with an Apgar score of 0, and required full resuscitation efforts. The infant did not survive the intense resuscitation efforts.

1. Was the critical care transport team responsible for the infant's outcome?
2. Is this a case of negligence?
3. What has to be proven for a negligence lawsuit?
4. What should have been done to prevent this outcome?

Analysis

The symptoms of preterm labor include a contraction every 10 minutes or more, frequent pelvic pressure, lower back pain, and abdominal cramping. This patient was indeed in preterm labor.

Terbutaline is used to relax the smooth muscles of the uterus. Terbutaline is not approved by the Food and Drug Administration (FDA) for use in preterm labor; however, it is widely used. Terbutaline is only approved by the FDA for the treatment of asthma and breathing problems.

In this case, the critical care transport team was responsible for the infant's death. The transport team's failure to monitor the fetus proved negligence on their part. In order for negligence to be proven, the plaintiff is required to prove the following: The critical care transport team had a duty to act, the team violated this duty to act, and the violation of conduct caused the injury or death of the infant.

The critical care transport team should have returned to the emergency department to stabilize, reassess, and ensure that all appropriate monitors were in place. By performing these simple steps, the situation would likely have resulted in a positive outcome.

Prep Kit

Ready for Review

- CCTPs should understand and comply with the legal principles and specific laws and regulations that govern their practice, and they should give attention to the various elements that contribute to the

quality of care they provide, namely, skills maintenance, continuing education, protocol familiarity and compliance, and interpersonal communications.

- The transport of critically ill or injured patients is a complex process requiring cooperation from everyone involved. Hospitals that participate in Medicare reimbursement programs are subject to EMTALA, which is intended to regulate hospitals and the manner in which they conduct transfers. Personnel who perform interfacility transports need to be familiar with EMTALA and how it applies to them.
- Hospitals or physicians found in violation of the EMTALA provisions face several possible penalties, including fines, termination from Medicare and Medicaid programs, and civil litigation against the hospital and/or physician for injuries arising from an EMTALA violation.
- Current EMTALA case law includes implications for interfacility transport, with the intention and purpose of preventing hospitals from "dumping" patients because of inability to pay. There are many EMTALA cases that have bearing on critical care transport, and as more cases are decided, interfacility implications may become more far-reaching.
- EMTALA has far-reaching implications that make CCTPs responsible to minimize the risks associated with interfacility transport.
- The provision of emergency care, regardless of the setting, is governed by civil law, as opposed to criminal law, and consists of statutes and regulations originating from a number of legal sources. CCTPs are required to have medical direction to practice, in the form of online medical control occurring by telephone or radio or off-line medical control in the form of approved standing orders or protocols.
- Areas of liability can arise in the provision of transport, most of which come from a violation of the patient's basic rights; violations include assault, battery, false imprisonment, and negligence.
- To withhold resuscitative efforts, there must be a written order from a physician. If there is a question as to the status of a DNR order, err on the side of providing resuscitation rather than withholding it.
- The right to refuse treatment is a basic right of all patients and is based on the belief that every person of adult age and sound mind has a right to determine what is or is not done to his or her body. Providing care without allowing this consent puts CCTPs at risk of liability for assault or battery, even if the care is necessary to save the patient's life. If a patient refuses care, the CCTP must explain to the patient the possible consequences that may result without treatment.
- Consent, which can be informed (express) or implied, must be obtained before proceeding with treatment or transport, and the rules for minors vary in cases of emancipation.
- The patient has the right to receive uninterrupted care, and CCTPs could be held liable for abandonment if care is interrupted.
- The patient has a right to "appropriate" care, which means that a CCTP is expected to have the same training, skills, and abilities as other CCTPs and is expected to exercise reasonable care and diligence when using the skills and knowledge in the care of a patient. Failure to meet this standard of care results in negligence on the part of the CCTP.
- The patient has a right to expect that information obtained before, during, or after transport will be kept confidential, and to reduce the risk of breaching confidentiality, CCTPs should relay communications about the patient's condition only to the appropriate personnel and not release patient identifying information over the radio.
- In preparation for transport, CCTPs should find out the patient's diagnosis and condition and should

ensure that the patient has a patent and secured airway; patent and secured IV access if necessary; appropriate monitoring as needed for cardiac rhythm, blood pressure, pulse oximetry, and end-tidal carbon dioxide; appropriate measures to treat hemodynamic instability; appropriate chemical or physical restraints if needed for confused or agitated patients; and appropriate spinal immobilization or pneumatic antishock garments for trauma patients as needed.

- In preparing for transport, CCTPs should communicate fully with the other members of the health care team at the sending and the receiving facilities, and a plan of care should be developed that is within the CCTP scope of practice.
- In preparation for transport, CCTPs need to obtain the reason for transfer and the name of the receiving hospital and/or physician; they need to determine whether a BLS or an ALS provider must accompany the patient in addition to the CCTP and whether the sending facility plans to provide an escort; and they need to obtain the treatments provided at the sending facility, the treatments and drug orders for the duration of the transfer, and any potential complications that may arise during transport.
- The sending physician must send copies of medical records with the patient to the receiving facility that document the patient's current diagnosis and condition, symptoms the patient is experiencing, treatments given, results of tests conducted, written consent of the patient or a person with legal authority agreeing to the transfer, and the physician's verification that the benefits of transferring the patient outweigh the risks. The sending physician must also send samples of pertinent laboratory specimens.
- Before transport, CCTPs should perform a brief physical examination to determine if there are changes in the patient's condition that would require additional personnel or resources during the transfer.
- CCTPs operate with off-line medical direction using established written protocols, and if treatment beyond protocol is indicated or consultation is needed, CCTPs should establish contact with medical control and proceed under online medical direction.
- EMTALA requires that the sending physician designate the qualifications of personnel accompanying the patient during transport and that all necessary and appropriate equipment, medications, and supplies that may be required by the patient are available in sufficient quantities and are within the scope of practice of the personnel accompanying the patient during transport.
- CCTPs must assess the patient, discuss potential complications with the sending physician, and ensure that the sending physician understands the capabilities and limitations of the transport team. During transport, CCTPs must monitor the patient's condition, provide the appropriate care within the scope of practice, inform medical control or the receiving facility of any changes in the patient's condition or of any extenuating circumstances, and maintain thorough documentation.
- On arrival at the receiving facility, CCTPs ensure the continuity of care by giving a detailed report to the designated receiving care provider that includes all patient information, the names of the sending physician and facility, the reason for transfer, all documentation and records (including the patient's history, medications, fluid intake, condition during transport, and tolerance of transport), any transfer specimens from the sending facility, and any laboratory and/or radiography results from the sending facility. CCTPs should be available to answer questions from the receiving care provider after the transfer of care is complete.
- Documentation to record certain essential elements of every transfer is important and should include the times of each set of vital signs and each assessment finding, the details and times of each intervention (such as drug doses and routes of administration), the patient's responses to all interventions and any adverse reactions, all communications with medical control, any deviations from

the transfer or standing orders, any diversions to another hospital or facility, and any refusals of treatment by the patient.

Vital Vocabulary

abandonment The termination of an established patient-care provider relationship by the care provider without simultaneous transfer of health care responsibility to another care provider of equal or greater competence.

assault The act of putting someone in reasonable apprehension of imminent harmful or offensive contact.

battery Harmful or offensive touching of another without consent.

breach of duty to act A case in which a health care provider does not conform to the standard of care by providing inappropriate care, failing to act, or acting beyond the scope of practice.

competence Capacity or ability to understand the nature and effects of one's decisions or actions.

consent Voluntary agreement by a patient with sufficient mental capacity to capably choose assessment, treatment, or transport offered by the care provider; a patient not accepting care demonstrates the right to refuse.

damage In a legal context, damage must result from a breach of duty for a health care provider to be found negligent. Usually, the injury is physical.

do not resuscitate (DNR) order A type of advance directive that describes which life-sustaining procedures should be performed in case of a sudden deterioration in a patient's medical condition.

duty to act A legal obligation of public and certain other ambulance services to respond to a call for help in their jurisdiction. When the duty to act applies varies from state to state; it can be when a CCTP initiates treatment or when the dispatch center confirms that a transport vehicle is being sent.

emancipation A legal status in which a person who is younger than the legal age in a given state is legally considered an adult; he or she gives up the right to maintenance and support from a parent, and the parent relinquishes the right to control the child.

Emergency Medical Treatment and Active Labor Act (EMTALA) A federal law passed by Congress in 1986, with a primary focus of preventing hospitals from transferring patients to other facilities because they are unable to pay for services. Initially, EMTALA was a portion of the Consolidated Budget Reconciliation Act of 1985 (COBRA).

false imprisonment Intentional and unjustifiable detention of a patient.

Health Insurance Portability and Accountability Act (HIPAA) A law enacted in 1996, providing for criminal sanctions and civil penalties for releasing a patient's protected health information (PHI) in a way not authorized by the patient.

implied consent Assumption on behalf of a person unable to give consent that he or she would have consented.

informed (express) consent A patient's voluntary agreement to be treated after being told about the nature of the disease, the risks and benefits of the proposed treatment, alternative treatments, and the choice of no treatment at all.

medical control The oversight designed to ensure that actions taken by providers on behalf of patients are appropriate; divided into direct medical control, available in real time via radio, and indirect medical control such as standing orders and protocols.

medical direction Supervision of medical care, provided online (telephone or radio) or off-line (protocols, standing orders, education, and quality improvement).

negligence Failure to provide the quality of care (as defined by applicable standards) usually expected with conditions requiring that care; negligence is established when the plaintiff proves four elements: duty to act, breach of the duty, injury to the patient, and the breach was the direct cause of the injury.

off-line medical control Medical direction given through a set of protocols, policies, and/or standards.

online medical control A type of medical direction in which the care provider is in direct contact with a physician, usually via two-way radio or telephone.

protected health information (PHI) Individually identifiable information (such as name, social security number, and date of birth), health information (such as laboratory results and medical history), and demographic information (such as address and telephone number) that is protected by HIPAA.

proximate (direct) cause The reason an injury occurred; the final element that must be proven for a health care provider to be found negligent.

scope of practice The body of knowledge, skills, and therapies that health care providers can legally apply in patient care, based on training, certification, medical direction, and applicable law.

tort A wrongful act that gives rise to a civil suit.

Bibliography

Dunn JD. *History of Federal Circuit Court Cases on the Emergency Medical Treatment and Active Labor Act*. Prepared January 20, 1997.

EMTALA Regulations, 42 CFR §489.24, and selected sections from 42 CFR.20. 2004 CFR Title 42, Volume 3. Available at: http://www.access.gpo.gov/nara/cfr/waisidx_04/42cfr489_04.html. Accessed April 25, 2008.

Frew SA. *Patient Transfers: How to Comply With the Law*. 2nd ed. Dallas, TX: American College of Emergency Physicians Publications; 1995.

Health Care Financing Administration. Interpretative Guidelines and Investigative Procedures for Responsibilities of Medicare Participating Hospitals in Emergency Cases, 42 CFR §489.24. In: Health Care Financing Administration. *State Operations Manual (Pub. 07)*. Washington, DC: Health Care Financing Administration; Appendix V. Available at: http://www.cms.hhs.gov/CertificationandComplianc/08_Hospitals.asp. Accessed April 25, 2008.


STARS
STAR-1
C-GBNH

Aircraft Fundamentals

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Objectives

1. Explain the role of the Commission on Accreditation of Medical Transport Services (CAMTS) and the concepts of visual flight rules, instrument flight rules, and a sterile cockpit (p 40).
2. Discuss the concept of flight following (p 41).
3. Describe the various types of air medical helicopters that have been used and that are currently in use (p 43).
4. Describe the advantages and disadvantages of the use of rotor-wing aircraft (p 42).
5. Describe the advantages and disadvantages of the use of fixed-wing aircraft (p 43).
6. Briefly discuss the emphasis of the air medical role to facilitate critical care transports (p 40).
7. Summarize Federal Aviation Administration guidelines that are pertinent to air medical transport (p 52).
8. List standards created to enforce safety (p 49).
9. Summarize crew resource management (p 50).
10. Understand the importance of taking safety precautions, including an understanding of crash and survival skills (p 51).
11. List techniques for safety and survival in the aircraft environment (p 56).
12. Discuss common employment criteria for medical flight team members (pp 53, 54).
13. Discuss the critical care provider certifications that are prevalent and required flight crew training (p 54).
14. Describe the various crew configurations used by air medical programs (p 53).
15. Describe accreditation of the air medical program (p 54).

Introduction

The CCTP may work in the aircraft environment as a part of the course of his or her duties. Some CCTPs may be permanently assigned to an aircraft, whereas others may rotate on to aircraft as part of their staffing assignments. The CCTP needs to be familiar with both rotor-wing (helicopter) and fixed-wing (airplane) operations. The aircraft environment is unique in that patient care is not the primary concern—that is, aircraft safety and aircraft operations will always come before patient care in this setting. This concept is difficult for some providers to understand, but is essential to the successful completion of the mission.

The Air Medical Role

Many organizational models for air transport programs are possible, although three dominate current practice. One is a hospital-based air program, with many hospitals sharing the responsibility and costs of the service. A second model is the public service agency—a structure that allows government agencies to

use the air transport program outside the medical setting while also serving the needs of critically injured and ill patients. The third model is the private service, which has recently gained attention and is growing because of its ability to meet the needs of health care systems. This model primarily offers interfacility transfers but in some systems also offers emergency scene responses.

The basic concepts of an air medical service are to rapidly transport CCTPs to a patient, stabilize the patient's condition, and transport that patient to a tertiary care center as rapidly as possible. The patients who benefit most from air medical transport are those who are critically ill or injured and who would have a negative outcome with a prolonged transport.

Although air transport offers many benefits, CCTPs must weigh the benefit to the patient vs the risk of the specific mode of transport. Before going this route, patients being transported should be evaluated and assessed for the effects of altitude and other forces that can adversely alter their conditions en route. For example, cardiac patients with activity-sensing pacemakers may encounter a severe complication if the pacemaker malfunctions because of flight vibrations. It is also necessary for air medical programs to consider the adverse effects of air transport on their employees. They, too, may have adverse reactions to aircraft humidity, noise, and vibrations, resulting in possible dehydration, hearing loss, and fatigue, respectively. Effects of altitude are discussed in Chapter 4.

Air medical transports are shown to decrease mortality rates and are indicated for all critically injured or critically ill patients. Nevertheless, they are often contraindicated in the following circumstances:

- Severe anemia
- Hemoglobinopathy
- Myocardial infarction (MI) within 10 days or complications in the 5 days before the flight (with the exception of a patient with an acute MI being flown to the catheterization lab, which is done routinely)
- Uncontrolled arrhythmia
- Pregnancies past 24 weeks' gestation
- Recent eye surgery affecting the globe
- Nonacute hypovolemia

If air medical transport is undertaken in such cases, special considerations—such as flying at a lower altitude than usual—may need to be taken to optimize patient outcome. Although patient conditions and possible complications must always be considered, terrain, weather, and geographic location of the closest facility must also be factored into whether air medical transport is the best option. In addition, it is important to recognize transport crew capabilities and to ensure their safety.

There are drawbacks associated with air medical transport, but history has shown that air medical transports decrease mortality rates and allow access to more specialized tertiary care facilities. Air medical transports allow rapid access into the health care system, especially for patients who may live in otherwise inaccessible areas. Such programs have proven that they are capable of providing a major source of support to community hospitals and rural areas confronted with critically ill or injured patients.

Communications

The **Commission on Accreditation of Medical Transport Services (CAMTS)** is a program dedicated to ensuring high-quality patient care and safety within ground, rotor-wing, and fixed-wing services. CAMTS has set many communication standards to help improve the overall safety of most critical care transport systems. Much of its effort has focused on rotor-wing and fixed-wing divisions because proper

communication procedures within the cockpit are important for ensuring the safety of both patients and crew.

Sterile cockpit is a term used by the pilot in command to describe the atmosphere during takeoffs, landings, and any other critical phase of flight. During this time, no communication is allowed that could distract the pilot in command unless it is related to the safety of the flight **Figure 3-1**. Air carrier standards state that a sterile cockpit should be enforced at all altitudes less than 10,000′; however, most helicopters fly only between 2,000′ and 5,000′. In such situations, the sterile cockpit times are determined by the pilot in command, but will always include takeoff, approach, and landing. Once the rotor-wing aircraft has reached its cruising altitude and speed, the pilot in command will permit crew members to take part in casual conversation.

Communications with operation centers and dispatchers should also be observed during this phase of the flight process. The rule of thumb should be that communications to operation centers, such as those that give lift-off times, ETAs, fuel, and number of crew members, should be relayed prior to lift-off, after the aircraft reaches its cruising altitude, or until the pilot declares a nonsterile cockpit. A sterile cockpit should also be observed while communicating with patients during flight. Most pilots have the option of "isolating" crews (by flipping a switch in the cockpit, the pilot can take the crew out of the communication loop with air traffic control [ATC] and ground units).

Figure 3-1 A sterile cockpit (no unnecessary communication) must occur during critical phases of flight to prevent potential distractions to the pilot.

If, during flight, the pilot in command experiences a mechanical issue or there is a dramatic change in the patient's condition, it may be necessary to land short of the original destination. In most cases, when mechanical issues arise, the pilot in command seeks out the closest airport as a landing site. If the patient's condition deteriorates to the point that immediate medical attention is needed, the pilot in command will land at the closest appropriate medical facility, where the patient will be taken inside for advanced medical care that is unavailable in the aircraft. The crew should also be alert at all times for potential mechanical malfunctions such as smoke in the patient care area, smell of abnormal odors, unusual vibrations not reported by the pilot, abnormal sounds, or fluids leaking into the patient bulkhead. Pilots may also choose to declare an "emergency" to ATC. This should immediately result in a sterile cockpit.

Instrument Flight Rules vs Visual Flight Rules

In the aviation industry, all parties must be familiar with the two possible modes of flight.

Visual flight rules (VFR) describe a mode of flight used when weather conditions are good,

meaning there is generally good visibility and minimal cloud cover. A pilot can fly VFR in many areas without being in contact with ATC and is responsible for keeping the aircraft clear of clouds; however, in populated areas such as large cities, the pilot must be in contact with ATC even when flying VFR.

Instrument flight rules (IFR) describe flight rules that apply when minimum cloud clearance and visibility requirements cannot be met. During these conditions, the pilot may not be able to see outside the aircraft (ie, owing to clouds or fog) and must rely on the instruments inside the cockpit to maintain control and navigation of the aircraft **Figure 3-2**. A pilot flying IFR must receive an ATC clearance prior to takeoff and maintain contact with ATC during the flight, which will ensure proper distance from other air traffic is maintained.

Very few rotor-wing programs conduct IFR operations because pilots who perform certified-instrument approaches need to be able to land safely and legally during IFR weather, and rotor-wing air medical aircraft often land at hospitals and unimproved landing zones. The few programs that conduct rotor-wing IFR usually require two pilots for these operations. Fixed-wing aircraft routinely operate IFR as they use **Federal Aviation Administration (FAA)**-certified approaches at airports for takeoff and landing and usually require two pilots for IFR operations as well.

Figure 3-2 A pilot depends on the instrument panel when flying under instrument flight rules.

Flight Following

A critical care transport is initiated when the communication center receives a request for service from an authorized agency such as local police, fire, EMS, or a referring hospital and then passes that request on to the flight team. The flight team then either accepts or declines the mission after taking the relevant factors, such as weather, terrain, and nature of injury or illness, into account. Some programs require that the pilot make the decision without any knowledge of the patient. This prevents pilots from being influenced and taking on extra risk, for example deciding to transport a pediatric patient when it is actually not safe. Once the aircraft launches, the communication center and/or ATC remains in contact with the aircraft throughout the flight.

When a pilot is flying IFR, the ATC is legally mandated to be in contact with the aircraft to ensure it maintains a safe distance (ie, separation) from other IFR aircraft. When a pilot is flying VFR, a type of monitoring called **flight following**, or radar advisory service, may be provided by an authorized ATC facility. The service consists of a controller notifying the aircraft about traffic in the area when the controller is available to do so **Figure 3-3**. The flight following provided by ATC is advisory only and provided by the controller when the workload allows. While most controllers try to accommodate requests for flight following, at times of peak workload, services may not be available.

When a pilot is flying VFR, it is good practice to maintain constant contact with other local traffic

and ATC facilities in proximity of the aircraft.

Figure 3-3 Flight following occurs when an air traffic control center stays in contact with a pilot.

When a pilot is flying VFR, flight following is not mandatory but is highly recommended because it can assist the pilot in avoiding a collision with terrain or other aircraft. When a pilot is flying IFR, a filed flight plan, flight clearance, and ATC contact are mandatory.

When a pilot is flying VFR in a helicopter, contact is usually maintained with one of two entities: ATC or an EMS communications center. If the pilot in command decides not to flight follow with ATC, real-time radio contact with a communications center must occur every 15 minutes.

During fixed-wing operations, when a flight plan has not been filed, the pilot in command must make contact with ATC or a communications center every 30 minutes. It is industry standard practice, however, to operate fixed-wing aircraft on an FAA flight plan, and most air transport agencies enforce either filing a flight plan or regular contact with their own communications center when in flight.

During patient transport in a ground unit, any lapse in communication must not exceed 45 minutes. During check-in times, CCTPs should provide updates of both the patient's condition and the geographic location. Regardless of who is tracking the various ambulances, airplanes, or helicopters, a complete record of these communications should be kept for 30 days.

The communications center should be secured at all times, with only authorized personnel allowed access to this facility. Personnel should be familiar with flight operations and specially trained in the procedure of flight-following aircraft.

Identifying Flight and Scene Locations

It is critical that the flight-following center utilize global positioning system (GPS) technology to be able to constantly track the aircraft's movement even when not in radio contact with the center. Several commercial flight-tracking programs are available that use a GPS signal that is sent from the aircraft and received by the center. In the event of an in-flight emergency requiring an emergency landing, the center can use this signal to immediately determine the aircraft's last known position.

The flight-following center also needs to have computer software available that can allow for a physical address to be input and converted to GPS coordinates. In the rotor-wing environment, responders to the scene frequently use a handheld GPS device to obtain coordinates for the geographic location. These devices are extremely accurate and if the coordinates are properly read and relayed, they will lead the aircraft to within a few hundred yards of the scene. To prevent inaccuracies in the relayed coordinates, at least two sets of coordinates should be obtained for all scene flights.

Rotor-Wing Transport vs Fixed-Wing Transport

As mentioned earlier, the CCTP needs to be familiar with both rotor-wing (helicopter) and fixed-wing (airplane) operations. Both types of transport have advantages and disadvantages.

Rotor-Wing Transport

Rotor-wing transport has the distinct advantage of vertical takeoff and landing, allowing the helicopter access to areas that are inaccessible by ground vehicles and/or fixed-wing aircraft **Figure 3-4**. Rotor-wing aircraft generally have sustained speeds in excess of 150 mph, can operate at altitudes less than 2,000′, and can move from point to point. (Depending on local terrain, the altitude at which an aircraft will be able to fly may vary and is always at the discretion of the pilot in command.) Air medical providers that use such aircraft serve all types of population bases, from dense urban to extremely rural. A relatively small number of helicopters can serve a large population because of the quick turnaround time for most flights.

Rotor-wing transport has some disadvantages. Helicopters are more restricted than fixed-wing aircraft regarding weather limitations, as they generally cannot fly IFR. They also have more interior space limitations, oftentimes making it difficult to perform complex procedures while the CCTP is in the aircraft. For example, because of space constraints, when being transported in a helicopter, a patient may be inaccessible from the waist down, or there may not be enough space behind the patient's head to allow intubation. These situations can have major management ramifications.

Figure 3-4 Helicopters can access locations that ground vehicles and fixed-wing aircraft cannot, making them ideal for certain situations.

Helicopters are also expensive, averaging between $2 and $5 million for a new aircraft. For this reason, there must be justification for the necessity of the helicopter to offset operating costs. Specifically, helicopter transport is more expensive when compared with ground transport, averaging almost four times the cost of an ambulance.

Helicopter transport is also subject to weight limitations. This concern becomes more problematic in the summer when the air is less dense, decreasing the amount of lift that the blades can achieve (ie, lift capacity).

Fixed-Wing Transport

As a general rule, transport by fixed wing is safer than transport by rotor wing. Fixed-wing aircraft use established landing areas (airports) and fly at designated cruising altitudes that minimize the risk of colliding with human-made objects. Most fixed-wing air medical aircraft are IFR certified and fly with a

two-pilot crew, further increasing safety.

Fixed-wing aircraft offer several advantages for medical transport. Two of their greatest advantages are their high speeds—ranging from 250 to 600 mph—and their ability to travel greater distances. Fixed-wing aircraft generally have the ability to carry multiple patients, ranging from two on smaller aircraft to hundreds on large military air medical aircraft **Figure 3-5**. There are usually no weight limitations regarding patient size, and the aircraft can carry multiple medical crew members and a variety of equipment.

There are some distinct disadvantages to fixed-wing aircraft, including the high cost of obtaining an aircraft for air medical purposes. Many air medical providers do not purchase or lease their own aircraft, but instead contract with various executive aircraft services to provide aircraft as needed. The cost of patient transport by fixed-wing aircraft can exceed $10,000 or more for short-distance flights and can exceed $100,000 for international flights. Most fixed-wing services secure reimbursement from patients or insurance companies prior to initiating flight because of the high costs involved.

Fixed-wing aircraft must use maintained landing fields, and restrictions on runway length may apply depending on the size of the aircraft. Last, fixed-wing aircraft require hangers, which increases the overall operating costs.

Figure 3-5 C-130 Medevac of Hurricane Katrina patients.

Rotor-Wing Aircraft

Mission Profiles for Air Medical Helicopters

On average in the air medical industry, 54% of flights will be for interfacility transports (hospital to hospital), 33% for scene responses, and 13% for other types of events (eg, organ procurement and search-and-rescue operations). Some air medical helicopters are also used to transport surgical staff to emergency scenes to provide patient care, such as field amputations.

Air medical helicopters may perform a wide variety of missions. The majority of air medical programs limit their capabilities to the transport of the sick and injured, however, and do not incorporate other services into their mission profile. A limited number of air medical programs also support search-and-rescue and law enforcement operations.

Types of Air Medical Helicopters

No standard makes or models of helicopters exist for medical transport **Table 3-1**. Instead, the type of helicopter used depends on several factors, including patient load capacity, over-water operations, requirements for VFR or IFR flight, altitude of operations, economy or budget of the program, and

(geographic) range of flights. The program type also varies based on community need. For example, some programs are hospital based, others are community based, and still others are operated by a local government program (ie, law enforcement, fire, or a county EMS program) or state government program (ie, state police). Likewise, some programs are nonprofit operations, whereas others are for-profit businesses. There is no single ideal program, as the best option for one community may not be suitable for another.

TABLE 3-1 Helicopters Commonly Used in the Air Medical Industry
Single-Engine
Bell 206 Long Ranger
Bell 407
Eurocopter AS 350
Twin-Engine
Bell 222/230
Bell 230
Bell 412
Bell 222U
AgustaWestland AW109
MD 902 Explorer
BK-117C1
EC145
Sikorsky 76A
Eurocopter AS-365 Dauphin

Most air medical programs use an aircraft vendor, which provides the aircraft and pilots and all of the required aircraft maintenance. With such an arrangement, it is the responsibility of the air medical program to provide the medical crew and medical equipment. This option may deliver significant savings when a program is initially started up, as a hospital or EMS agency may not have to make a large capital outlay for the purchase of an aircraft. A vendor usually operates several bases, which allows for sharing of maintenance personnel, maintenance facilities, check pilots, and spare aircraft. The vendor and the air medical program usually participate jointly in billing and share a predetermined percentage of the reimbursements.

Single-Engine vs Twin-Engine Air Medical Helicopters

Air medical helicopters are divided into two categories: single-engine and twin-engine models **Figures 3-6 and 3-7**. Both types of aircraft have distinct advantages and disadvantages, so each program must determine which type of aircraft best fits its overall mission profile.

A longstanding debate in the history of helicopter aviation is whether a twin-engine aircraft truly is better than a single-engine aircraft. Intuitively, it makes sense to have two engines instead of one. It is

more reassuring to have two engines for long flights over water or inhospitable terrain. However, the turbine engines used today have become increasingly reliable and have few failures. Thus, a single-engine aircraft is more economical. Many communities that would not otherwise be able to afford aircraft coverage are currently served by a single-engine aircraft.

Several factors need to be taken into account when deciding between twin-engine and single-engine aircraft, including the types of missions flown, the space available to land the aircraft, the required payload, and the available money to operate the program. Programs that mainly perform interfacility transports tend to require larger aircraft to handle items such as neonatal isolettes for neonates in critical condition **Figure 3-8**, balloon pump consoles for patients with heart failure **Figure 3-9**, and other large and bulky equipment.

Figure 3-6 Single-engine helicopter (Bell 407).

Figure 3-7 Twin-engine helicopter (BK117 C1).

Figure 3-8 Flight team loading a neonatal isolette.

Figure 3-9 A console for an intra-aortic balloon pump, secured for transport. Note that this piece of equipment takes up a substantial amount of room and may limit the number of other items you transport in the crew cabin.

The advantages of a single-engine aircraft compared with a twin-engine aircraft are as follows:

- Aircraft start-up times of 3 to 5 minutes for a single-engine aircraft compared with 8 to 10 minutes for a twin-engine aircraft
- Less downtime for maintenance
- Lower maintenance costs
- Lower operating costs
- Lower fuel requirements
- Smaller landing zones needed

The advantages of a twin-engine aircraft compared with a single-engine aircraft are as follows:

- Two-patient capacity vs one-patient capacity in the single-engine aircraft
- Larger openings for patient loading and unloading
- Larger patient care area
- Dual engines in case one fails (especially useful for transport over water)
- Greater lift capacity

Fixed-Wing Aircraft

Mission Profiles for Air Medical Fixed-Wing Aircraft

Fixed-wing aircraft are typically used for flights of 200 nautical miles (300 miles) or greater, although they may sometimes be used for shorter distances. For example, in some rural, mountainous states in the United States, fixed-wing transport is used for flights as short as 60 nautical miles (90 miles) because of severe weather that prohibits helicopters from flying. Fixed-wing air medical transport would also be more commonly used for international transports, for example in the military.

Types of Air Medical Fixed-Wing Aircraft

The two main types of fixed-wing aircraft are propeller driven (turbo prop) and jet aircraft. A turbo prop aircraft has a jet engine and propellers.

Fixed-wing aircraft can be single engine or multi-engine. As with helicopters, the type of aircraft utilized once again depends on the transport needs. For example, some fixed-wing models are more spacious than others **Figures 3-10 and 3-11. Table 3-2** lists commonly used fixed-wing air medical aircraft.

Air Medical Efficacy

One of the most heated debates in emergency medicine today is the efficacy of air medical transport. Since the inception of air medical transport, critics have questioned whether air transport offers measurable (ie, quantifiable) benefits. Advocates on both sides of the debate provide compelling evidence to support their theories.

Benefits of Air Medical Transport

Emergency departments in community hospitals have declined from just over 5,000 in 1992 to approximately 4,600 in 2002, a trend that is expected to continue. The number of Level I trauma centers has also declined slightly. The remaining Level I trauma centers in the United States maintain 95% bed capacity, resulting in frequent diversions of patients to other comparable facilities. With the use of air medical transport, 65% of the US population has access to a Level I trauma center within 60 minutes, and half the US population has access to 10 Level I or II trauma centers within 60 minutes.

TABLE 3-2 Aircraft Commonly Used in the Air Medical Industry

Propeller-Driven
Beechcraft King Air B100, B200, C-90B, 90, and 200
Cessna
Rockwell Turbo Commander 690B
Jet
Lear 25, 35, 36, and 55
Citation II
Challenger
Gulfstream

Hawker 800
Pilatus PC-12

Figure 3-10 Propeller-driven aircraft used for air medical transport include the Beech Super King Air 200C.

Figure 3-11 Jet aircraft used for air medical transport include the Lear 35A.

In some regions, air transfer could mean the difference between life and death. For a patient with a myocardial infarction in a typical suburban area, the difference between ground and air transfer may be only minutes; however, in some areas of Alaska, Canada, Central and South America, Africa, Asia, or Australia, conventional ground transfer could take hours to reach definitive care. An aircraft may only take one fourth of that time.

Air transfer is also about specialty. Most patients who require critical care transport are not considered a routine transfer. These patients usually receive multiple drips such as nitroglycerin, dopamine, amiodarone, and heparin. In addition, the same patient may need continuous sedation and neuromuscular blockade, necessitating patient transfer to a ventilator by the flight crew. A flight crew member must be skilled in many areas to make this transfer quickly and without delay.

In addition, specialty care is increasingly being housed in urban specialty centers and is less available in nonurban settings, meaning that patients in nonurban settings who require specialty care may need transport over longer distances than in the past. Air medical transport has been found to significantly reduce treatment times in the following situations, all of which involve specialty care:

- Incident-to-catheterization lab time for patients with acute MI

- Incident-to-operation room time for surgical patients
- Incident-to-thrombolytic time for patients with acute MI and stroke

The greatest reduction in time to reach treatment is seen with patients who bypass the emergency department and are directly admitted at the receiving facility. Many flight teams have preestablished protocols for direct admission of certain types of patients, which allows them to receive specialized care immediately upon arrival. A flight crew's protocols may allow a higher level of care than a critical care transport ground crew's protocols.

Specialty resource centers, such as cardiac, stroke, pediatric, and burn centers also have critical care ground transfer units available that can be used by the flight team if weather prevents the aircraft from flying. In some regions, referring hospitals are required to have a registered nurse accompany critical patients (depending on state laws or regulations). In some cases, this causes difficulty because the ambulance is leaving its first-due coverage district (ie, the area where the ambulance is the primary medical transport provider and is expected to be first on the scene, rather than serving as a backup) and the hospital is short one registered nurse. Therefore, the benefit of air transfer allows the flight team to retrieve a patient, receive transfer of care, and transfer following local protocols.

The benefit of air medical transport in mass-casualty situations has been definitively proven. Postdisaster analysis indicates between 60% and 80% of patients transport themselves to the nearest facility during a mass-casualty incident, quickly overwhelming the capabilities of the nearby hospitals. In such cases, it is prudent to begin transport of patients by air to outlying hospitals because the majority of ambulatory patients flock to the closest hospitals.

Hazards of Air Medical Transport

Air medical transport should be used only after conducting a risk–benefit analysis. Incorporating a helicopter into a chaotic rescue scene increases the risk to responders and patients, even when established safety measures are followed. Even the most skilled helicopter pilots cannot control the amount of debris that is blown about, nor can they totally eliminate the hazards associated with the main rotors and tail rotors (with the exception of fully enclosed tail rotors).

Only patients who are critically injured or seriously ill should be considered as candidates for air medical transport. The decision to transport a patient by air should be made after carefully considering all of the transport options. Some patients will clearly benefit from the speed and advanced skill of air medical transport teams, whereas other patients could be transported by ground more quickly and with less potential danger.

Controversies

Your service may or may not transport prisoners. Some services have opted not to provide these transports for the safety of the patient and crew. Instead, a service staffed by law enforcement personnel performs this service.

Financial Implications of Air Medical Transport

The cost of air medical transport varies greatly depending on the region, distance flown, care provided, and other factors. According to the 2000 Annual Transport Statistics and Fees Survey, the average cost ranges from $2,600 to $6,200 per flight. In 2002, Medicare reported that the average cost of air medical transport via helicopter was between $5,000 and $10,000, requiring Medicare to increase its

reimbursement rates for this type of transport. The number of helicopter transports has skyrocketed in recent years. Medicare reported that it had a 58% increase in number of flights reimbursed in 2004 relative to the number reimbursed in 2001. These data confirm that helicopters are expensive to fly and maintain, with operational costs per helicopter averaging $1 million per year.

Not surprisingly, to obtain reimbursement from insurance companies, helicopter use for medical transport must be justified. Many insurance companies balk at paying $10,000 for transport and will aggressively review whether such helicopter usage was appropriate. In response to aggressive billing reviews, Medicare—whose standards often filter down to other payers—established a list of criteria that justify the use of air medical transport. To satisfy the Medicare criteria, many flight programs routinely list all applicable justifications for air transport within the medical report to facilitate insurance reimbursement.

Helicopter transport is 400% more expensive than fixed-wing transport for flights of more than 101 nautical miles (1 nautical mile = 1.5 miles). For this reason, many services choose to send patients by fixed-wing aircraft, if available, for flights exceeding 100 nautical miles. Some air medical helicopter programs do not charge for their services and are instead funded by taxes or donations. Other air medical services offer a membership program that allows people to pay an annual subscriber fee for their household; this fee covers the cost of any air medical transport during that period.

Special Populations

Any high-risk complication of pregnancy, childbirth, or miscarriage qualifies as criteria for air medical transport.

Determining Air Medical Transport Suitability

When you are considering whether to use air medical transport, the requesting EMS provider or physician must avoid three critical errors:

- Requesting air medical transport for a patient when a more suitable means of transport is clearly indicated
- Requesting air medical transport when the patient could arrive at the receiving facility more quickly by ground transport
- Requesting air medical transport for a patient when cardiac arrest is ongoing or imminent

In most situations, air medical transport compares unfavorably to ground transport on the basis of safety, cost, and availability. On average, air medical transport costs at least four to five times more than ground transport over the same distance. In terms of availability, one air medical aircraft may serve a population of 500,000, whereas 20 or more ambulances may be available to serve the same population. Given this disparity, providers who are requesting air medical transport need to determine whether they will be needlessly tying up resources that could be better utilized elsewhere.

One other consideration to take into account when determining ground vs air transportation is the level of care that can be given. In some states, the highest level of ground provider is a paramedic. When considering flight programs, registered nurses are usually the highest level of provider, thus allowing the higher level of care such as balloon pumps, certain critical medications, and IV infusions of nonparamedic approved medications.

Thirty nautical miles (34.5 miles) or 30-minute transport is considered to be the minimum for which air medical transport is quicker than ground transport. (The industry-wide average for miles transported per flight is 61 miles.) However, factors such as traffic, construction, and weather have to be considered

when making this determination. Some patients who require extended extrication time or have limited accessibility may benefit from air medical transport. Other patients may benefit from the advanced caregiving skills of the flight team, including their ability to perform rapid-sequence intubation, place chest tubes, or administer blood.

Other Justifications for Air Transport

A helicopter may sometimes be requested because the ground EMS unit is overwhelmed by the severity of the patient's injuries and needs the expertise of the flight crew. Air medical programs are frequently called to scenes where the ground EMS units have waited on the helicopter to perform advanced procedures such as rapid-sequence intubation. This situation is not strictly limited to ground EMS units, but is frequently observed in smaller hospitals as well. The medical flight team from the helicopter is often well respected in the medical community and is viewed as a "safety net" that can be relied upon when the provider on the ground requests assistance. As a member of the flight crew, you should be fast, proficient, and skilled in your area or expertise. By being proficient and skilled, you can ensure that your team is maintaining a limited on-scene time, which helps reduce the overall flight time to higher levels of care.

Transport of Patients in Cardiac Arrest

It seems counterintuitive to not utilize air medical transport for a patient who is in cardiac arrest or on the precipice of arrest. Cardiac arrest represents one of the most serious medical situations and would, in theory, stand to benefit the most by air medical transport. Nevertheless, most air medical providers do not routinely transport patients in active cardiac arrest for the following reasons.

First, it is against federal air regulations for passengers or crew onboard an aircraft to not be restrained in seatbelts during takeoffs and landings or as requested by the pilot. Providing effective CPR in the aircraft often requires the medical crew to be unbuckled.

Second, CPR is often futile, with fewer than 5% to 8% of all patients who receive CPR being resuscitated. Use of air medical transport for a patient in cardiac arrest is, therefore, generally viewed as a poor use of resources.

Last, to provide effective resuscitation, CPR generally requires a minimum of three providers: one provider performing chest compressions, one managing the airway, and one providing pharmacologic therapy. Rotor-wing aircraft typically carry only two medical providers, thus limiting the effectiveness of resuscitation. Although a patient in cardiac arrest should not be placed in the aircraft and flown, the CCTP must realize that occasionally a patient may arrest in flight and should be prepared to deal with this situation.

Triage of Air Medical Transport Patients

Intuitively, the use of air medical transport makes sense: It expedites transport of critical patients to appropriate facilities. However, numerous studies have concluded that air medical transport tends to be overused. This section discusses considerations when triaging critical care patients.

Overtriage in the Field

A retrospective review of pediatric trauma patients transported by helicopter reveals a trend toward **overtriage**, in which a patient is considered to be in more serious condition than he or she actually is. For example, a study in central Ohio found that nearly 70% of pediatric trauma patients transported by air were discharged from the emergency department. Similarly, a study at Children's National Medical Center in Washington, DC, found that 85% of pediatric trauma patients sent to the hospital by air were

actually overtriaged and did not require air medical transport. A study that reviewed pediatric trauma patients flown to hospitals in Los Angeles, CA, found that 33% were overtriaged and were discharged home from the emergency department. However, many of these cases involved appropriate use of air transport: 37% of these patients had multiple injuries, 14% were intubated in the emergency department, 18% were admitted to the intensive care unit, and 4% were taken directly to the operating room. To curtail the trend toward overtriage, some are advocating for better triage training for prehospital personnel.

An estimated 10% or fewer of all patients (pediatric and adult) sent to hospitals require multiple interventions. By contrast, more than 90% of the patients transported by rotor-wing or fixed-wing aircraft require multiple interventions. The patient acuteness (ie, the seriousness of the patient's condition) is much higher in the air medical industry because of the triage by experienced providers prior to the initiation of air medical transport.

Undertriage in the Field

Although many believe air medical transport is overused, there is some benefit to overtriage. **Undertriage** in the field, in which a patient is considered to be in less serious condition than he or she actually is, is a problem as well.

Some trauma physicians state that an overtriage rate of 25% is acceptable to ensure that those patients who most desperately need care are not overlooked. A recent literature review, however, indicates that this number can be as low as 10% and still encompass most outliers. Most trauma surgeons agree that a discharge rate from the emergency department of less than 10% is an indication of undertriage and suggests that patients who would most benefit from quick transport and trauma team activation are not being appropriately triaged.

Air Medical Use Criteria

Table 3-3 lists guidelines for determining whether use of air medical transport from one facility to another is appropriate. When you are making this determination, it is important to identify those patients who will *not* benefit from air medical transport. **Table 3-4** identifies some of the absolute and relative contraindications to air medical transport.

Current **helicopter emergency medical service (HEMS)** criteria use both mechanism of injury and physiologic parameters to determine which patients might benefit most from transport in helicopters. Strictly basing the need for air medical transport on the mechanism of injury is no longer a clinically accepted practice because it has resulted in needless trauma team activations and emergent transports of stable patients. In contrast, new models that use both mechanism of injury and physiologic parameters to triage patients have been found to be effective at including appropriate patients and excluding patients who do not require trauma activation.

HEMS providers should have frequent discussions with the local trauma team about which criteria are used to triage patients. In addition, a formal system to track the "time to discharge" on all patients flown should be implemented. Air medical services whose overtriage rates exceed accepted limits should create opportunities to educate local providers about triage criteria.

Controversies

Controversy exists concerning specialty referral centers refusing transfers from outlying emergency departments unless their particular air medical service is used—even when ground transport would be

perfectly acceptable. This practice needs to be addressed in the industry.

TABLE 3-3 Patient Characteristics That Warrant Air Medical Transport

Burns ≥ 18% total body surface area (second degree or greater)
Burns resulting from an explosion with severe respiratory distress *or* altered level of consciousness (patient not alert)
Burns with sustained unconsciousness
CO inhalation or HazMat exposures with sustained unconsciousness
Postresuscitation from near drowning Diving accident with suspected neck injury SCUBA accidents
Electrocution or lightning strike with sustained unconsciousness
Long fall (from heights exceeding 6′) with sustained unconsciousness
Stab, gunshot wound, or other penetrating trauma to the torso or head with sustained unconsciousness
Childbirth or miscarriage with high-risk obstetric complication: • Viable premature infant (≥ 20 weeks' gestation) • Viable multiple births (≥ 20 weeks' gestation) • Maternal bleeding disorder • Maternal use of blood thinners
Traffic or transportation crashes with patient pinned *and* sustained unconsciousness
Traffic or transportation crashes with sustained unconsciousness *and* high mechanism: • All-terrain vehicles • Automobile vs bicycle or motorcycle • Ejection • Personal watercraft • Rollover crash • Vehicle off bridge/height
Need for advanced life support or specialized care not available by ground services
Time-critical illness or injury: • Stroke or acute coronary syndrome with potential for interventional care • Amputated limb with potential for reattachment

Controversies

Organ donation is a major concern in a critical care unit, but should not be relevant to critical care transport. CCTPs care for critically ill patients as though they are expected to survive. The patient's status as an organ donor should have no impact on care.

TABLE 3-4 Patients Who Will Not Benefit From Air Transport
Contraindications
Patients with terminal illnesses
Patients with do not resuscitate orders
Patients in arrest without return of spontaneous circulation
Patients whose conditions will overwhelm the capabilities of the
crew
Relative Contraindications
Severe anemia
Hemoglobinopathy
Myocardial infarction within 10 days or complications in the
5 days before the flight
Uncontrolled arrhythmia
Pregnancy > 24 weeks' gestation
Recent eye surgery affecting the globe
Nonacute hypovolemia

Air Medical Crashes

Currently, 770 helicopters in the United States are dedicated to air medical service. Patient transports are increasing at a rate of 5% per year as the industry continues to grow. As the number of air medical providers has increased over the years, so has the number of air medical crashes.

To some extent, the trend toward more air medical crashes is simply linked to the concurrent rise in the number of aircraft in the fleet and the number of missions flown. In the United States between 1998 and 1999, flight hours increased by 12%. This trend is expected to continue.

Helicopter EMS is one of the most dangerous forms of aviation, with a high accident rate compared with other sectors of aviation.

Causes of Air Medical Crashes

The **National Transportation Safety Board (NTSB)** has found that aircraft crashes are never caused by one single event. Instead, they are caused by a series of factors that ultimately contribute to the final event. The FAA Advisory Circular Number 60-22 states, "One bad decision often leads to another. As the string of bad decisions grows, it reduces the number of subsequent alternatives for continued safe flight."

When crash statistics are examined, they reveal that 55% of crashes occur during scene flights; however, scene flights account for only 35% of HEMS missions. Forty percent of flights occur at night; however, they account for more than 60% of the crashes. The NTSB has identified four major factors contributing to HEMS crashes:

1. Human error (68% of crashes)
2. Weather (30% of crashes)

3. Mechanical failure (25% of crashes)
4. Controlled flight into terrain (20% of crashes)

The NTSB has concluded that weather poses the single greatest hazard to HEMS operations (human error is the number 1 cause of accidents, whereas weather is the main contributing factor). Although weather is a contributing factor, in only two crashes was adverse weather the primary cause of the crash. Notably, the cause of weather-related crashes is not the pilot's disregard for weather at takeoff, but rather encounters with unpredicted **instrument meteorological conditions (IMC)** (cloudiness or low visibility) during flight.

As helicopters become more reliable, the chance of mechanical failure has dropped. In the last 15 years, the number of crashes due to mechanical failures has decreased by almost 10%.

Of the HEMS accidents in the past 10 years, only 30% occurred on patient-related missions; the remaining 70% of crashes occurred during maintenance flights, check flights, relocation, and public relations flights. The average crash rate for HEMS has increased. HEMS is one of the most dangerous forms of helicopter aviation and carries some of the highest line-of-duty death rates for EMS providers, second to motor vehicle collisions.

Making the Industry Safer

Within the industry, several solutions have been proposed to reduce the number of air medical crashes. Crew resource management training for all flight team members is now the industry standard. All new flight team members are required to attend initial training, and most programs offer annual refresher training.

Another important safety measure is information sharing between competing agencies. In the air medical industry, the term **helicopter shopping** describes the practice of making sequential calls to numerous air medical providers in an attempt to find a service that will accept a mission request that has been declined by other services based on safety factors such as poor weather, limited landing zone availability, exceptional distances, or other factors. To prevent helicopter shopping, air medical providers should share their decisions to decline certain missions based on safety issues with surrounding air medical services, even if they are in direct competition with the other services.

Air medical programs also need full support from their business's administration and the aircraft vendor when they choose to decline a mission. Some believe that stiff competition in the air medical industry prompts some services to take missions even when safety is in question. Although this may have been a factor in the past, currently no data support this claim. With intense scrutiny on the air medical industry from the federal government, other oversight agencies, and the community, air medical services have implemented numerous guidelines to prevent flight crews from being pressured into taking questionable flights.

To facilitate cooperation among the various parties involved in the transport decision, joint agency training is encouraged between air medical providers and different agencies. Air medical providers should routinely train with other area HEMS programs, EMS agencies, fire departments, and hospital providers. This training should include aircraft familiarization, landing zone requirements, flight activation criteria, and flight request training.

Advanced technology is now available that can increase the safety of flight operations. Many HEMS programs are now implementing night vision goggles **Figure 3-12** on the aircraft. Unfortunately, the implementation throughout the industry is slow because of the cost of the equipment, required modifications to the aircraft cabin, and extensive flight crew training.

Figure 3-12 Night vision goggles.

As mentioned earlier in this chapter, flight-tracking software is now used throughout the industry to assist with realtime tracking of the aircraft at all times. The practice of flight following helps locate an aircraft that crashes or loses radio contact. Some air medical programs have also begun implementing ground-proximity warning systems and obstacle detection and alerting systems on the aircraft.

Most rotor-wing aircraft do not have real-time weather data available in the aircraft. Instead, the pilot must rely on weather data obtained before launch as well as weather information given by the flight-following center. To provide for better in-progress forecasts, some aircraft now have weather display and alerting systems installed that can give pilots realtime weather data and warnings.

Cockpit voice recording and flight data recorders have been used for years in fixed-wing aircraft. Flight safety advocates are now asking for rotor-wing aircraft to have the same technology so that investigators can better analyze the factors that lead to a crash or near-crash situation.

Crew Resource Management

With the implementation of cockpit voice recorders and flight recorders in the 1960s and 1970s, investigators found that many crashes were not the result of mechanical or technical malfunctions of the aircraft or its systems, nor were they caused by poor piloting skills or lack of knowledge or experience from the crew. Instead, more than 80% of crashes involved human error—primarily related to the inability of the crew to respond appropriately to situations in which they found themselves.

Crew resource management evolved as a result of that statistic and the KLM/Pan Am crash on Tenerife, Canary Islands, in 1977, in which two 747s collided on the runway. This was the deadliest crash in aviation history, and was primarily due to unclear communication between the KLM pilot and the control tower regarding takeoff. The pilot understood that they were cleared for takeoff, but the control tower was only confirming position for takeoff, not actually clearing the flight for takeoff. The control tower made a clarifying statement, but KLM could not hear it because of interference. The Pan Am aircraft was taxiing on the main runway when the KLM aircraft started to take off. The KLM flight engineer heard a transmission from the Pan Am crew and asked the pilots if Pan Am was clear of the runway; the KLM pilot assumed Pan Am was clear and continued with takeoff. All KLM passengers and crew members died (248 total people), and most (335) of the 396 people on the Pan Am flight died.

In response to this crash and alarming statistics, the National Aeronautical and Space Administration (NASA) held a workshop in 1979 focusing on ways to improve air safety. Before this workshop, the captain or pilot in command of the aircraft was considered to be the only person who was able to make decisions regarding the operations of the aircraft. The other crew members, including the first officer, had

minimal input into the final decision that was made. The new procedure advocated as a means to overcome this reliance on only one person's judgment was called **crew resource management**, defined as "using all available resources—information, equipment, and people—to achieve safe and efficient flight operations."

Crew resource management challenged the assumption that the pilot should make decisions alone. Flight crews were encouraged to speak up when they did not agree with the pilot; pilots were encouraged to listen and take into account flight crew input. In short, the aviation industry learned to accept that human-to-human interactions are an integral part of any team performance.

Today, the **International Civil Aviation Organization (ICAO)** requires crew resource management for airlines in 185 countries. A study by the University of Texas Human Factors Research Project found that commercial airline flights experience an average of four threats per flight. Most of these threats or errors are caught through crew resource management and have no detrimental consequences.

Crew resource management is not concerned with the technical aspects of flying an aircraft. Instead, it focuses on the cognitive and interpersonal skills needed to successfully complete the flight:

- Cognitive skills take into account the mental processes needed to maintain situational awareness, make decisions, and solve problems.
- Interpersonal skills focus on individual and group behavior, communication, and teamwork.

Crew resource management allows for subordinate crew members to participate in the decision-making process, but it does not imply that all decisions are made by committee without considering rank. That is, the amount of participation by subordinate crew members depends on the situation. For the most part, the medical crew member's role in crew resource management focuses on passive monitoring. Such crew members typically do not provide flight instructions to the pilot, nor will they be involved in technical decisions made in regard to operation of the aircraft. Instead, the obligation of the medical crew member is to intervene if the level of skill being displayed by the pilot falls below a safe standard (eg, if the medical crew member notices that the aircraft is on an imminent collision course with a radio tower, or to advise the pilot of nearby air traffic).

One area of crew resource management in which all members of the crew should hold equal empowerment is the decision of whether to accept a mission. In the past, it was often solely the pilot's decision to accept or decline a mission. More recently, this practice has been changed by the majority of air medical providers and replaced by the "three to go, one to say no" rule (also called the 51% rule, which means it takes all members of the crew to agree to take the mission, but if one member says no, the mission is declined). Under this rule, every member of the crew—regardless of time or grade—has the power to decline a flight. Although the pilot does have the ultimate authority over all operations of the aircraft, if any crew member does not feel comfortable with a mission, the mission should be aborted.

Medical crew members must never wonder what is going on in regard to operation of the aircraft. If this occurs, either the pilot has not announced his or her actions or is not aware of the situation. Consider the following scenario: The pilot says he or she is going to hold in a hover. The aircraft begins to hover, but then slowly drifts backward. The crew needs to communicate to the pilot that they are drifting backward or they may miss an opportunity to correct an error.

Types of Human Errors

According to the NTSB, "Accidents are never caused by one single event. Instead, they are caused by a series of factors that contribute to the final event." Human factors contributing to error can be classified into three types—those that involve skill deficiency, perception errors, and decision-making errors.

Pilots and other members of the flight crew are expected to have the basic skills to fly an aircraft

safely. When there are deficiencies in these basic skills, a number of hazards can occur. Examples of the types of errors that can occur include losing control of the aircraft on the runway, flying at an improper speed, and not following standard operating procedures in the event of an emergency.

Errors in perception involve **spatial disorientation** (covered in Chapter 4), a **somatogravic illusion**, or a mistake in judgment regarding distance, altitude, or airspeed. Spacial disorientation may occur during a flight that becomes out of control in midair. Errors in perception also occur during nighttime landings and landings that involve reduced visibility.

Errors in decision making also may lead to an undesirable outcome. Decision-making errors include failing to choose the appropriate emergency procedure and failing to take corrective action when the aircraft is flying below minimum altitude.

Air Medical Safety

Air medical transport has inherent hazards but is rich with rewards. Air medical providers encounter a complex myriad of hazards. They are expected to rapidly launch after a request has been made (most rotor-wing programs require launch within 5 minutes of the request), placing stress on the flight crew to become airborne. Air medical aircraft often land at chaotic scenes where a hasty landing zone has been established **Figure 3-13** and where a variety of emergency personnel are operating in close proximity to the landing zone. Air medical providers are called to situations where patients are facing catastrophic injury or illness, requiring the flight crew to give maximum attention and decision-making abilities to the treatment of the patient. Frequently the aircraft is called because the patient's condition is so serious that it has exceeded the capabilities of the medical personnel on the ground, putting additional stress on the air crew. Finally, the aircraft environment is physically demanding and can induce heat illness, dehydration, motion sickness, and physical and mental exhaustion in air medical personnel after extended operations.

Figure 3-13 Chaos of a scene flight.

Risk Factors

Retrospective studies regarding air medical crashes conducted by the NTSB, the FAA, and the **Air Medical Physician Association (AMPA)** have found several areas within HEMS operations that need improvement as follows:

- Weather forecasting
- Flight operations during instrument meteorological conditions
- Personnel training

- Design standards
- Crashworthiness
- Operations management

Other areas of HEMS operations identified as risk factors for air medical personnel include the following:

- Unprepared landing sites
- Complacency
- Additional stress of responding and caring for critical patients

Safety reports also indicate that communication problems with air traffic control and collision with ground objects are problems that warrant improvement.

Adding to the risk is the time-critical nature of the job. As mentioned, rotor-wing providers are generally expected to launch within 5 minutes of receiving a request for services. Because of the rapid preparation involved, crews do not have the luxury of conducting a leisurely preflight inspection of the aircraft, nor do they have plenty of time to check and recheck weather. Instead, crews must move quickly to provide timely, life-saving care. Use of crew resource management has improved the decision-making process after receipt of a request. Most program policies have all flight crew members perform a quick walk-around the aircraft prior to initial start up. The rule of thumb is that three sets of eyes are better than one. Usually all crew members report to the front of the aircraft after performing the walk-around and report any abnormal findings before the pilot enters the cockpit.

As mentioned earlier, a disproportionate percentage of crashes occur at night. A study by the AMPA in November 2002 concluded that the time of day when the flight takes place has a significant impact on safety. The study found that more crashes occurred when crews were en route to pick up a patient than at any other time during flight.

The FAA has taken steps to increase the safety of HEMS operations. In 2004, the Helicopter Air Ambulance Task Force was created to make recommendations to reduce the crash rate. Some of the recommended safety measures include the following:

- Stricter weather guidelines
- Night vision goggles
- Instrument-rated pilots and aircraft
- Dual pilots
- Enhanced pilot minimum qualifications
- Better usage criteria

As yet, these are FAA recommendations and not regulations.

Federal Aviation Administration Guidelines

All aircraft fall under one or both of the following guidelines set forth by the FAA:

- **Title 14 Code of Federal Regulations (CFR) Part 91** governs the operation of all aircraft within the United States, including the waters within three nautical miles of the US coast. All aircraft must abide by this guideline. General aviation (private) aircraft operate under these less restrictive rules.
- **Title 14 Code of Federal Regulations (CFR) Part 135** governs the operations of all commuter or on-demand commercial operations. The guidelines for 14 CFR Part 135 operations are much stricter than those for 14 CFR Part 91 aircraft and apply to almost all air medical operators.

One of the significant differences between 14 CFR Part 91 and 14 CFR Part 135 is the minimum weather conditions in which the aircraft is allowed to operate. Section 91.155 in 14 CFR Part 91, titled "Basic VFR Weather Minimums," states that helicopters must remain clear of clouds when operating less than 1,200′ above the ground. By contrast, the stricter 14 CFR Part 135 guidelines specify that HEMS aircraft must have a weather minimum of at least 1,000′ ceilings and 3 miles of visibility. A pilot could potentially accept a mission under 14 CFR Part 91 weather requirements, fly to the scene, and then be under 14 CFR Part 135 weather requirements for the transport to the hospital. This discrepancy poses a major safety hazard in that the aircraft would be allowed to launch under weather standards that are not conducive for patient transport with the hopes that the weather will improve by the time the patient is loaded on the aircraft.

If a flight is unable to be flown under 14 CFR Part 135 weather minimums for the complete flight, the flight should not be attempted. A retrospective review of 55 HEMS accidents that occurred from January 2002 to January 2005 found that 10 of the flights crashed while operating under 14 CFR Part 91 weather minimums; they would not have been able to take the mission if they were operating under 14 CFR Part 135 for the complete mission. For the purposes of the CCTP, only Part 135 is relevant as all commercial operators must abide by Part 135 regulations. Only a few government operators (such as law enforcement) can operate solely under Part 91.

The FAA, in addition to providing the requirements of 14 CFR Part 91 and 14 CFR Part 135, requires that air medical transport programs designate someone as **operational control**. This term is usually given to the chief pilot or operational manager for an air medical program and denotes the person who has the ultimate authority to initiate, conduct, and terminate the EMS mission. Although the pilot and the medical crew members have the right to accept or refuse a mission, the person in operational control has ultimate authority over all of the flight operations for the program.

Weather-Related Issues

Weather has an obvious impact on the ability to accept and complete missions. Some mission requests result in a **weather decline**—that is, an air medical provider is requested, but the pilot and the flight team determine that the weather does not meet established company or FAA guidelines for flight operations. The FAA states the following weather requirements in FAR Part 135, Section 135.201:

> *Except when necessary for takeoff and landing, no person may operate under VFR*
>
> *(a) An airplane*
>
> *(1) During the day, below 500 feet above the surface or less than 500 feet horizontally from any obstacle; or*
>
> *(2) At night, at an altitude less than 1,000 feet above the highest obstacle within a horizontal distance of 5 miles from the course intended to be flown or, in designated mountainous terrain, less than 2,000 feet above the highest obstacle within a horizontal distance of 5 miles from the course intended to be flown; or*
>
> *(3) A helicopter over a congested area at an altitude less than 300 feet above the surface*

CAMTS has established weather guidelines that take into account several variables, including local area response, crosscountry response, nonmountainous and mountainous areas, day light, night with high light, and night with low light. These standards exceed the requirements established by the FAA and have been accepted by many air medical providers. All air medical providers, regardless of whether they are CAMTS certified, are encouraged to follow the CAMTS-recommended weather standards for operations.

Crew Safety Precautions

CAMTS strongly emphasizes the need for adequate crew rest and ongoing safety precautions, both of which affect CCTPs and patients. The following are standards that CAMTS suggests for all CCTPs:

- Shifts longer than 24 hours are discouraged.
- Personnel should have at least 8 hours of rest before any shift longer than 12 hours.
- Personnel should not be on duty more than 16 hours within any 24-hour span.
- Crew members who must work longer than 16 hours must have the right to take an unscheduled break.

Other requirements have been adopted by CAMTS for pilots. Some CAMTS standards indicate that pilots must have the following:

- A minimum of 2,000 total flight time hours, with 1,500 of the hours in a helicopter
- 1,000 hours qualified as pilot-in-command time
- 200 hours of night flying
- A minimum of 500 hours of turbine time, with 1,000 hours encouraged
- 5 hours of geographic orientation with another pilot before accepting a mission alone

The Air Medical Crew

Being part of the air medical crew is competitive. Air medical personnel must have flight experience, but the only way to obtain experience is to be part of the crew. Most air medical services routinely have between 10 and 50 qualified applicants for a single flight position. Within the medical industry, a flight position is one of the most difficult to obtain. Working as a flight paramedic, a flight nurse, a flight respiratory therapist, or a flight surgeon is often viewed as the apex of the profession. That being said, an interested applicant should not be discouraged. Often you have to have considerable ground experience at both scene response and interfacility transports before being considered for a flight position. If your dream is to be a flight crew member, get the required experience, and apply broadly.

Because of the severity of the patients seen on the aircraft, flight team members must be clinically competent and prepared to deal with a myriad of medical and traumatic emergencies. Medical flight team members are sometimes only called to provide rapid transport to an appropriate facility, and other times are called to a scene or a hospital because the severity of the patient's condition requires their expertise. Flight team members must be willing to obtain numerous advanced certifications so that they will be prepared to care for the most critical patients.

Air Medical Crew Configuration

Several different medical crew configurations are used throughout the industry. Studies have not shown a definitive increase in survivability with any particular crew combination. Ultimately, the best crew configuration is one that is able to offer the most optimal care to the patient.

Air medical transport improves mortality rates when the HEMS crew capabilities exceed that of noncritical care ground personnel. In particular, air medical programs that operate under physician-level standing protocols experience lower mortality rates. Those programs that operate under aggressive protocols with an advanced scope of practice have the best opportunity to improve patient outcome. Protocols alone, however, do not make the CCTP more expert than ground personnel. Continued practice, experience, and education combine with aggressive medical protocols to lead to better patient outcomes.

In the air medical environment, the most common configuration is flight nurse/flight paramedic, a synergistic combination that provides a great amount of experience and diversified training in the medical

crew. The flight nurse is well experienced in the critical care environment, whereas the flight paramedic is well adapted to airway management and emergency scene management. Both providers are proficient in dealing with the comorbid or multitrauma patient.

The nurse/respiratory therapist configuration offers advantages in that the most important aspect of treatment is airway management. Some air medical transport agencies fly a nurse/nurse combination, although this configuration is controversial as nurses may not have the necessary prehospital experience and airway management experience. Similar to the nurse/nurse combination is the paramedic/paramedic combination, whose configuration does not provide the advanced scope of practice that would be seen with the addition of a flight nurse.

Air medical services that transport patients on balloon pumps may utilize perfusionists on the flight team. These highly trained technicians are experts in the operation of intra-aortic balloon pumps and adult and pediatric extracorporeal membrane oxygenation machines.

A few air medical transport teams include physicians; however, given the increased scope of practice of flight team personnel, most air medical services have found that having a physician onboard does not significantly increase patient survival.

Air Medical Crew Employment

Working on a rotor-wing or fixed-wing aircraft is demanding and requires personnel to be highly proficient in a myriad of prehospital and critical care skills. Paramedics and nurses may treat a critical trauma or medical patient in fewer than 10% of their cases; by contrast, flight crew personnel encounter critical patients on almost every mission. **Table 3-5** lists industry minimums and recommendations for training flight nurses, flight paramedics, and flight respiratory therapists. Individual states may require additional training prior to employment as a flight crew member.

TABLE 3-5 Industry Minimums and Recommendations for Air Medical Personnel

Position	Required Minimums	Recommended
Flight nurse	Five years of experience as a nurse with critical care and emergency department experience Current certifications in: • Advanced Cardiac Life Support (ACLS) • Pediatric Advanced Life Support (PALS) • Neonatal Advanced Life Support (NALS) or Neonatal Resuscitation Provider (NRP) • International Trauma Life Support (ITLS) or PreHospital Trauma Life Support (PHTLS) • Cardiopulmonary Resuscitation Provider (CPR)	Bachelor's/Master's degree Advanced Trauma Life Support (ATLS) audit Certified Emergency Nurse (CEN) Certified Transport Registered Nurse (CTRN) Certified Flight Registered Nurse (CFRN) Critical Care Registered Nurse (CCRN) Emergency Medical Technician certification Paramedic certification TNATC, TNCC, ATCN Instructor credentials in ACLS, PALS, NALS, ITLS, PHTLS, and CPR
	Five years of experience as a	

Flight paramedic	paramedic in a busy 911-response EMS system National Registry Paramedic (NREMT-P) • Advanced Cardiac Life Support (ACLS) • Pediatric Advanced Life Support (PALS) • Neonatal Advanced Life Support (NALS) or Neonatal Resuscitation Provider (NRP) • International Trauma Life Support (ITLS) or PreHospital Trauma Life Support (PHTLS) • Cardiopulmonary Resuscitation Provider (CPR)	Bachelor's degree Critical Care Paramedic (CCEMT-P, or CCP) Certified Flight Paramedic (FP-C) Paramedic instructor Instructor credentials in ACLS, PALS, NALS, ITLS, PHTLS, and CPR
Flight respiratory therapist	Bachelor's degree in respiratory therapy Registered Respiratory Therapist (RRT) Three years of critical care experience (including adults, pediatrics, and neonates) EMT certified Current certifications in: • Advanced Cardiac Life Support (ACLS) • Pediatric Advanced Life Support (PALS) • Neonatal Advanced Life Support (NALS) or Neonatal Resuscitation Provider (NRP) • International Trauma Life Support (ITLS) or PreHospital Trauma Life Support (PHTLS) • Cardiopulmonary Resuscitation Provider (CPR)	Paramedic certification Instructor credentials in ACLS, PALS, NALS, ITLS, PHTLS, and CPR Member of the American Association of Respiratory Care (AARC) and member of the Surface and Air Transport specialty section

Rotor-Wing Operations

Rotor-wing aircraft are generally operating at maximum capacity when they complete 1,000 flights or

more per year. When a service completes 1,000 flights, it can be safely assumed that there were at least 1,500 requests for services.

One of these requests for service comes in the form of a **standby**. A standby generally occurs when an HEMS provider is notified of a potentially life-threatening emergency call. As the paramedics on the ambulance are responding, they may put the local rotor-wing provider on standby, notifying it of a potential request for a response. When placed on standby, members of the air medical crew will check weather suitability (and may decline the mission if the weather does not meet FAA minimums both for launch and for the duration of the flight), gather necessary equipment (ie, blood, warmed fluids), and conduct a preflight inspection on the aircraft **Figure 3-14**. They will also obtain landing coordinates and appropriate radio frequencies. The flight crew will then wait at the aircraft until they are launched or cancelled.

Figure 3-14 The air medical crew conducts a preflight inspection.

Some programs have an **auto launch** protocol. Under this system, when the aircraft is placed on standby, the aircraft launches en route to the scene instead of waiting for the formal launch request. This measure is frequently taken for responses that involve distances of greater than 30 nautical miles. This procedure is often not practical given the considerable cost and inherent hazards in launching the aircraft.

Some emergency medical services make an HEMS standby request automatically as part of their dispatch protocol, depending on the severity of the call. In such a case, the EMS places the aircraft on standby based on the 9-1-1 caller's information instead of waiting for the first emergency responder to arrive. When the location of the scene is greater than 30 miles, the aircraft will usually go ahead and launch to be closer to the scene if it is actually needed. There are clear advantages to early notification of HEMS in more expeditious response, reducing both underuse and overuse, and employment of the myriad of first-hand and crash reporting data often received in the 9-1-1 center.

Establishing a Helicopter Landing Zone

One of the most dangerous aspects of rotor-wing air medical operations is landing the aircraft at scenes where a landing zone has been hastily established. Ideally, the aircraft should be landing at predesignated landing zones that have already been carefully assessed for hazards. Many fire departments and EMS agencies have established numerous landing zones throughout their response area. The coordinates for these landing zones are recorded and given to their dispatch center to pass along to the HEMS program upon launch activation.

When a landing zone needs to be established, several factors must be considered. Ideally, the landing zone will be at least 100′ × 100′ **Figure 3-15**. Some programs require 60′ × 60′ during the day and 100′ ×

100′ during the night. The actual landing surface does not have to be this wide; rather, the site needs to be clear of any obstacles for at least 100′ × 100′. In particular, the landing zone should be free of the following obstacles:

- Wire (no high-tension power lines within half a mile)
- Towers (no television, radio, or cellular towers within half a mile)
- Trees
- Signs and poles
- Low-height ground obstacles such as stumps, small trees, fence posts, mailboxes, and rocks
- Buildings
- Vehicles
- People
- Animals
- Loose debris (traffic barrels or trash)

The landing zone should be a hard surface, rather than a field or other undeveloped or uncleared surface. A hard landing surface facilitates moving the stretcher to and from the aircraft, and fewer personnel are required to load the patient. Unimproved surfaces also have the potential for more dust, dirt, and debris to be blown about by the rotor wash from the aircraft. The landing zone also needs to have less than a 5° slope to avoid the danger of the blades coming in contact with an object or a person on a slope. Most rotor blades droop significantly when they are idling on the ground.

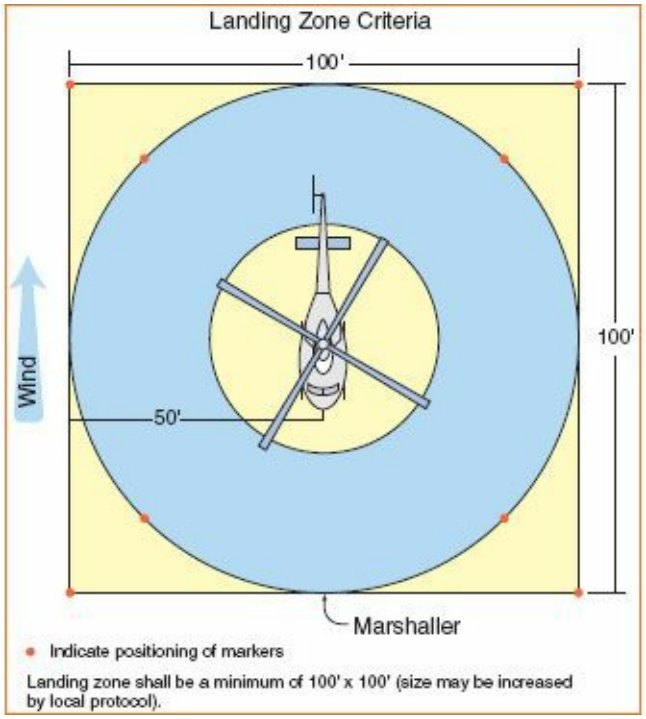

Figure 3-15 Establishing a landing zone.

In ideal circumstances, the landing zone will be outlined by four markers, one on each corner of the landing zone **Figure 3-16**. The markers could be cones for daytime operations and lighted markers for nighttime operations. In the absence of markers, vehicles can be used to establish the landing zone by blocking off access to the area. Emergency vehicles can also be placed so that their headlights cross to mark the landing zone **Figure 3-17**. When emergency vehicles are used to establish the landing zone, it is recommended that they have all of their emergency lights on to enable the aircraft to quickly identify the

landing zone. On clear nights, the aircraft can see the lights from emergency vehicles from several miles away, making it easy to identify the landing zone.

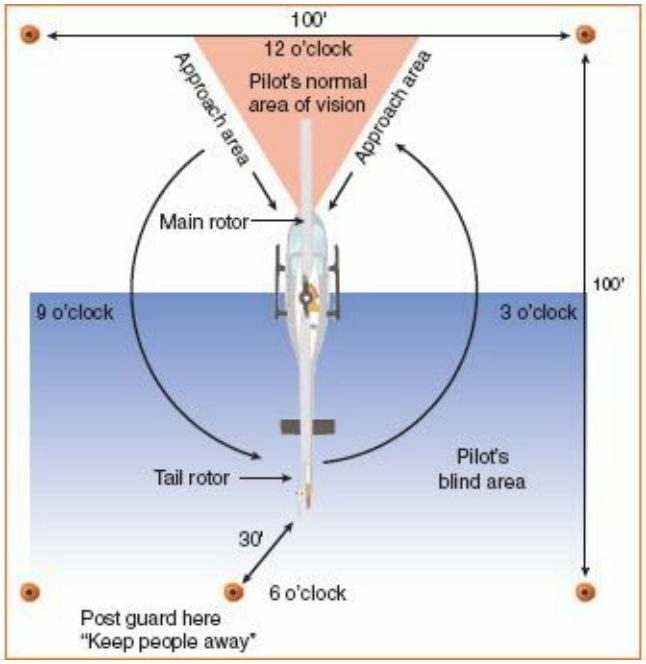

Figure 3-16 Landing zone with cones/warning devices in place.

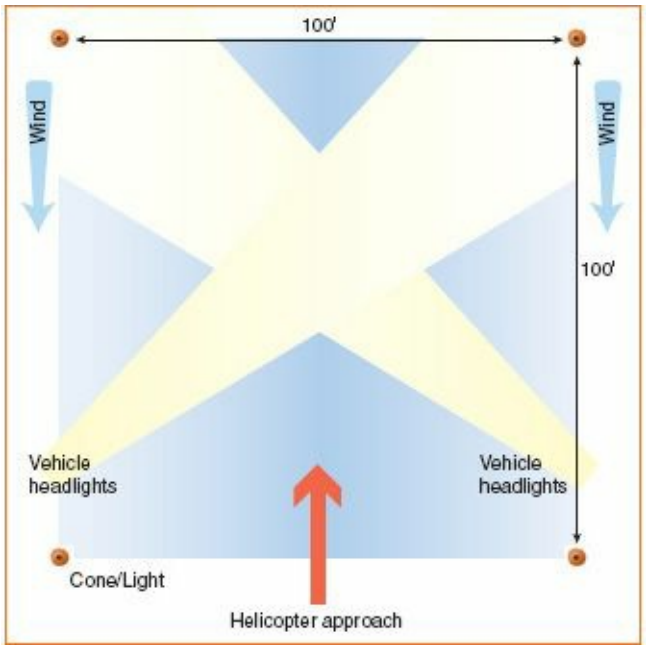

Figure 3-17 Use of headlights to mark a landing zone.

The use of strobe lights is discouraged at the landing zone because of their propensity to cause temporary blindness or disorientation of the flight crew. The use of any flammable type of lights such as flares is not allowed at the landing zone.

Landing zone personnel should be taught proper hand signals to assist the helicopter with landing **Figure 3-18**. Although basic helicopter hand signals are universal, it is recommended that the pilot coordinate the training of hand signal use, as he or she may prefer particular signals.

Precautions After Landing the Helicopter

All landing zone personnel should wear eye and ear protection at all times to protect themselves from

flying debris and the noise of the aircraft. All loose items should be secure, including helmets, equipment, and sheets from the patient stretcher.

No personnel should ever operate near the tail rotor. The tail rotor spins so fast that it is nearly invisible. At night, it is almost impossible to see the tail rotor. This is the area of greatest hazard on the aircraft and all precautions should be taken to ensure that no one comes near the tail of the aircraft. When patients are being loaded into the aircraft by personnel such as fire fighters, paramedics, or nurses, one member of the flight team (usually the pilot) should stand between the auxiliary personnel and the tail rotor to ensure that no one operates in that area. No personnel other than the flight crew should be aft of the loading door of the aircraft.

At no point should anyone approach the aircraft unless directed by the flight crew, and then only from the front right side of the aircraft in full view of the pilot. With some aircraft, the lowest point of blade dip is at the front of the aircraft. Most aircraft are designed to be approached from a 90° angle to the doors. Every aircraft is different, and personnel must abide by manufacturer recommendations and pilot instructions. The key point is never approach unless the pilot indicates it is safe to do so and then only following the pilot's directions.

Hot Loading

Hot loading refers to loading a patient into a helicopter while the helicopter is running. Whether or not a service performs a hot load depends on many factors, including the type of landing zone, type of helicopter, and scene time. For example, a hot load may be necessary when there is no time to power-down and power-up the helicopter, or if there is any question about being able to start up the helicopter in the location where it landed.

When you are loading and unloading a rotor-wing aircraft, certain precautionary measures must be observed. Failure to observe proper safety procedures could result in severe injury or death. Each service must follow a specific, consistent hot load procedure. This procedure needs to consider the helicopter's design; for example, some helicopters load from the side while others load from the rear. The type of load performed should be discussed before landing at the scene. As always, crew must wear proper personal protective equipment.

Although the specific procedure varies, it is critical to have safeguards in place to ensure that no one approaches the tail rotor. One method to address this is to assign a crew member as a tail rotor guard. The tail rotor guard and pilot must be in direct communication with each other or must be able to see each other. The tail rotor guard must remain in position until all assisting personnel have safely moved away from the helicopter.

Before a hot load is done, the crew needs to determine that it is safe before starting. When the hot load is being performed, the pilot and at least one other person remain in the helicopter. The helicopter must not be approached until a crew member signals to do so. The air medical crew and assisting personnel approach the helicopter only once they have been given the go-ahead. The aircraft should be approached in a crouched position. If the helicopter is on a slope and conditions permit, loading personnel should approach the aircraft from the downhill side. Once the patient is loaded, any assisting ground personnel exit the aircraft.

Aircraft Safety and Survival

Numerous books have been written about safety and survival. This section briefly addresses some of the key points in aircraft safety and survival of a hard landing or crash. Medical flight team members must have thorough knowledge of aircraft safety and survival. A large portion of this knowledge must come from hands-on training; it cannot be accomplished by simply reading a chapter in a book, or even by

reading a complete text dedicated to safety and survival.

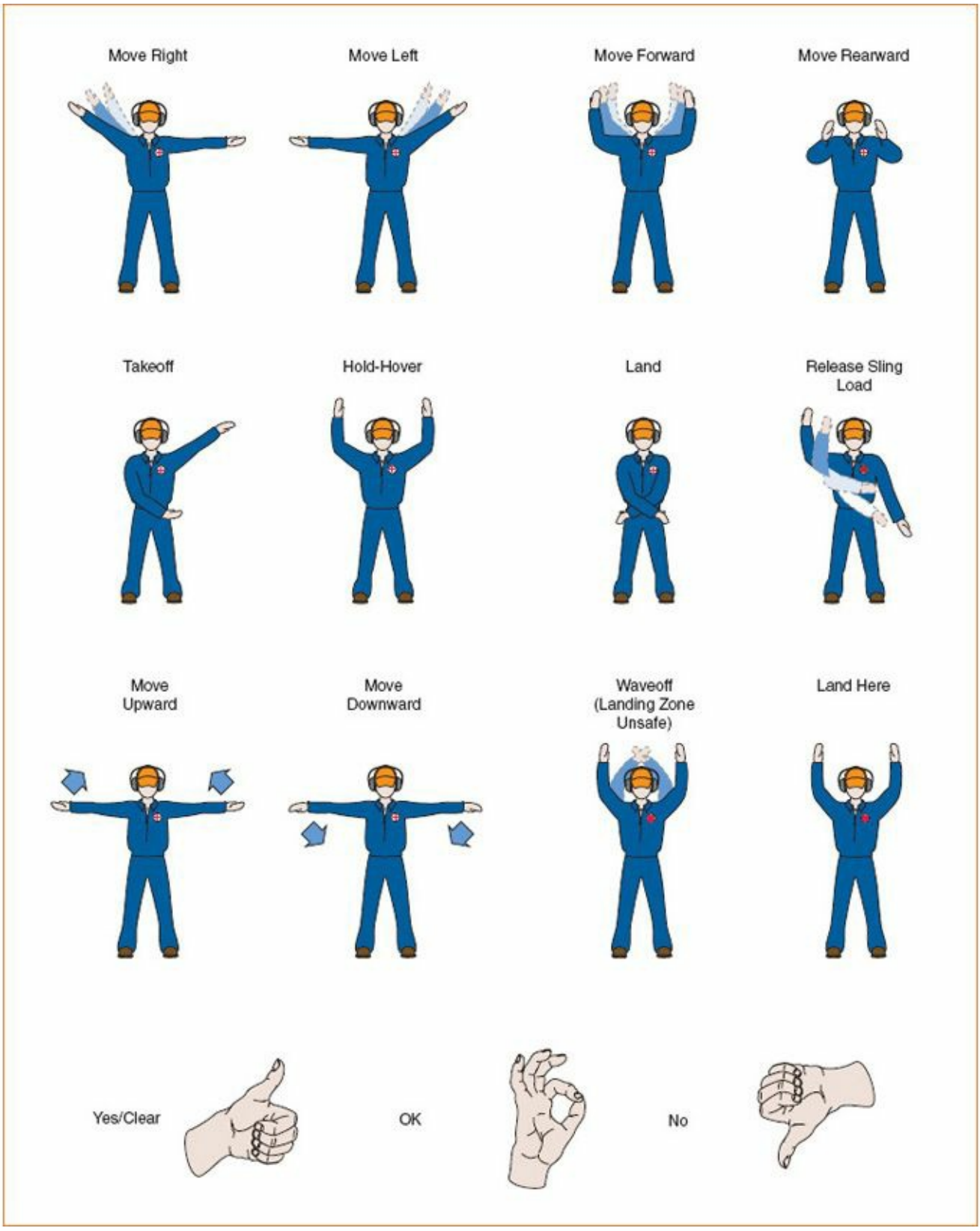

Figure 3-18 Helicopter hand signals.

Safety Training

All flight team members must have initial training on aircraft safety and survival, and then undergo refresher training annually. Every member of the flight team must be familiar with the different types of aircraft that are operated by the program. It is also useful for flight team members to be familiar with the different types of aircraft that are used by programs located in nearby areas in case they operate on a dual

mission and need to assist with loading or offloading patients. Also, if flight team members are hospital based, they will routinely assist with aircraft loading and unloading on their hospital's helipad.

A comprehensive safety training program needs to address the following issues:

- Aircraft familiarity
- Behavioral characteristics of aircraft during hard landing sequence/crash landing
- Equipment storage
- Personal protective equipment
- Egress training

For example, flight team personnel need to routinely train on evacuating the aircraft while blindfolded to simulate a smoke-filled environment. During training, personnel should be instructed to use aircraft exits that they normally would not use, so as to simulate blocked or jammed doors. Programs that fly over water should also incorporate water egress training using a simulator called an aircraft dunker.

Daily Safety Briefing

To protect patients and all members of the critical care transport team, it is imperative to be familiar with the inherent occupational safety risks. Critical care transport teams should hold **daily safety briefings** at the beginning of each scheduled shift. These briefings should include the daily plan, crew member duties, equipment and aircraft issues, weather expectations, and emergency situations **Figure 3-19**.

Although crew members share in all aspects of each mission, individual crew members often agree to assume primary responsibility for certain duties during the shift. For example, one crew member may agree to manage blood products, and the other may be responsible for medications. Even though one crew member may take primary responsibility for a task, the overall responsibility of all aspects of the shift is shared. Other issues that are usually addressed include reminders of general safety issues such as maintaining two pairs of hands on the stretcher, no storage of medical equipment on a step or skid, and crew comfort level.

During the daily safety briefing, the pilot in command also briefs crew members on weather forecasts and determines if the crew will be ready to respond or if a weather check is needed before making a "go or no go" transport decision.

Figure 3-19 Before each shift, the pilot gives the daily safety briefing.

Crashes and Hard Landing Operations

Several circumstances may result in unscheduled landings or hard landings. An unscheduled landing may

be a normal landing but occurs at a point other than the intended landing site. A **hard landing** is when the aircraft contacts the ground harder than normal, possibly resulting in injury or aircraft damage. Unscheduled landings are primarily due to either unexpected weather (IFR situations with a VFR-rated pilot or aircraft) or a non–life-threatening mechanical problem with the aircraft. Hard landings can result from any of the following:

- Engine failure
- Mechanical failure
- Wire strike
- Pilot error
- Incapacitated pilot

Use of Radios and Emergency Locator Transmitters

In the event of an aircraft emergency, medical flight crew members may be required to switch VHF radio frequencies to the international mayday frequency of 121.5 MHz. This frequency is always monitored by air traffic controllers, military agencies, and search-and-rescue groups.

Most aircraft today carry an **emergency locator transmitter (ELT)** on board; indeed, most aircraft operators are mandated by the FAA to have an ELT on board **Figure 3-20**. ELTs are designed to activate with 4g of force in the event of a crash. The ELTs transmit a distress radiobeacon to satellites in geostationary and low-altitude earth orbits. These satellites then transmit the signal to distress radiobeacons, which are monitored at ground receiving stations referred to as local user terminals. These terminals receive and process the satellite downlink, allowing rescue agencies to coordinate search-and-rescue missions.

Preparing for a Crash or Hard Landing

Crew members may only have seconds to react if a hard landing or crash landing is going to occur. If the crew realizes that the aircraft will impact the ground, the oxygen needs to be immediately shut off. If there is a patient on board, the head of the stretcher should be elevated to 30°, or per manufacturer's instructions for a hard landing. Crew members should also make sure their seatbelts are tight and their helmet straps are secured. They should ensure that they are free from entanglement hazards such as the communication system or monitor cables and cords. Crew members should brace for impact, ideally maintaining one hand on the aircraft door to maintain a point of reference for exit after the crash.

Figure 3-20 An emergency locator transmitter.

The best time to prepare for a crash is before it happens. CCTPs must be physically and mentally

prepared to survive **Figure 3-21**. The following rules should be practiced until they become instinctive:

- Never exit a crashed helicopter until the blades stop turning. After a crash, blades may not function properly and could cause grave injuries if they come into contact with a person.
- After exiting the helicopter, all crew members should meet at the 12 o'clock position, which is directly off the nose of the aircraft (shown in Figure 3-16), *before* attempting to rescue crew members or patients still in the aircraft. Individual safety comes *first*. If there is fire or debris at the 12 o'clock position, crew members should assemble at a safe location nearby.
- If the pilot is incapacitated, crew members should be proficient in operating throttle, fuel, and battery switches to decrease the likelihood of fire and explosions.
- In the event of a crash into water, the helicopter will likely flip because it is top heavy. Crew members should place one hand on the ceiling of the aircraft to use as a reference point. Doing so enables them to reference up and down and in and out directions. The general rule is to follow your bubbles as they always lead to the surface. Reference points should also be used in darkness and when smoke is present.

Figure 3-21 After a crash, CCTPs must properly exit the aircraft and avoid exposure to fire.

Dealing With the Aftermath of a Hard Landing

If the aircraft experiences a hard landing, several important steps must be followed. Upon landing, the flight team must ensure that all motion of the aircraft has stopped before attempting to exit. This is especially important in the rotor-wing environment, where the blades may still be spinning after the aircraft has landed. A good rule of thumb is to slowly count to five after the aircraft comes to a complete stop before attempting to exit. Once flight team members have exited the aircraft, their next priority is to attempt the immediate rescue of the other flight team members. If they are unable to extricate the other crew members or if a fire prevents such rescue, flight team members must proceed to a safe distance from the aircraft and assemble at a predesignated location. Unless otherwise stated by a specific program, the predesignated meeting point is at the 12 o'clock location in front of the aircraft. Once the flight team has assembled at this location, they can begin treating any injured persons and make plans for a rescue attempt for the patient in the aircraft if one was onboard.

Once the aircraft has cooled down and the immediate threat of fire has been resolved, the flight team needs to develop a plan of action. This plan must include the salvage of survival gear from the aircraft along with any usable pieces of the wreckage that may be used for shelter or signaling.

Safety and Survival After a Crash

As with a crash, the best time to prepare for survival is before the need to survive. Preparation and education in survival techniques increase the likelihood of a positive outcome and should be included in every critical care transport program.

At a minimum, the following elements should be included in survival training programs:

- Location and contents of the aircraft survival bag. Crew members need to be intimately familiar with each piece of survival equipment and its intended use.
- Directions for construction of a temporary shelter to protect crew members and patients from the elements. This training should include evaluation of a suitable location free from future dangers, the basic parts (roof, floor, and walls), and assembly of temporary shelter using limited resources.
- Survival priorities. This training should prepare crew members to prioritize their basic physiologic needs, using the **rule of 3s**, which prioritizes the basic physiologic needs for survival as follows: The average person can survive 3 minutes without oxygen, 3 hours without shelter, 3 days without water, and 3 weeks without food.

Upon exiting the aircraft after a crash, crew members must size up the situation, the available equipment, and the number and severity of everyone's injuries. Unless help is immediately visible, it is good practice to stay within close vicinity of the aircraft. It is much easier for rescuers to locate the wreckage of an aircraft than it is to find individual crew members. By staying near the aircraft, the team members also are able to salvage and use most of the parts of the aircraft for survival **Table 3-6**.

Previous rehearsal of emergency procedures usually results in automatic action on the part of the individual during a crisis. Through such training, you will also learn your own limitations. By knowing yourself, you can anticipate the fears you are likely to experience. The anticipation of fear can help you avoid panic, which is deadly. It is important to maintain a realistic, yet positive, attitude and remember what is at stake.

Air medical crews must be proficient in safety and survival operations. They need to routinely practice egress operations from the aircraft and activities after the crash. People will act in accordance with how they are trained. Proper, frequent training saves lives in the event of an aircraft emergency.

Increasing the Survival Odds

There are six important tips for increasing survival:

1. Conserve strength, fluid, and heat by moving as little as possible and as slowly as possible.
2. Prepare emergency signals, make shelter, inventory supplies, and ration all food.
3. Guard against infections and intestinal disorders.

TABLE 3-6 Aircraft Parts List and Usage

Aircraft Part	Potential Use
Battery	Signal lights, communication, fire starter
Cowlings	Signal panels, water collection, fire pit, shelter
Doors	Shelter, windbreak, signal panel
Engine oil and fuel	Fire starter, signal fire
Fuselage	Shelter
Seats	Insulation, fire material, signal material

Tires	Black smoke signal

4. Do not travel in adverse weather if injured or confused. If you stay put, eventually someone will find you. Numerous searches have found that search parties were inadvertently moving in concert with the victims, often within hundreds of yards of each other without realizing it.
5. Stay with the downed aircraft. The wreckage is easily spotted from the air, and it is the first place where the search will begin. Determine your location, and scout the immediate area for shelter, water, and food.
6. If you must travel, do not separate parties. Mark your path, leave messages behind, and keep a log.

The ability to signal is vitally important in increasing survivability. The first rule of signaling is to increase your visibility—whether by staying near the aircraft wreckage or moving to a nearby clearing or higher elevation. It is important to carefully consider the decision to leave the wreckage, as it is one of the first places where rescue crews will begin their search.

Hydration is of paramount importance following a crash. In survival operations, finding potable water is the immediate priority after shelter has been obtained. A person who uses thirst as a guide drinks only two thirds of his or her daily water requirement. To prevent dehydration, use the following guide:

- At temperatures below 100°F (38°C), drink 0.5 L of water every hour.
- At temperatures above 100°F (38°C), drink 1 L of water every hour.

Crash survivors must also guard against despair and protect themselves from pain, cold, thirst, fatigue, boredom, loneliness, and fear or panic. The number one killer of crash survival victims is panic. The four most common causes of death in the survival setting are panic, carelessness, faulty and inadequate equipment, and incorrect survival behavior. Powerlessness and despair increase fear and must be overcome. Decrease fear as follows through:

- Training
- Faith in your equipment
- Faith in the technical knowledge of the immediate superior
- Concentration on the task to accomplish
- Trust in providence/God

One of the most critical elements in ensuring survival is also the easiest to manage. Immediately after the crash, once you are in a safe area and have rescued all survivors, stop and sit down. Lean back against a tree or a boulder and relax. Force yourself to take six slow deep breaths. Think about your situation and plan out exactly what you need to do. This begins conservation of your physical and mental resources. By taking these steps, you have increased your survival chance by 50%!

Water Ditching

A recent NTSB report indicated that of 63 pilots who died after a crash into water (**water ditching**), 78% were located inside the aircraft and died of drowning; only 20% died as a result of their injuries or because of nonfatal incapacitation. Every year, approximately 30 to 40 general aviation ditchings occur in US coastal or inland waters. Most crew members will have less than 15 seconds' notice that the aircraft is going to ditch. If ditching is imminent, immediately assume the crash position and disconnect your headset to prevent an entanglement hazard. If there is time, tighten your seatbelt, turn off the oxygen, lay the patient flat, and secure all items.

Airplanes and helicopters act differently in water. Airplanes will usually float for several minutes before sinking. By contrast, after the helicopter makes contact with the water, it will usually capsize because it is top-heavy, which results from the weight of the engines and transmissions.

Once the aircraft makes contact with the water, four important steps need to be followed to successfully egress the aircraft.

First, resist the urge to exit the aircraft immediately; instead, sit and count to five to ensure that all motion of the aircraft has stopped. The rotor blades may continue to turn after the aircraft has hit the water, and counting to five will prevent crew members from exiting the aircraft and running into the path of spinning blades.

Second, identify and open your exit using the deliberate offset method **Figure 3-22**. To find the exit handle, put your hand on your knee, with your knee against the cabin wall. Start low and feel your way up until you find something you recognize, such as the armrest or a door seam. From there, work your way up to the exit handle. This exit may be jammed shut or blocked, so always know an alternate exit.

Third, grab hold of a reference point that you are familiar with in the direction of your exit. Do not release your belt until you have a secure grip on a reference point. If the aircraft is inverted, under water, or dark, you can become disoriented as soon as you release your seatbelt. Using the hand-over-hand method, you should always maintain one hand on a reference point and not let go of it until you grip another reference point.

Figure 3-22 The deliberate offset method. **A.** Put your hand on your knee, with your knee against the cabin wall. **B.** Starting low, feel your way up until you find something you recognize. **C.** From there, work your way up to the exit handle.

Fourth, once the exit is open, keep a hold on that reference point, release your seatbelt with the other hand, and pull yourself through your exit. As you exit the aircraft, do not kick; you could get stuck in wires or debris. If you become stuck, don't panic: Try backing up a bit and rotating a little before proceeding. Be prepared to encounter aviation fuel in the water, which can blind you. Also, remember to continually exhale as you ascend to the surface to prevent barotrauma. If you become disoriented in water as to the direction of the surface, you can follow your bubbles or swim opposite of the direction that causes pressure to build in your ears.

Summary

The air medical environment is a complex mix of aircraft operations and treatment of the critically ill and injured. Medical crew personnel must have a strong knowledge both in aircraft operations and in critical care treatment. Working in the air medical environment is filled with risks, and providers must take every step possible to limit their exposure to danger. Many different agencies are actively working to reduce the

risk of aircraft crashes, and flight team personnel are encouraged to become involved and aggressively work to make the industry safer. The air medical transport industry continues to grow, and flight team personnel must be ready to meet the myriad of challenges that they will inevitably face as air medical transport professionals.

Case Study

AT 3:25 AM ON A SUNDAY MORNING, you and your crew are fast asleep at your flight base. Your flight tones sound, alerting you and your crew of a pending flight.

Dispatch: Air One, Air One, you are requested for a scene flight. Coordinates and landing zone information will follow with acceptance of flight.

You and your crew jump out of bed, don your flight suits, and head for the aircraft. The pilot checks the weather to ensure that the ceiling and visibility are within night weather limitations. He gives the thumbs ups and alerts dispatch that the flight is a go. Before liftoff, the flight crew performs their walk-around of the aircraft.

The flight crew load into the aircraft, donning their flight helmets and seatbelts. The pilot asks, "Flight crew ready?" You respond, "Ready on right," and your partner responds, "Ready on left." The radio communication continues.

Dispatch: Air One is off the ground and en route to scene.

Dispatch: Air One, Air One, ready to copy coordinates.

Pilot: Dispatch, Air One is ready to copy coordinates.

Dispatch: Air One, your coordinates are 34° 3′ 5′ N, 118° 14′ 57′ W You will be landing in a parking lot, which has a 100′ × 100′ area cleared of all obstructions and hazards. The fire department is on scene and will be coordinating the landing zone. The fire department will be on frequency 15 for further communication.

Dispatch: Air One medical crew, ready to copy patient information.

Air One Medical Crew: Dispatch, Air One medical crew is ready to copy patient information.

Dispatch: You are responding to a two-vehicle motor vehicle collision. Your patient is a 25-year-old male with major head trauma, distended abdomen, and a deformed right femur. Vital signs are blood pressure 90 by palpation, pulse of 132 beats/min, respiratory rate of 28 breaths/min.

Air One Medical Crew: Copy patient information.

Air One Medical Crew: Local fire department, this is Air One, we have a 5-minute ETA to the scene. Can you provide us with updated landing zone information?

Local Fire Department: Air One, you will be landing in a parking lot that has been cleared of all vehicles. Local law enforcement has secured the scene to prevent any vehicles from entering into the landing zone. The landing zone is 100′ × 100′. Power lines are on the north side of the landing zone. Power lines will be marked with a police vehicle under the lines with its running lights on. The landing surface is level and absent of further obstructions and hazards.

Air One Medical Crew: Copy landing zone information, we have an ETA of 2 minutes. Because of the patient's condition, we will be remaining hot once we land. Do you have a landing zone coordinator who will be maintaining a secure and safe landing zone after we exit the aircraft?"

Local Fire Department: Roger that, we will ensure that bystanders are kept away from the aircraft.

The scene becomes visible to Air One. The pilot performs a high reconnaissance of the landing zone. The pilot and crew all agree that the landing zone is adequate and safe to land. The pilot makes a slow approach into the landing zone; the flight crew is looking out the right and left windows. You state "clear right" and your partner states "clear left." The pilot brings the aircraft in slowly and touches down. The flight crew awaits permission from the pilot to exit the aircraft.

The crew members exit the aircraft and are instructed on the location of the patient. They are informed that the patient is located in ambulance 32. The flight crew approaches the ambulance and enters through the rear door of the ambulance. They receive an updated report on the patient. The medic on the scene reports that the patient needs to be intubated, but is "clamped down" and was unable to be intubated without the use of rapid-sequence intubation. The flight crew performs a rapid trauma assessment, attaches the patient to transport monitors, and prepares to perform rapid-sequence intubation. The patient is successfully intubated and packaged for transport. He is transported to the aircraft via stretcher.

As the flight crew approaches the aircraft, they provide a quick debrief of the safety around the aircraft. They inform the ground crew that they will approach the aircraft from the front, once the pilot gives the go-ahead. They ensure that all loose objects are secure and that the ground crew has eye and hearing protection. Once the flight crew has ensured that it is safe to approach the aircraft, they enter into the landing zone from the front of the aircraft, maintaining eye contact with the pilot. Once the flight crew and ground crew reach the aircraft, your partner climbs into the aircraft to assist with loading. You remain on the outside of the aircraft to assist the ground crew. The patient is lifted off the stretcher via the backboard and loaded into the aircraft.

All of a sudden you hear a loud crash, and debris of metal is flying everywhere. Everyone is ducking and taking cover. The pilot immediately shuts down the aircraft. It becomes apparent that the ambulance stretcher rolled into the tail rotor, causing major damage to the tail rotor. Luckily, no one on the scene was injured. The pilot informs dispatch of the incident and states that they are "grounded" and requires the aircraft mechanic to report to the scene. The flight crew informs the ground crew that the patient needs to be transported by ground. The patient is unloaded from the aircraft, transferred to another ambulance, and transported by ground to a local Level I trauma center, with the flight crew on board.

1. What is the purpose of performing a walk-around of the aircraft?
2. What is the specific reason for responding "ready on right" and "ready on left"?
3. What is the purpose of high reconnaissance?
4. What went wrong?
5. Could they have flown the patient if there was minimal damage to the aircraft, especially since the patient's condition was critical?
6. Was it necessary for the flight crew to accompany the patient to the hospital with the ground crew?

Analysis

The tail rotor is one of the most dangerous areas in rotor operations. Contact with an operating tail rotor is usually serious or fatal. This scenario illustrates the importance of assigning a tail rotor guard and ensuring that all personnel and equipment are kept away from the tail rotor.

During a hot load, the aircraft remains running and the pilot maintains the aircraft. Hot loads may be necessary when a patient's condition is critical, but they impose several safety risks, including walking into the rotor, dipping rotor blades, and debris blowing into the rotor blades. A landing zone coordinator must be assigned and is responsible for ensuring that no one approaches the aircraft. He or she also

minimizes other hazards, for example by ensuring that hats are removed, no one carries anything over the head, and everyone is aware of the danger zones.

A cold load occurs when the aircraft is completely shut down during loading of the patient. This is the safest type of load.

Regardless of the type of load being performed, no one should approach the aircraft during start up or shut down. The aircraft should not be started until the landing zone is clear of all personnel.

Walk-arounds are performed to ensure that all doors, bolts, and hatches are secure, to prevent any doors from opening during flight, and to ensure that there are no loose bolts or hatches that could loosen in flight and damage the aircraft.

The statement "ready on right" or "ready on left" informs the pilot that the flight crew is ready for take-off and that they are looking out the side windows for other aircrafts or obstructions that may not be visible to the pilot. This is part of crew resource management, which holds the entire crew responsible for the safety of the crew and aircraft.

The purpose of the high reconnaissance is to allow the pilot and crew to view the scene and to identify potential hazards that the ground crew may not have identified. At this time, the pilot ensures that the landing zone is adequate.

This incident could have been prevented if the ground and flight crews had assigned someone to maintain the stretcher. Any incident that can cause possible damage to the aircraft requires the aircraft mechanic to inspect the aircraft and repair any damage that has occurred, including bird strikes, unusual noises, or vibration. Safety always comes first in air medical transport.

Once the flight crew accepted the patient and "took over care" they had to maintain that patient care. Flight medical crews are often considered a higher level of care than ground medical crews. Therefore, if they fail to accompany the transport, it would be considered abandonment.

Prep Kit

Ready for Review

- The CCTP needs to be familiar with both rotor-wing (helicopter) and fixed-wing (airplane) operations. Aircraft safety and operations are the primary concern.
- Critically ill or injured patients and those who would have a negative outcome with prolonged transport by ground units benefit most from air medical transport.
- Evaluate the benefits vs risks before transporting a patient via air. Consider the potential effects of altitude on the patient and the terrain, weather, and geographic location of the closest facility.
- VFRs are used when there is generally very good visibility and minimal cloud cover.
- IFRs apply when adverse weather conditions exist, visibility is poor, or the cloud cover is low.
- An authorized ATC facility may provide flight following to aircraft flying VFR, in which the controller notifies the aircraft about traffic in the area.
- If a pilot flying VFR in a helicopter decides not to flight follow with ATC, real-time radio contact with a communications center must occur every 15 minutes.
- During fixed-wing operations, when a flight plan has not been filed, the pilot in command must make contact with ATC or a communications center every 30 minutes.
- While transporting a patient in a ground unit, communication lapse must not exceed 45 minutes.
- There are two types of air medical transport: rotor wing (helicopter) and fixed wing (airplane). Rotor-

wing transport is used most commonly. Fixed-wing transport is typically used for flights of 200 nautical miles (300 miles) or greater, international transports, and transports that cannot occur by rotor wing because of weather conditions.

- Advantages of rotor-wing transport include vertical takeoff and landing (ability to access areas where other vehicles cannot) and ability to maneuver.
- Disadvantages of rotor-wing transport include weather limitations (must fly VFR), interior space limitations, weight limitations, and cost.
- Advantages of fixed-wing transport include that it is generally safer than rotor wing, ability to fly IFR, ability to fly at high speeds, ability to travel great distances, ability to carry multiple patients, and no weight limitations.
- Disadvantages of fixed-wing transport include high cost for operating the aircraft, performing maintenance work, and maintaining a hanger for housing the aircraft.
- One of the benefits of air medical transport is specialty. A flight crew's protocols may allow a higher level of care than even a critical care transport ground crew's protocols.
- Hazards of air medical transport relate primarily to rotor-wing hazards, such as moving tail rotors.
- The cost of air medical transport is generally high. There must be solid justification for helicopter use in order for insurance companies and/or Medicare to reimburse flight programs.
- Most air medical providers do not routinely transport patients in active cardiac arrest.
- Patients who will not benefit from air transport include those with terminal illnesses, do not resuscitate orders, and arrest without return of spontaneous circulation; and those whose conditions will overwhelm the capabilities of the crew.
- Relative contraindications to air medical transport include severe anemia, hemoglobinopathy, myocardial infarction within 10 days or complications in the 5 days before the flight, uncontrolled arrhythmia, pregnancies past 24 weeks' gestation, recent eye surgery affecting the globe, and non-acute hypovolemia.
- Helicopter EMS is one of the most dangerous forms of aviation with a very high accident rate compared with other sectors of aviation.
- Aircraft crashes are never caused by one single event, but rather by a series of factors. Human error is the number one cause, and weather is the main contributing factor.
- Crew resource management was developed to address communication issues that resulted in fatal crashes.
- All members of the crew should hold equal empowerment regarding whether to accept a mission. If any crew member does not feel comfortable with a mission, the mission should be aborted.
- Two FAA guidelines apply to air medical operations: Title 14 CFR Part 91 and Title 14 CFR Part 135. The guidelines for 14 CFR Part 135 operations are much stricter than those for 14 CFR Part 91 aircraft and apply to almost all air medical operators.
- The FAA requires that air medical transport programs designate someone for the position of operational control. This is the person who has the ultimate authority to initiate, conduct, and terminate the EMS mission, and has ultimate authority over all of the flight operations for the program.
- A weather decline is when an air medical provider refuses a mission because the weather does not meet established company or FAA guidelines for flight operations.
- CAMTS publishes safety standards to which CCTPs and pilots should adhere. They have also

established weather guidelines that exceed FAA requirements and have been accepted by many air medical providers.

- The most common crew configuration in the air medical environment is flight nurse/flight paramedic. Other configurations include nurse/respiratory therapist, nurse/nurse, and paramedic/paramedic. Some services may include a perfusionist on the flight team.
- A standby is when an HEMS provider is notified of a potential need for air medical transport. While on standby, the air medical crew determines whether to accept the mission, prepares for the call, and awaits confirmation or cancellation.
- Some programs have an auto launch protocol, in which the aircraft launches en route to the scene when placed on standby instead of waiting for the formal launch request.
- Landing a helicopter can be very dangerous if the landing zone is not properly established.
- Landing zone personnel must use proper hand signals and must wear proper eye and ear protection at all times. No personnel should ever operate near the tail rotor.
- When loading patients into the aircraft, no one should approach the aircraft unless directed by the flight crew.
- One member of the flight team should be assigned as tail rotor guard to ensure that no one approaches the tail rotor.
- All flight team members must have training in aircraft safety and survival. Training should also include aircraft familiarity, hard landing sequence/crash landing, equipment storage, personal protective equipment, and egress training.
- Critical care transport teams should hold daily safety briefings at the beginning of each scheduled shift, covering the daily plan, crew member duties, equipment and aircraft issues, weather expectations, and emergency situations.
- A hard landing is when the aircraft contacts the ground harder than normal, possibly resulting in injury or aircraft damage.
- The best time to prepare for a crash is before it happens. Crash safety procedures should be practiced until they become instinct.
- Preparation and education in survival techniques should be included in every critical care transport program.
- Air medical crew should receive crash survival training, including location and contents of the aircraft survival bag, construction of a temporary shelter, and survival priorities.
- Be familiar with how to improve your odds of survival. It is crucial to avoid panic and anticipate your fears. Know how to signal, stay with the downed craft, and find potable water in order to stay hydrated.
- Water ditching is the term for crashing into water. Helicopters usually capsize because they are top heavy.

Vital Vocabulary

Air Medical Physician Association (AMPA) An organization of physicians and air medical professionals that promotes safe patient transport.

auto launch A protocol that launches an aircraft at the time it is placed on standby rather than waiting for

the formal launch request. An auto launch is frequently activated when the distance to be covered for the response is greater than 30 nautical miles.

Commission on Accreditation of Air Medical Transport Services (CAMTS) An organization dedicated to improving the quality of patient care and safety of the transport environment for both rotor-wing and fixed-wing providers. CAMTS also provides voluntary accreditation of critical care transport agencies.

crew resource management A system that originated as the result of a NASA workshop in 1979 as a means to improve air safety. It incorporates equipment, procedures, and crew concerns to make the best decision during flight operations, focusing on interpersonal communication, leadership, and decision making.

daily safety briefing A meeting held at the beginning of each critical care transport shift, in which the daily plan, crew member duties, equipment and aircraft issues, weather expectations, and emergency situations are reviewed.

emergency locator transmitter (ELT) A device that activates upon the crash of an aircraft and broadcasts a signal that rescuers can use to locate the downed aircraft.

Federal Aviation Administration (FAA) A US governmental agency within the Department of Transportation that regulates and oversees civil aviation within the United States; established and enforces the Title 14 CFR that govern aircraft operation within the United States.

flight following The practice of maintaining constant contact with other local traffic and air traffic controllers.

hard landing An excessively firm contact with the ground, potentially resulting in injury or aircraft damage.

helicopter emergency medical service (HEMS) Use of a rotor-wing aircraft to deliver air medical service, for which the goals are to rapidly transport CCTPs to a patient, stabilize the patient's condition, and rapidly transport that patient to a tertiary care center.

helicopter shopping The practice of making sequential calls to numerous air medical providers in an attempt to find a service that will accept a mission request that has been declined by other services based on safety factors such as poor weather, limited landing zone availability, exceptional distances, or other factors.

hot loading Loading a patient into a helicopter while the helicopter is running.

instrument flight rules (IFR) A mode of flight used when adverse weather conditions exist, visibility is poor, or cloud cover is low. The pilot may not be able to see outside the aircraft, must rely on instruments, and must be in constant contact with an air traffic controller who assists in maintaining proper separation from other air traffic.

instrument meteorological conditions (IMC) Weather conditions (eg, cloudiness or low visibility) in which a pilot must fly under instrument flight rules, depending on instruments to guide the aircraft.

International Civil Aviation Organization (ICAO) An agency of the United Nations that defines standards for international air navigation and, as part of its role in developing safe practices, requires crew resource management.

National Transportation Safety Board (NTSB) An independent federal agency that promotes transportation safety, including aviation, railroad, highway, maritime, pipeline, and hazardous materials safety; investigates transportation crashes to identify the cause and make safety recommendations.

operational control The person who has the ultimate authority to initiate, conduct, and terminate the EMS mission; in an air medical program, usually the chief pilot or operational manager.

overtriage Considering a patient to be in more serious condition than he or she actually is.

rule of 3s A means of prioritizing the basic physiologic needs for survival: The average person can survive 3 minutes without oxygen, 3 hours without shelter, 3 days without water, and 3 weeks without food.

somatogravic illusion An error in perception that occurs with acceleration, as the otolith organs are displaced rearward, similar to when a person is looking up. This perception of a nose-up altitude may cause the pilot to push the nose of the aircraft down inappropriately at night or in unlit terrain.

spatial disorientation An error in perception that may result from a person's inability to determine his or her position, altitude, and motion in relation to the surface of the earth or to a significant fixed object during flight.

standby The situation in which an HEMS provider is notified of a potentially life-threatening emergency call and makes preparations to respond to the call. During standby, the flight crew checks weather data, obtains landing coordinates, and conducts a preflight inspection of the aircraft.

sterile cockpit The time when unnecessary communication that could distract the pilot is banned in the cockpit—usually during takeoffs, landings, and any other critical phase of flight at the discretion of the pilot in command.

Title 14 Code of Federal Regulations (CFR) Part 91 Established by the FAA, this guideline governs the operation of all aircraft within the United States, including the waters within 3 nautical miles of the US coast.

Title 14 Code of Federal Regulations (CFR) Part 135 Established by the FAA, this guideline governs the operations of all commuter or on-demand commercial operations.

undertriage Considering a patient to be in less serious condition than he or she actually is.

visual flight rules (VFR) A mode of flight used when weather conditions are good, meaning there is generally very good visibility and minimal cloud cover; the pilot is responsible for maintaining separation from other aircraft.

water ditching Crashing into water.

weather decline A situation in which an air medical provider is requested, but the pilot and the flight team determine that the weather does not meet established FAA guidelines and therefore decline to make the flight.

Bibliography

American Psychological Association. Making air travel safer through crew resource management. Available at: http://www.psychologymatters.org/crm.html. Accessed June 12, 2008.

Bachman J. State grounds WakeMed's request for air ambulances. Available at: http://www.wral.com/news/local/story/115183. Accessed February 5, 2009.

Baker SP, Grabowski JG, Dodd RS, et al. EMS helicopter crashes: what influences fatal outcome? *Ann Emerg Med*. 2006;47:351–356.

Baxt WG, Moody P, Cleveland HC, et al. Hospital-based rotorcraft aeromedical emergency care services and trauma mortality: a multicenter study. *Ann Emerg Med*. 1985;14:859–864.

Baxt WG, Moody P. The impact of rotorcraft aeromedical emergency care service on trauma mortality. *JAMA*. 1983;249:3047–3051.

Benson NH, Hunt RC, Tolson J, Stone CK, Sousa JA, Nimmo NJ. Improved flight following through continuous quality improvement. *Air Med J*. 1994;13:163–165.

Blumen IJ, UCAN Safety Committee. *A Safety Review and Risk Assessment in Air Medical Transport: Supplement to the Air Medical Physician Handbook (suppl)*. Salt Lake City, UT: Air Medical Physicians Association; November 2002.

Boyd DR, Cowley RA. Comprehensive regional trauma/emergency medical services (EMS) delivery systems: the United States experience. *World J Surg*. 1983;7:149–157.

Centers for Disease Control and Prevention, National Center for Health Statistics. Visits to the emergency department increase nationwide. Available at: http://www.cdc.gov/nchs/pressroom/02news/emergency.htm. Accessed June 12, 2008.

Collett H. Air medical helicopter transport. *Hosp Aviat*. 1998;7(7):5–7.

Cowley RA. Trauma center: a new concept for the delivery of critical care. *J Med Soc NJ*. 1977;74:979–987.

Dodd RS. The cost-effectiveness of air medical helicopter crash survival enhancements: an evaluation of the costs, benefits and effectiveness of injury prevention interventions. *Air Med J*. 1994;13:281–293.

Federal Aviation Administration. FAR part 135 guidelines. Available at: http://www.access.gpo.gov/nara/cfr/waisidx_05/14cfr135_05.html. Accessed June 12, 2008.

Federal Aviation Administration. FAR part 135 guidelines. Available at: http://www.access.gpo.gov/nara/cfr/waisidx_05/14cfr91_05.html. Accessed June 12, 2008.

Kilroy C. Special report on United Airlines 232. Available at: http://www.airdisaster.com/special/special-ua232.shtml. Accessed June 12, 2008.

Lerner EB, Moscati RM. The golden hour: scientific fact or medical "urban legend"? *Acad Emerg Med*. 2001;8(7):758–760.

McNicholl BP. The golden hour and prehospital trauma care. *Injury*. 1994;25(4):251–254.

Military Assistance to Safety and Traffic (MAST). *Report of Test Program by the Interagency Study Group*. Washington, DC: DHEW publication HSM-72-7000.

National Oceanic and Atmospheric Administration. Emergency locator beacons. Available at: http://www.sarsat.noaa.gov/emerbcns.html. Accessed June 12, 2008.

National Safety Council. *Injury Facts: 2004 Edition*. Itasca, IL: National Safety Council; 2004.

Kiggans S. Unsafe acts in the hornet. Available at: http://www.safetycenter.navy.mil/media/approach/issues/NovDec06/Unsafe_Acts_in_the_Hornet.htm. Accessed June 12, 2008.

Proctor HJ, Acai SA Jr. Assets and liabilities of helicopter evacuation in support of emergency medical services. *NC Med J*. 1976;37:25–28.

Royal Aeronautical Society. Crew resource management: a paper by the CRM Standing Group of the Royal Aeronautical Society. Available at: http://83.146.40.74/crm/reports/crm-now.htm. Accessed June 12, 2008.

Shatney CH, Homan SJ, Sherck JP, Ho CC. The utility of helicopter transport of trauma patients from the injury scene in an urban trauma system. *J Trauma*. 2002;53(5):817–822.

Springer B. The IFR bullet: can it kill our accident rate? *Air Med J*. 2005;24:29–31.

Wright RM Jr. Air Medical Service: an industry under scrutiny. *Rotor*. Winter 2004–2005:6–8.


AIR MEDICAL CREW

Flight Physiology

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Objectives

1. Describe the composition of the atmosphere (p 70).
2. List the three physiologic zones in the atmosphere (p 71).
3. Discuss the seven primary gas laws affecting flight physiology (p 72–75).
4. Define the four types of hypoxia (p 76–77).
5. Describe the four stages of hypoxia as they relate to altitude (p 78).
6. Recognize the signs and symptoms of altitude-induced hypoxia and the appropriate treatment (p 80).
7. Summarize the advantages and disadvantages of pressurized and nonpressurized aircraft (p 80–81).
8. Recognize the physical and physiologic effects of rapid cabin decompression (p 81).
9. Define the effective performance time and the time of useful consciousness (p 76).
10. Describe the primary forces that act on an aircraft (p 81).
11. List the primary stressors of flight and their physiologic impacts (p 82–89).
12. Define the factors affecting tolerance of the stressors of flight (p 89–90).
13. Identify different dysbarisms and trapped gas disorders (p 90–91).
14. Describe assorted exacerbations of medical conditions due to altitude (p 75, 90).

Introduction

CCTPs must have a thorough understanding of flight physiology and the implications for their patients and for themselves. Critical care transport frequently involves air and ground transportation. Critical care providers who are solely ground based must still have a comprehensive knowledge of flight physiology because frequently they will prepare patients for air transport and will receive patients who have been flown in. Knowledge of flight physiology involves not only recognition of barometric maladies, but also, more important, the prevention of such problems.

The air medical environment is complex and extremely dynamic. Critical care providers frequently encounter comorbidities or multitrauma patients who are already in a severely compromised condition. Many conditions will be exacerbated by changes in the barometric pressure. Likewise, the forces experienced during flight can have significant impact on disease pathophysiology. Therefore, CCTPs must have a strong understanding of the effects of altitude on the body.

This chapter discusses the various gas laws that are pertinent to flight medicine and several common problems that can occur as the result of changes in barometric pressure. Related to barometric pressure, this chapter also discusses the advantages and disadvantages of pressurized and nonpressurized aircraft and the impact of decompression at altitude. CCTPs must have a solid understanding of the potential catastrophic consequences that can be experienced with rapid cabin decompression.

CCTPs must also be aware of the primary stressors of flight and the factors that can affect tolerance toward the stressors. Aircrew members need to also be familiar with different illusions of flight and spatial disorientation that can have disastrous impacts on flight operations. Some spatial disorientation can also have negative effects on patients and can precipitate serious medical conditions. A thorough

knowledge of flight physiology is essential to keep the flight crew airworthy and to prevent complications in the transport of critically ill and injured patients.

The Atmosphere

In his book *Basic Flight Physiology,* **flight surgeon** Richard Reinhart defines three distinct parts to flight: the aircraft, the flight crew, and the environment (the **atmosphere**). All three maintain a relationship of balance; however, the atmosphere directly affects the aircraft and the flight crew. The atmosphere is likened to an ocean of gases with defined layers of stratification.

The atmosphere extends from the surface of the earth to 348 miles (560 km), which is the beginning of space **Figure 4-1**. This chapter primarily discusses the components of the lower atmosphere in which all aviation occurs with the exception of space exploration. The atmosphere varies with time of day, season of the year, and latitude. Because of these variables, only averages are discussed in this chapter.

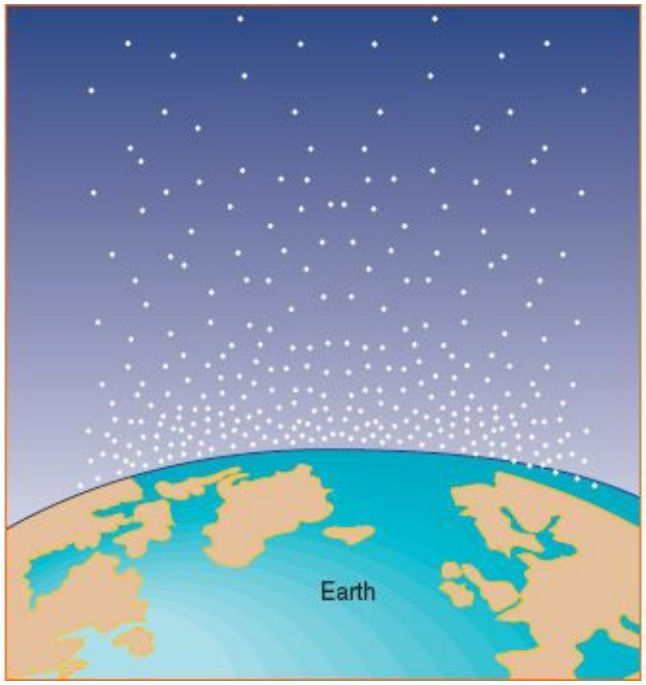


Figure 4-1 The density of the atmosphere decreases the farther you are from sea level.

Atmospheric Composition

The composition of the atmosphere is constant and is defined as a percentage of gases instead of absolute amounts. Although the percentage of gases is constant, the density of the gases varies with the altitude. Three gases constitute almost 99% of the atmosphere, which remains constant from the surface to altitudes of 250,000′. Oxygen constitutes 21% of the atmosphere. A by-product of photosynthesis, oxygen is the most critical gas needed to sustain life. Nitrogen is the most abundant gas in the atmosphere, accounting for 78% of the total volume. Nitrogen is responsible for the majority of the composition of the atmosphere and is an inert gas that is odorless, colorless, and tasteless. This gas is not readily used by humans; however, it is a critical element for life. Nitrogen is present in the human body in abundant quantities and can be responsible for evolved gas disorders at altitude or after rapid ascent while scuba diving. Argon constitutes approximately 0.93% of the atmosphere. The other trace gases that are found include carbon dioxide, neon, helium, methane, krypton, and hydrogen. No matter what the altitude, the percentage of oxygen will always be about 21%. During ascent in altitude, the molecules of oxygen spread out and become less numerous in each breath. This is why it can be difficult for human beings to breathe at high altitudes, such as at the top of Mount Everest.

Layers of the Atmosphere

There are several distinct layers of stratification of the atmosphere. Gravity holds the layers of the atmosphere in place. Five distinct layers have been identified using thermal characteristics (temperature changes), chemical composition, movement, and density. The density of the atmosphere decreases with height because the weight of the molecules actually compresses the gas near the earth's surface.

The first layer is called the **troposphere**. The troposphere extends from sea level to about 26,405′ over the poles and nearly 52,810′ above the equator. Virtually all weather occurs in the troposphere because of the presence of water vapor and strong vertical currents. In this layer, clouds form, rain falls, wind blows, and humidity varies depending on the climate. Winds in the troposphere become stronger westerly with increasing west to east winds at altitudes higher than 35,000′. The strong jet stream is located above 35,000′, and maximum winds in the jet stream average 200 mph at about 30° latitude north or south.

The troposphere is the densest portion of the atmosphere. Temperature varies from 62.6°F (17°C) to –68.8°F (–56°C) and decreases proportionately with increases in altitude. The **tropopause** is the layer between the troposphere and the stratosphere, which is the next layer. The tropopause ranges in height from 30,000′ at the poles to more than 60,000′ at the equator. More solar energy is received by the earth near the equator, causing the air to heat and expand. This expansion of rising air increases the height of the tropopause. Conversely, the cool air at the poles results in contraction and shrinkage of the air, resulting in a lesser height of the tropopause. The tropopause and the troposphere are known as the lower atmosphere.

The layers above the tropopause include the stratosphere, stratopause, mesosphere, thermosphere, and exosphere, in that order. Critical care transports would not usually occur at these altitudes. **Figure 4-2** shows the layers of the atmosphere and how they compare with the zones of the atmosphere, to be discussed next.

Physiological Zones of the Atmosphere

The atmosphere is divided into three distinct zones that directly correlate to a human's response to hypoxia. These zones are the **physiological zone**, the **physiologically deficient zone**, and the **space equivalent zone**.

Physiologic Zone

The physiologic zone is the area of the atmosphere that contains the oxygen and barometric pressure needed for a normal, healthy person to live. This zone extends from sea level to 10,000′. In this zone, the barometric pressure falls from 760 mm Hg at sea level to 523 mm Hg at 10,000′. The pressure at 10,000′ is still enough to maintain an adequate **partial pressure of oxygen (arterial) (Pa_{O_2})** without the use of supplemental oxygen, pressurization, or protective equipment. However, it is important for CCTPs to realize that at 10,000′, many healthy people will begin to experience very mild effects of hypoxia, such as a mild headache, and almost all patients with comorbidities will be hypoxic at this altitude. Part 135.89 of the Federal Aviation Regulations (FAR) states that commercial pilots must use supplemental oxygen when flying above 10,000′.

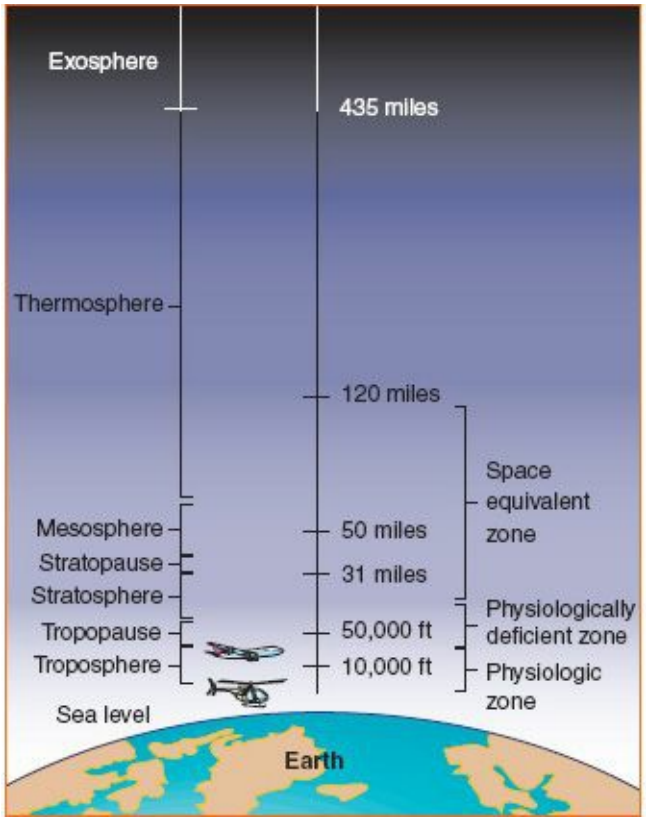


Figure 4-2 The layers and zones of the atmosphere.

Rapid changes in altitude at this level can produce trapped gas conditions such as ear or sinus problems; however, these problems are considered relatively minor compared with the physical impairments that occur at higher altitudes. Altitudes above 5,000′ can also affect night vision unless supplemental oxygen is used.

Physiologically Deficient Zone

The area from 10,000′ to 50,000′ is called the physiologically deficient zone. Above 10,000′, barometric pressure begins to decrease to levels that will result in hypoxic hypoxia, which is discussed later in this chapter. Barometric pressure at this level ranges from 523 mm Hg at 10,000′ to 87 mm Hg at 50,000′. At these altitudes, the effects of trapped gases become more pronounced, and protective equipment, supplemental oxygen, and pressurized aircraft are necessary.

Space Equivalent Zone

The space equivalent zone extends from 50,000′ to 120 miles. At this point in the atmosphere, 100% supplemental oxygen is no longer adequate because of the inadequate barometric pressure. Pressure suits and sealed cabins are also required. Two additional hazards can be present. Exposure to atmospheric conditions could result in the boiling of body fluids as the fluids turn to a vapor. Personnel may also be exposed to increased levels of radiation from the sun in this zone. Currently, no commercial aircraft used for air medical transport operate in this zone.

Barometric Pressure

Barometric pressure, also called atmospheric pressure, is a direct result of the weight of air. Barometric pressure varies with location and time because the amount and weight of air above the earth vary with time and location. Barometric pressure is also related to the density of the air, which is related to air temperature and height above the earth's surface. Thus, **barometric pressure** is the weight per unit area of all of the molecules of the gas above the point at which the measurement was taken, with temperature

and humidity as variables. Barometric pressure is also related to weather and is one of the most important factors that determine weather.

Barometric pressure is reported in different measurements, most commonly inches of mercury in the United States, but millibars are used in most countries that use the metric system, such as in most of Europe. There are different scientific definitions of the atmosphere. The two most prevalent definitions are the US Standard Atmosphere and the International Standard Atmosphere. The US Standard Atmosphere has been recognized for the longest time; however, the International Standard is now more widely recognized across the globe.

Gas Laws

It is critically important that CCTPs have a thorough knowledge of the assorted gas laws as they relate to both flight and hyperbaric medicine. The various gas laws have an important role not only in patient care, but also in the overall safety of the flight crew. There are seven gas laws that are integral to flight physiology, some of which are interdependent.

Boyle's Law

Robert Boyle studied the relationship between the volume of a dry gas and its pressure. To study the relationship of volume and pressure, he fixed the amount of gas and its temperature. Boyle found that when he increased the volume of the gas, the pressure decreased. Likewise, a decrease in volume resulted in an increase in pressure. **Boyle's law** can be remembered by the saying, "Boil Very Prudently," where Boyle = Volume (Very) × Pressure (Prudently). A practical example is that as altitude increases, the atmospheric pressure decreases. This means there is less outside pressure on gas molecules holding them close together. As a result, the gas molecules expand, or drift apart. For example, a bag of unopened chips on a commercial air flight expands as the altitude of the aircraft increases. During descent, the bag of chips shrinks back to its original size.

Boyle's law has numerous implications in aviation medicine. Any gas trapped in the chest, such as in a pneumothorax, will expand approximately 35% when going from sea level to 8,000′ **Figure 4-3**. At 18,000′, a given volume of gas will expand to twice its size. The key to minimizing the effects of altitude is to maintain the lowest possible cabin pressure during flight. As a general rule-of-thumb, altitude will not become a significant factor until 3,000′ above ground level. Most people, except people with significant respiratory disorders, tolerate altitudes of 5,000′ with minimal disturbances. However, below 10,000′—even as low as 3,500′—there can be significant effects on unhealthy people. Partly because of these facts, most helicopter transports are conducted at or below 3,000′ above ground level.

Several disease processes can have a drastic effect with increasing altitude. An injury such as a pneumothorax can quickly become a tension pneumothorax as altitude increases. Patients with an open skull fracture are at risk for pneumocephalus because air becomes trapped inside the skull and expands with altitude, causing a significant increase in intracranial pressure. Patients with **pneumoperitoneum** (the collection of air within the peritoneum) or **pneumomediastinum** (the collection of air within the mediastinum) are also susceptible to the changes in altitude. Although these disease processes might not cause a high-tension event within the body, they cause significant discomfort.

Boyle's law also applies to the expansion of any trapped gas within the body in such places as the middle ear, sinuses, stomach, and intestines. There are also many pieces of medical equipment that are exquisitely sensitive to an increase or decrease in barometric pressure. The cuffed balloon on endotracheal tubes can double in size from 5,000′ to 10,000′, causing the balloon to rupture or cause tissue necrosis if allowed to remain hyperinflated for a length of time. Therefore, cuffed balloons should

be filled with saline before flights planned for above 6,000′, or cuff pressure should be monitored and adjusted.

IV fluids also are sensitive to changes in barometric pressure. The rate of flow of fluids in a glass bottle, such as nitroglycerin, will increase because of the changes in altitude and the inability of the bottle to expand or contract. The rate of fluids that are allowed to free flow can increase drastically at altitude as pressure causes expansion inside the bag. Because of the short height of aircraft cabins, most IV fluids are unable to flow readily and can frequently back up in the IV tubing. Therefore, the use of pressure infusers to administer fluids is recommended. The use of a "dial-a-flow" to administer IV fluids is not considered reliable, and IV infusion pumps should be used instead.

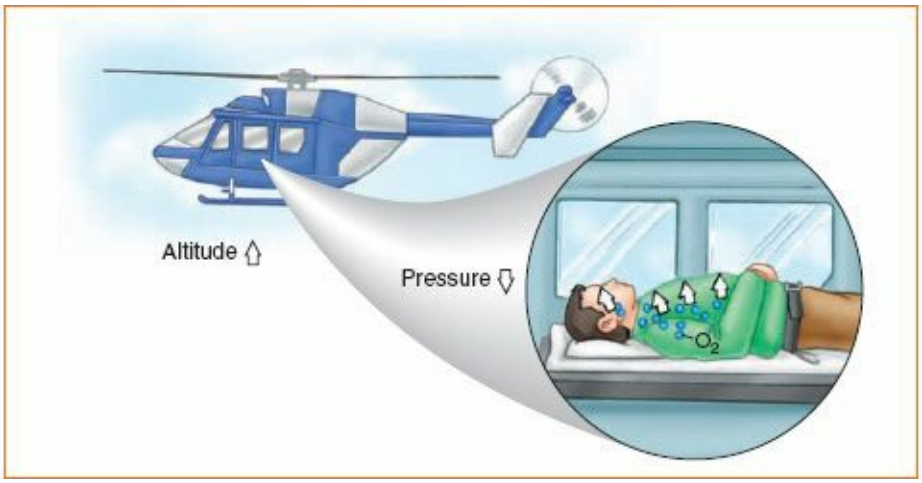


Figure 4-3 Boyle's law. As altitude increases, atmospheric pressure decreases, and gases inside the body expand.

Although most services do not use pneumatic antishock garments (PASGs) and their use is no longer routinely recommended, providers must still be aware of the dangers of PASGs at altitude. If a patient is flown wearing PASGs, CCTPs must give close attention to the PASGs, and as soon as the Velcro begins to "pop off," the relief valve must be opened or the PASGs will come apart. The same concept applies to air splints at altitude. Some models of air splints have pressure relief valves that will activate, but other models do not and must be adjusted manually.

All patients who have a nasogastric or orogastric tube inserted should be transported with the tube open or frequently vented. Patients with colostomy bags should frequently have the built-up gas "burped" to prevent overpressurization and failure of the colostomy bag.

Charles' Law

Discovered in 1787 by Jacques Charles, **Charles' law** states that the volume of a gas is directly proportional to the temperature, with the pressure remaining constant. The practical application is that as the air heats up, the volume increases, allowing the molecules to spread out, making the air less dense. It is easier for a helicopter to fly in cold weather because the gas molecules are more compressed and allow more lift as the rotor blades spin. In hot weather, gas molecules are farther apart and provide less lift. There are fewer molecules for the blades to "push off of." For this reason, the amount of weight a helicopter or airplane can carry is less on a hot, humid day than on a cold, dry day.

Charles' law can be remembered by the phrase "Charles' cold" or "Charles Celsius." Charles' law is significant in flight medicine because aircraft cabins get very, very cold at altitude without supplemental heat **Figure 4-4**. This cold temperature puts patients and crew at significant risk for hypothermia. CCTPs need to take measures to keep themselves and their patients warm.

Dalton's Law

In 1800, John Dalton postulated **Dalton's law**, which states that the total pressure of a gas mixture is the sum of the individual pressures. Simply put, all of the parts equal the whole. He discovered that in a gas mixture, gas molecules are unaffected by each others' motion because there is so much space between the molecules. This law is also referred to as the law of partial pressure. The partial pressure is the pressure of a single gas in the mixture.

Dalton's law illustrates that increasing altitude results in a proportional decrease of partial pressures of gases found in the atmosphere. Although the percentage concentration of gases remains stable with increasing altitude, partial pressure decreases in direct proportion to the total barometric pressure.

Even though the percentage of oxygen is a constant, the partial pressure will decrease proportionately as atmospheric pressure decreases, or vice versa. Pressure is required to facilitate the passing of oxygen from blood to the cells. A decrease in pressure can lead to hypoxia in the body. Although the proportion of oxygen remains about 21% in the atmosphere, there are fewer molecules that the body can use. Dalton's law is extremely important in critical care medicine because when supplemental oxygen is given, Dalton's law can be used to calculate the expected partial pressure of oxygen (P_{O_2}) that should be obtained when arterial blood gas values are checked. Dalton's law can be remembered by the phrase "Dalton's gang," because oxygen molecules that were "ganged up" at lower altitudes spread apart at higher altitudes, making less oxygen available for breathing **Figure 4-5**.

Fick's Law

Fick's law was first established in 1855 by Adolph Fick. This law states that the diffusion rate of a gas is proportional to the difference in partial pressure, proportional to the area of the membrane, and inversely proportional to the thickness of the membrane. In practical terms, the rate of diffusion is affected by atmospheric pressures, the surface area of the membrane, and the thickness of the membrane. This is the primary gas law for the diffusion of oxygen across the alveolar membrane. For example, an elderly patient with chronic obstructive pulmonary disease (COPD) who also has pneumonia will have decreased gas exchange at altitude because of decreased atmospheric pressure, decreased surface area of alveoli, and increased membrane thickness, from the long-standing COPD and the exudate from the pneumonia **Figure 4-6**.

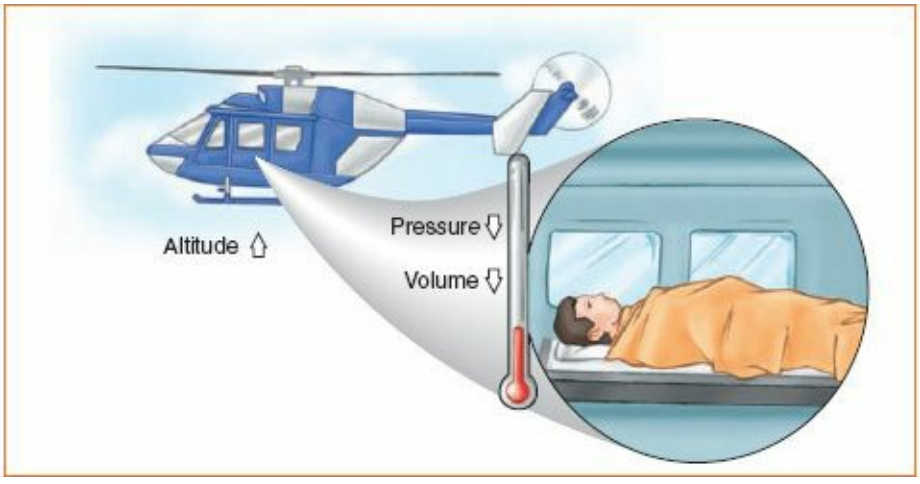


Figure 4-4 Charles' law. The volume of a gas is directly proportional to the temperature, with the pressure remaining constant.

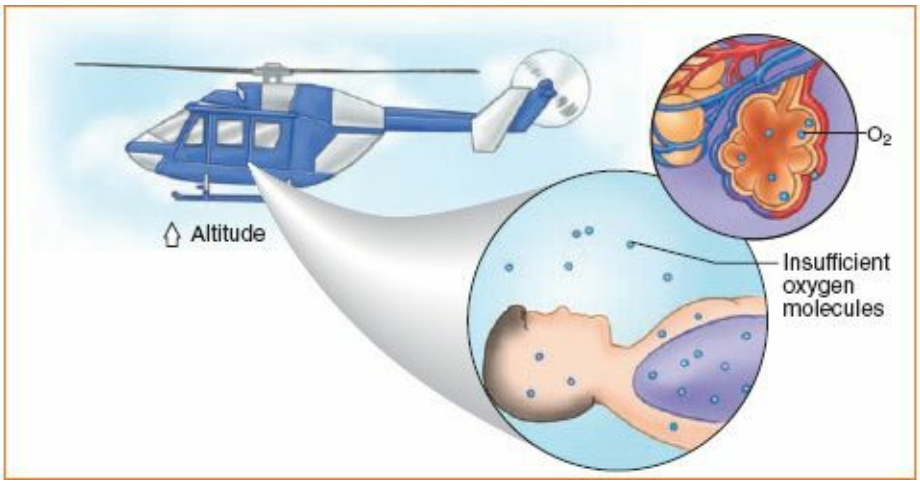


Figure 4-5 Dalton's law. The total pressure of a gas mixture is the sum of the individual pressures. Insufficient oxygen molecules can lead to hypoxia.

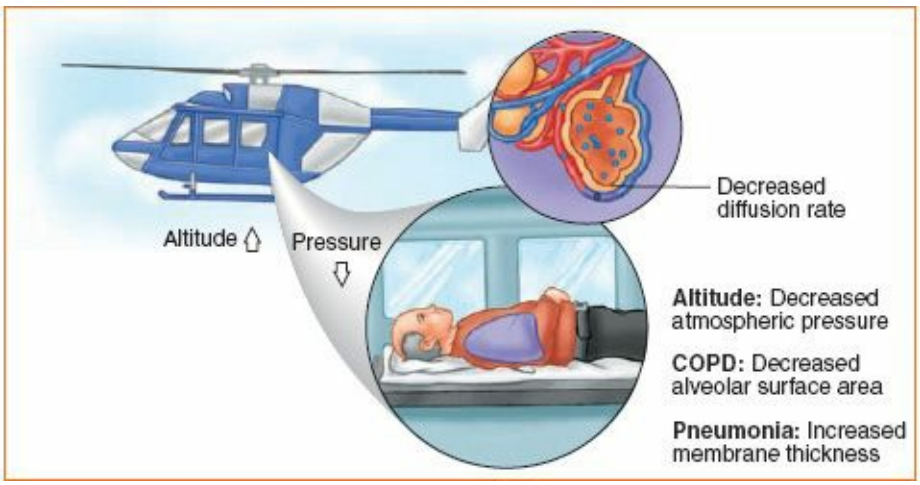


Figure 4-6 Fick's law. The diffusion rate of a gas is affected by atmospheric pressures, the surface area of the membrane, and the thickness of the membrane. A patient with COPD and pneumonia will have decreased gas exchange.

Henry's Law

In 1800, J.W. Henry postulated **Henry's law**, which states that the amount of a gas in a solution varies directly with the partial pressure of a gas over the solution. In other words, as the pressure of a gas over a liquid decreases, the amount of gas dissolved in the liquid will also decrease. As more pressure is applied over the liquid, more gas can be dissolved in the liquid. In practical terms, this law states that molecules of a gas can be dissolved in a liquid and remain in the liquid as long as the liquid is in a pressurized, closed container.

All carbonated beverages are an example of Henry's law. Carbon dioxide is dissolved in a liquid to produce carbonation. The beverage will remain carbonated as long as the bottle or can remains sealed. Once the beverage is opened, the pressure above the liquid falls, and carbon dioxide will begin to leave the solution, producing bubbles in the beverage.

The effect of Henry's law is evidenced in the body by decompression sickness, which is discussed later in this chapter. In this case, inert gases in the body tissue (primarily nitrogen) are maintained in equilibrium with the partial pressure of the same gases in the atmosphere. As barometric pressure decreases, the partial pressure of nitrogen in the atmosphere decreases as well. This decrease in barometric pressure leaves the tissues saturated with nitrogen at a higher pressure than the ambient

barometric pressure. As the body attempts to establish a new equilibrium, nitrogen comes out of solution in the form of gas bubbles and travels in the venous blood system to the lungs **Figure 4-7**.

Universal Gas Law

The **universal gas law** is also known as the ideal gas law because it states how a hypothetical gas should act if there are no variables affecting it. It states that a change in density is directly related to a change in temperature and pressure. Many gases have properties similar to the universal gas law at ambient temperature and pressure.

Gay-Lussac's Law

In 1809, French chemist Joseph Louis Gay-Lussac found that there is a correlation between the pressure and the temperature when volume is constant. **Gay-Lussac's law** can be expressed as a ratio; for example, if pressure increases, temperature increases, and vice versa **Figure 4-8**. Think of a room full of people (molecules); as the room gets smaller (pressure increases), the people will bump into each other, creating friction that will generate heat. As the room gets larger (pressure decreases), the people will not bump into each other and no heat is generated; thus, the room is cooler.

Graham's Law

Graham's law was formulated by Scottish physical chemist Thomas Graham. His law, also known as Graham's law of effusion, states that the rate at which a gas moves through a small hole, avoiding interaction with other particles along the way, is related inversely to the square root of the mass of one mole of its molecules. Thus, if the molecular weight of one gas is four times that of another, it would diffuse through a porous plug or escape through a small pinhole in a vessel at a rate half that of the smaller molecule.

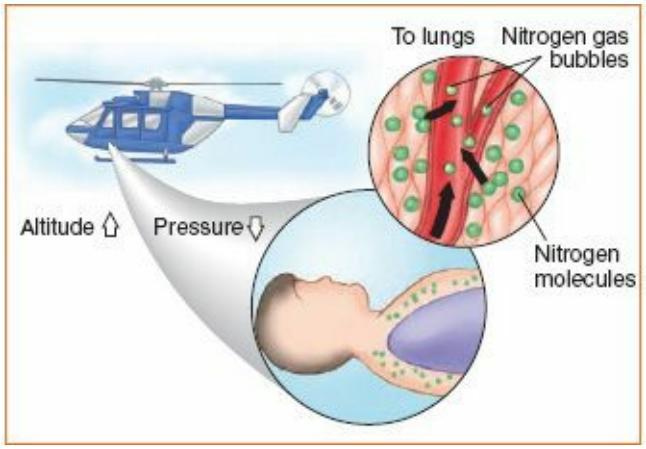


Figure 4-7 Henry's law. As the pressure of gas over a liquid decreases, the amount of gas dissolved in the liquid will also decrease. In decompression sickness, nitrogen saturates the tissues, then forms gas bubbles that travel to the lungs.

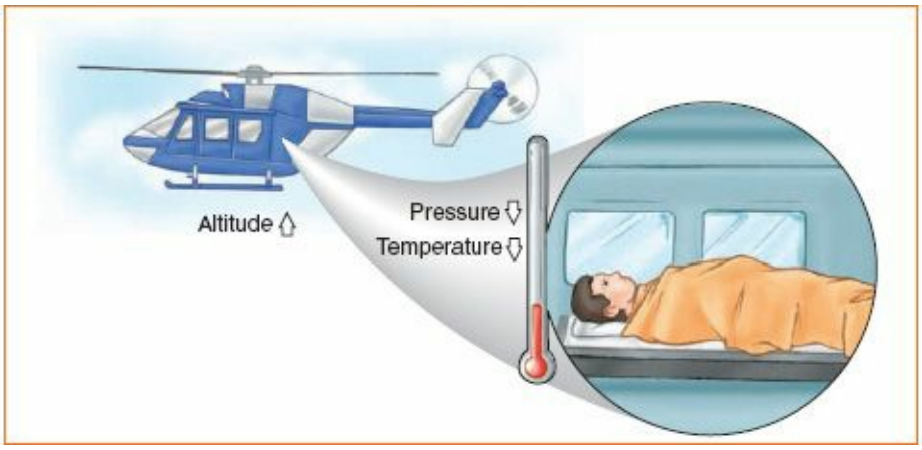


Figure 4-8 Gay-Lussac's law. As pressure decreases, temperature decreases. Remember to keep your patients warm.

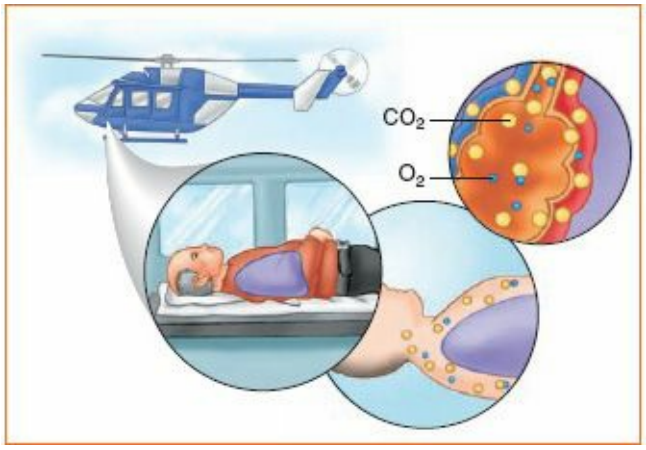


Figure 4-9 Graham's law. The rate at which a gas moves through a small hole is related inversely to the square root of the mass of one mole of its molecules. Carbon dioxide diffuses more readily into the bloodstream than does oxygen.

A practical example of this law is the ongoing process of the diffusion of oxygen and carbon dioxide in the blood and the transfer of oxygen from blood into the cells. Carbon dioxide molecules are much more massive than oxygen molecules, and carbon dioxide also has 22 times the solubility of oxygen; thus, the diffusion rate is much quicker than that of oxygen **Figure 4-9**.

Formulas Based on the Seven Gas Laws

The following formula, based on Henry's law, is used to determine how much additional oxygen will be needed to compensate for the altitude and barometric pressure changes:

$$\frac{\text{Initial } F_{IO_2} \times \text{Initial Barometric Pressure}}{\text{Barometric Pressure at Cruising Altitude}} = \text{Adjusted } F_{IO_2}$$

(F_{IO_2} is the fraction of inspired oxygen)

If the barometric pressure is 760 mm Hg at sea level and 600 mm Hg at 6,000′ above sea level and the patient's initial F_{IO_2} is 70%, with this formula, the adjusted F_{IO_2} would be:

$$\frac{70\% \text{ Initial } F_{IO_2} \times 760 \text{ mm Hg}}{600 \text{ mm Hg}} = 88.7\% \text{ Adjusted } F_{IO_2}$$

To ensure that the patient continues to receive 70% FIO_2, the ventilator would need to be adjusted to 88% FIO_2.

Types of Hypoxia

Hypoxia, a main hazard in aviation, can have catastrophic results. This physiologic effect can occur in otherwise healthy people at altitudes less than 10,000′, but can greatly affect patients with impaired pulmonary function at much lower altitudes.

With all of the advances in aircraft technology, the US military still reports 8 to 10 incidents of hypoxia during flight every year. Most of these incidents are attributed to a failure in cabin pressure with a concurrent failure in the oxygen delivery system. In most of these incidents, the pilots were able to recognize the situation and take immediate corrective action to avoid catastrophe.

A common misconception in aviation is the belief that one can recognize the early signs of hypoxia and take immediate corrective actions. This misconception is deadly and dangerous. One of the earliest effects of hypoxia is impaired judgment. This impaired judgment can limit the aviator's ability to recognize hypoxia and, consequently, his or her ability to take immediate corrective actions. Numerous aircraft crashes have documented aircrews responding to hypoxic events with inappropriate and dangerous actions. Also, early hypoxia mimics fatigue and hypoglycemia, making it difficult to recognize. Fatigue and hunger contribute to hypoxia as well.

TABLE 4-1 Relationship Betweeen Effective Performance Time and Altitude

Altitude	Effective Performance Time (Standard Ascent Rate)	Effective Performance Time (After Sldden Decompression)
18,000′	20-30 min	10-15 min
22,000′	8-10 min	5 min
25,000′	4-6 min	1.5-3.5 min
35,000′	30-60 s	15-30 s
43,000′	9-12 s	5 s
> 50,000′	9-12 s	5 s

Data source: Federal Aviation Administration.

Effective Performance Time and Time of Useful Consciousness

There is a limit to how long a person can function with an inadequate level of oxygen. This limited time is called **elleclive perlormance lime** Table 4-1, and the **time of useful consciousness** is the period between a person's sudden deprivation of oxygen at a given altitude and the onset of physical or mental impairment to the point at which deliberate function is lost.

It is important to realize that the times in Table 4-1 were obtained by using healthy military volunteers in a standard altitude chamber. The effective performance time and time of useful consciousness vary with each person and are dependent on individual tolerances, the method of hypoxia induction, and the environment before hypoxia. Any exercise will reduce these times considerably. For example, on exposure to hypoxia at 25,000′, the average person has a time of useful consciousness of 3 to

5 minutes. The same person after performing 10 deep knee bends will have a time of useful consciousness in the range of 1 to 1.5 minutes. Also, an aircrew member who was breathing 100% oxygen before the onset of hypoxia will have a longer period of compensation than an aircrew member who was breathing ambient air. Rapid cabin depressurization also dramatically affects the effective performance time and the time of useful consciousness. When aircraft cabin decompression occurs above 33,000′, there is an immediate reversal in oxygen flow in the alveoli. A higher Pa_{O_2} in the pulmonary capillaries occurs, causing the oxygen reserves in the blood to be depleted, reducing the effective performance time by up to half.

The time of useful consciousness varies from 5 minutes to 1 minute; however, if the crew is subjected to rapid depressurization, this time can be only a matter of seconds. A **rapid decompression** can reduce the time of useful consciousness by up to 50% because of the forced exhalation of the lungs during decompression and during an extremely rapid ascent.

Another common misconception regarding hypoxia is the belief that those living at higher altitudes do not require supplemental oxygen in order to reach more extreme heights. Although there is some truth to this notion, it does not afford the protection that most assume. Living at higher altitudes causes the body to produce red blood cells at a faster rate, facilitating oxygen transport. A slight physiologic advantage exists; however, an aircrew member who, for example, lives at 5,000′ may be able to fly 1,000′ to 2,000′ above the 10,000′ recommended ceiling without ill effects, but tolerance is not extended an additional 5,000′.

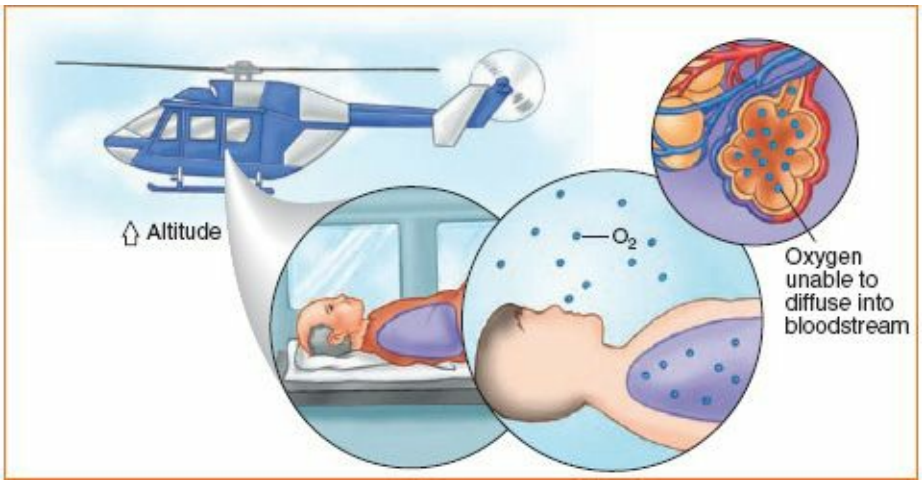


Figure 4-10 Hypoxic hypoxia results from a lack of oxygen or an inability of oxygen to diffuse into the bloodstream.

Hypoxic Hypoxia

Hypoxic hypoxia (also referred to as altitude hypoxia) results from inadequate ventilation or a reduction in the Po_2 and is characterized by a lack of oxygen entering the blood **Figure 4-10**. Hypoxic hypoxia can occur from a variety of causes, including lung disease, right-to-left shunt in the heart, airway obstruction, a reduction in gas exchange area in the alveoli, and a low Po_2. Increased altitude also leads to hypoxic hypoxia.

In the air medical environment, personnel will most likely encounter this condition as a result of reduced atmospheric pressure that causes a reduced alveolar Pa_{O_2}. Simply put, there is not enough oxygen available in an inspired breath at higher altitudes owing to decreased levels of Po_2 oxygen. (Remember Dalton's law: oxygen molecules are further apart, so less oxygen is available.) Diffusion of oxygen is directly dependent on the Po_2. As oxygenated blood flows through the capillaries, the Po_2 level

decreases. The part of the capillaries farthest from the lungs requires at least 1 mm Hg of pressure for diffusion of oxygen to the cells. As altitude increases, the pressure decreases below 1 mm Hg, and aerobic metabolism is inhibited. As metabolism becomes depressed, cellular death begins. Clinical hypoxia actually starts to develop within a few hundred feet from the ground; however, symptoms do not begin to manifest themselves until heights above 5,000′.

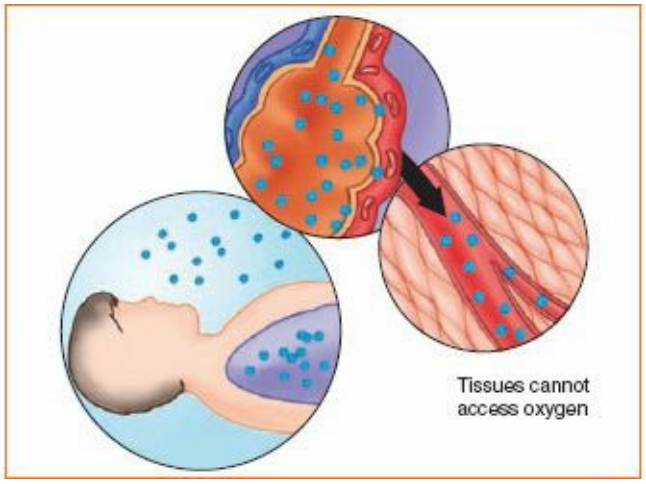


Figure 4-11 In histotoxic hypoxia, plenty of oxygen is available, and it can bind to the hemoglobin, but the tissues cannot access it.

Histotoxic Hypoxia

Histotoxic hypoxia is the cell's inability to adequately use oxygen. Plenty of oxygen is available, but tissues cannot accept it, or oxygen cannot offload from the hemoglobin **Figure 4-11**. During histotoxic hypoxia, the venous hemoglobin oxygen saturation is higher than normal because the oxygen is not being unloaded to the tissues because the tissues are unable to metabolize the delivered oxygen. Histotoxic hypoxia is a result of poisoning, such as by cyanide, which is one of the major toxins generated by housefires. This condition is also exacerbated by the use of narcotics, chewing tobacco, and alcohol.

Stagnant Hypoxia

Stagnant hypoxia occurs when there is failure to transport oxygenated blood. This form of hypoxia occurs when there is a reduction in the flow of blood, but not necessarily a complete stoppage **Figure 4-12**. Outside of the aviation environment, this condition is frequently seen in heart failure and major myocardial infarctions. In flight medicine, stagnant hypoxia can result from venous pooling in the patient during accelerated maneuvers, such as steep turns and other maneuvers that increase the gravitational load on the aircraft. Another practical example is blood pooling in the lower extremities of patients and crew members who sit in an aircraft for extended periods.

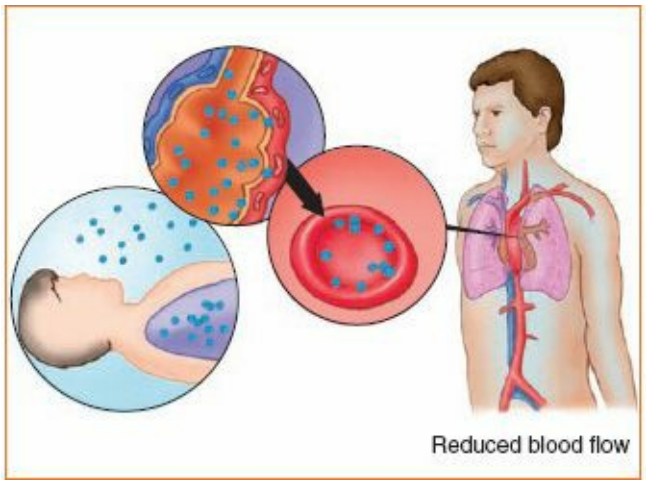


Figure 4-12 In stagnant hypoxia, there is a failure to transport oxygenated blood.

Hypemic Hypoxia

Hypemic hypoxia (also known as anemic hypoxia) occurs when a lack of hemoglobin molecules are present (as seen with exposure to chemicals such as carbon monoxide) or a lack of red blood cells (as seen with hemorrhage or anemia) causes a reduction in the ability of the blood to carry oxygen to the tissues **Figure 4-13**. Oxygen is abundant; however, it is unable to bind to hemoglobin because the amount of hemoglobin is insufficient. Blood loss and anemia are the two most common causes of this condition. Excessive smoking is a major cause as well: smokers are susceptible to hypoxia at lower altitudes than nonsmokers. At sea level, a smoker's apparent altitude is approximately 7,000′. There are several other causes, including carbon monoxide poisoning, the use of nitrite and sulfa drugs, and sickle cell disease, that reduce the amount of hemoglobin available to transport oxygen. Normally, 1 g of hemoglobin carries 1.34 mL of oxygen. In a healthy adult, hemoglobin has the ability to transport 20 mL of oxygen in 100 mL of blood. A reduction of hemoglobin by half reduces the body's transport ability by half. The blood may be completely saturated with oxygen; however, it is insufficient to meet the metabolic demands of the body.

Special Populations

When transporting a pregnant patient by air, oxygen supplementation should always be considered. Even if a pregnant woman's oxygen saturation is 100% (by pulse oximetry), the fetus may still be compromised. The pregnant patient's circulatory system will shunt all richly oxygenated blood to her own vital organs, robbing the fetus and placenta of much-needed oxygen. A normal fetal Pa_{O_2} level is around 45 mm Hg (in a pregnant patient with 90% to 100% Pa_{O_2}). If a pregnant patient's Pa_{O_2} level decreases as a result of hypoxia, the oxygen status of the fetus can quickly become compromised because the fetus has limited reserves.

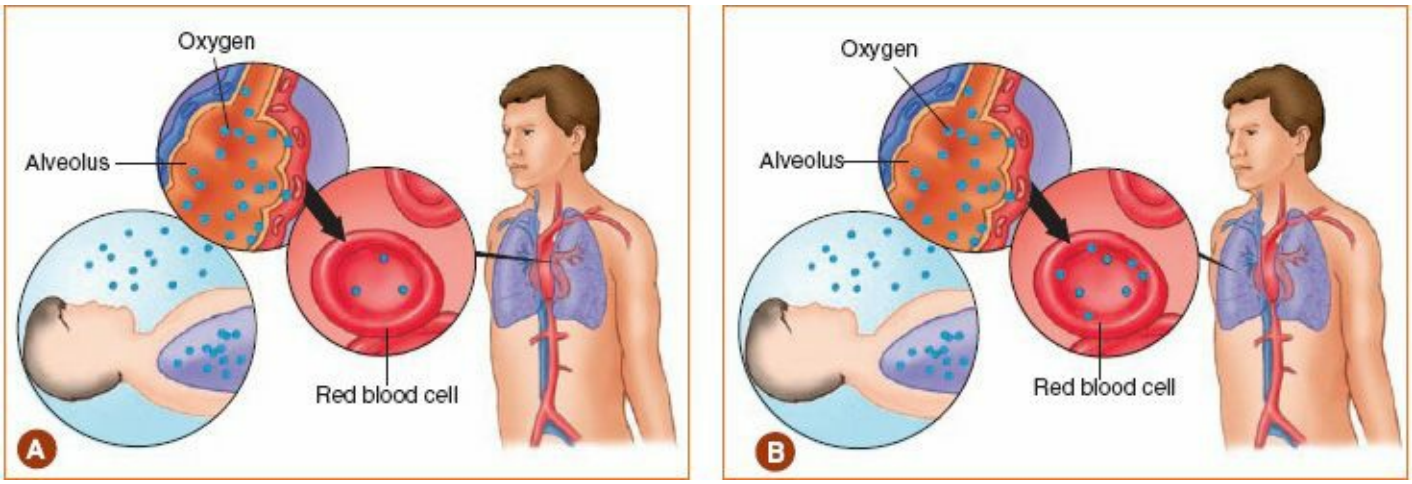


Figure 4-13 With hypemic hypoxia, there is a reduced ability of the blood to carry oxygen to the tissues. **A.** Impaired attachment of oxygen to hemoglobin. **B.** Reduced number of red blood cells.

Four Stages of Hypoxia as They Relate to Altitude

The symptoms of hypoxia can be divided into stages directly related to the altitude, approximate barometric pressure, and the oxygen saturation of the blood. These stages can be divided into the **indifferent stage**, **compensatory stage**, **disturbance stage**, and **critical stage**. Symptoms of hypoxia are listed in **Table 4-2**.

Indifferent Stage

This stage is experienced between sea level and 10,000′; however, the indifferent stage may manifest itself at altitudes as low as 5,000′. Mild hypoxia can cause night vision to deteriorate at altitudes above 5,000′. The military has recognized this problem and requires fighter pilots to breathe oxygen from the ground up during night-flight operations. Physiologically, electrocardiographic changes have been known to occur at altitudes as low as 5,000′. Tachycardia is common, and there is also a slight increase in alveolar ventilation noted during this stage. Oxygen saturation in this stage varies from 98% to 87%. This stage is referred to as the indifferent stage because of the minor physiological effects on the body.

Compensatory Stage

The second stage is called the compensatory stage. As the name implies, the body is able to provide short-term physiologic compensation against the effects of hypoxia. The compensation is dependent on the physical shape of the flight crew member, the physical activity level, and the duration of exposure. In this stage, the respiratory rate and depth may increase and cardiac output increases. This stage is experienced between 10,000′ and 15,000′ (between 39,000′ and 42,000′ if breathing 100% oxygen). Hemoglobin saturation in this stage varies from 87% to 80%.

TABLE 4-2 Symptoms of Hypoxia
Confusion/altered mental status Fatigue
Headache
Changes in vision acuity • Tunnel vision • Blurred vision and/or inability to focus

• Difficulty focusing from near to far • Loss of night vision
Euphoria
Tingling in the hands and feet
Feelings of air hunger Tachypnea
Cyanosis of the skin Irregular heart rhythms
Short-term memory loss
Decreased muscular coordination
Loss of hearing
Diminished sense of pain
Diminished sense of touch and feel
Difficulty speaking; stammering
Loss of self-criticism
Overconfidence
Overly aggressive behavior

Disturbance Stage

In the disturbance stage, tissue can no longer depend upon the physiologic compensatory mechanisms for sufficient oxygen supply. This stage is characterized by subjective and objective symptoms of hypoxia **Table 4-3**. During altitude testing, some subjects did not experience the subjective symptoms before becoming unconscious from hypoxia. The following can be affected by hypoxia: respiratory system, senses, mental processes, manifestations of personality, and psychomotor functions.

TABLE 4-3 Stages of Hypoxia

	Indifferent Stage	Compensatory Stage	Disturbance Stage	Critical Stage
Altitude	0′-10,000′	10,000′-15,000′	15,000′-20,000′	20,000′-25,000′
Oxygen saturation (%)	98-90	89-80	79-70	69-60
Symptoms	Decrease in night vision	Drowsiness Poor judgment Impaired coordination Impaired efficiency	Impaired flight control Impaired handwriting Impaired speech	Circulatory failure

Senses

Vision, hearing, and sense of touch are affected during the disturbance stage. Visual ability decreases as the eye muscles become weak and uncoordinated. Sensations of touch and pain become diminished and eventually are lost. Weakness and loss of muscular coordination are experienced and become worse with the degree of hypoxia. This loss of muscle coordination, in conjunction with confusion, quickly becomes a deadly combination. Accurate communication in the aircraft environment is imperative to the overall safety and success of the mission. Although hearing is one of the last senses to be impaired, loss of hearing from hypoxia will further confound the safety and success of the mission.

If a medical crew member is having difficulty following simple commands or performing simple tasks, the crew member and the other members of the medical and flight team should consider hypoxia. At times, crew members cannot recognize hypoxia in themselves but can recognize it in others.

Cognition

One of the most dangerous hallmarks of hypoxia is the early impairment of intellect that makes it impossible for people to comprehend their own disability. As the cerebrum becomes hypoxic, the victim loses his or her ability to make coherent judgments and calculations. Reaction time becomes slower, and the short-term memory is severely impaired. All of these impairments prevent the person from recognizing the ongoing effects of hypoxia.

A common misconception among flight crews is that they can learn all of the early signs of hypoxia and be able to take immediate corrective action. Unfortunately, because one of the earliest effects of hypoxia is impaired judgment, the flight crew can recognize hypoxia but may take inappropriate corrective actions. Aviators have been known to have adequately recognized hypoxia but, in an attempt to correct the problem, immediately disconnect themselves from their only oxygen supply.

Personality Manifestations

Hypoxia can cause the emergence of symptoms similar to those a person may exhibit while under the influence of alcohol. These symptoms include the following:

- Aggressiveness
- Euphoria
- Irritability
- Overconfidence
- Depression

Psychomotor Functions

As hypoxia is induced, muscular coordination decreases. When the partial pressure of alveolar carbon dioxide falls below 25 mm Hg, muscular coordination deteriorates. As the partial pressure continues to fall, muscle coordination deteriorates to levels that are incompatible with coordinated activity. The first resulting problems are speech difficulty, illegible handwriting, and poor coordination in flying the aircraft. As the degree of hypoxia increases, delicate and fine muscular movements become impossible, and gross motor movements become significantly impaired. It is interesting to note that stammering and illegible handwriting are two of the hallmark signs of typical hypoxic impairment.

Critical Stage

The last stage of hypoxia is called the critical stage. This stage occurs at 20,000′ and above (44,800′ and above with the use of 100% oxygen). Within 3 to 5 minutes of hypoxygenation, judgment and coordination deteriorate to the point of inadequate or inappropriate function. In this stage, mental confusion is quickly followed by incapacitation, unconsciousness, and death, if uncorrected. Hemoglobin saturation in the critical stage drops to less than 65%.

Hyperventilation

Hyperventilation symptoms mimic those of hypoxia, so it is critical that flight crew members first address the possibility of hypoxia before assuming that the problem is caused by hyperventilation. Hyperventilation *and* hypoxia result in confusion, poor judgment, and inappropriate corrective maneuvers.

Hyperventilation is caused by the subconscious reaction to a stressful situation. This reaction is

manifested by an abnormal increase in the volume of inspiratory and expiratory air and by tachypnea, which results in respiratory alkalosis as the carbon dioxide is blown off. This aberration in blood gases can have serious consequences. The lack of carbon dioxide also affects the automation of respiration. Normal control of respiration is mediated reflexively through the chemoreceptors in the aorta and the carotid artery by arterial oxygen deficiencies. In healthy people without a hypoxic drive, ventilation rate and depth are controlled by carbon dioxide and acid-base balance of blood circulating through the respiratory center in the medulla. Excessive elimination of carbon dioxide with hyperventilation results in a rapid decline in sodium bicarbonate in the blood. This decline causes an elevation in the pH of the blood. Cellular activity is dependent on proper acid-base balance. When the pH of the blood falls out of normal range, homeostasis is interrupted in the cell, and cellular activity quickly declines or stops.

Hyperventilation leads to several important physiologic changes that begin a cascade of events. As minute volume (tidal volume × rate) increases, the partial pressure of carbon dioxide decreases, which results in an increase in the pH of the blood. The cerebral blood vessels respond to the decrease in carbon dioxide by vasodilation. This vasodilation causes blood to shunt to various parts of the body while starving other areas. Oxygen is used up in the areas with pooled blood, whereas immediate cellular hypoxia and anoxia occur in the areas with decreased circulation, which is why routine hyperventilation of head-injured patients is no longer recommended. Unconsciousness quickly follows the induction of prolonged or significant hypoxia into cerebral tissue.

Hyperventilation can cause assorted symptoms, including the following:

- Light-headedness
- Feelings of suffocation
- Drowsiness
- Tingling in the extremities
- Painful muscle spasms
- Ataxia
- Disorientation
- Unconsciousness

One of the most disastrous effects of hyperventilation is that it promulgates panic. Maintaining control is one of the few remedies to prevent or overcome panic. Hyperventilation contributes to the overwhelming sense of panic. If a flight crew member is unable to control his or her hyperventilation, panic may ensue related to progressive hypoxia.

Recognition and Treatment of Altitude-Induced Hypoxia

The key to recognition of altitude-induced hypoxia is a thorough knowledge and understanding of basic flight physiology. As previously mentioned, many aviators have erroneously believed that they can recognize hypoxia as it is occurring and immediately correct the problem. Recovery from hypoxia is rapid when sufficient oxygen is supplied. People who are on the precipice of unconsciousness can regain full mental abilities within 15 seconds after receiving high-flow oxygen. In fact, studies show that a hypoxic person who rapidly breathes in 100% oxygen may experience sudden dizziness, which is quickly resolved, followed by complete restoration of function. This condition is sometimes referred to as the **oxygen paradox**.

Prevention is the key to treatment, and avoidance of hypoxia is the key to safety. If hypoxia is detected, the flight crew must immediately use supplemental oxygen and descend to below 10,000′. Hypoxic crew members are a valid reason to declare an emergency with air traffic control.

Supplemental Oxygen Requirements

As discussed in Chapter 3, air medical transport providers are subject to one of two rules, part 91 or part 135 of the FAR. The FAR part 135.89 governs the use of supplemental oxygen by pilots and provides rules for pressurized and nonpressurized aircraft. In nonpressurized aircraft (helicopters included), at altitudes from 10,000′ through 12,000′, each pilot must use oxygen continuously if the duration of flight at this altitude is longer than 30 minutes. Pilots must use oxygen at all times above 12,000′. In a pressurized aircraft when the cabin altitude exceeds 10,000′, the same rule applies. At altitudes from 25,000′ through 35,000′, each pilot must use continuous oxygen unless the aircraft is equipped with an approved quick-donning-type mask. Pilots must wear an oxygen mask continuously above 35,000′. The more stringent rules regarding oxygen use as altitude increases are because of the dramatic decrease in time of useful consciousness as altitude increases; above 35,000′, pilots will have mere seconds to respond to a sudden loss of cabin pressure.

Passengers will be provided supplemental oxygen as well, in accordance with FAR part 91.211. At cabin altitudes above 15,000′, all occupants will be provided with supplemental oxygen. In a pressurized aircraft, there must be a 10-minute supply of oxygen available for each occupant if the aircraft is to be operated above 25,000′. These rules for supplemental oxygen are for crew and nonpatient passengers only. A sick or injured patient will likely need supplemental oxygen at all altitudes to prevent hypoxia.

Pressurized and Nonpressurized Aircraft

The most effective method to protect people from the physiologic effects of reduced barometric pressure is by pressurizing the aircraft. This pressurization is accomplished by increasing the barometric pressure in the aircraft above the ambient pressure outside the aircraft. The higher the differential pressure required between the aircraft cabin and the ambient pressure, the stronger and heavier the fuselage construction must be. A higher differential pressure also requires a greater capacity of the pressurization system. Aircraft pressurization requirements increase engineering and maintenance costs of the aircraft and require more power.

There are two primary methods used for pressurizing aircraft: the isobaric system and the differential control method for pressurization. The majority of civilian and military aircraft use isobaric systems for pressurization. The differential control (also known as conventional or isobaric-differential) method is designed primarily for military aircraft and has an aircraft cabin pressure that fluctuates with altitude.

Isobaric Control System

The **isobaric pressurization system** is the most frequently used method for pressurizing aircraft. This system is designed to maintain the aircraft cabin at a constant pressure (usually between 5,000′ and 8,000′) despite falling barometric pressure outside the aircraft. This system is designed to maximize the comfort of the crew and passengers and does not require the use of supplemental oxygen as long as the system is working properly. This type of system also minimizes the effects of fatigue on the crew and passengers and allows for maximum mobility of the crew and passengers.

The drawback of the isobaric system is that it requires a heavier fuselage and airframe to keep the higher pressure inside the aircraft cabin. It also adds more weight to the aircraft because of the equipment needed to pressurize the aircraft, along with the heavier fuselage. Another drawback is the explosive rate of decompression (see rapid decompression discussed in “Aircraft Depressurization”) that can occur when pressure inside the aircraft cabin is much greater than the ambient outside pressure.

Differential Control Method

A differential control system is used primarily by tactical military aircraft. The differential system is used by aircraft that fly above the designed and engineered limits of the isobaric system. This system does not maintain a constant cabin altitude. Instead, it is designed to ensure that the cabin pressure does not exceed the outside pressure by a predetermined amount, for example 5 psi. As altitude varies, so does the internal cabin pressure. The rate of pressure change, however, is less drastic than that of the outside pressure and is designed to slowly adjust pressures within the predetermined ratio. Most differential controls maintain the cabin pressure at a pressure the same as at 8,000′ until the aircraft climbs above 23,000′. At that point, the differential control maintains the cabin at a 5-psi difference from the outside pressure.

The drawback to the differential control method is that supplemental oxygen and pressure suits must be available and used at certain altitudes. However, advantage is gained in a lightweight aircraft fuselage and less of a risk of explosive decompression. Any military aircraft that can potentially take gunfire uses the pressure differential system. Therefore, if there is penetration into the aircraft cabin, the rate of depressurization is less than if the aircraft cabin were maintained at a higher cabin altitude and there is less risk for catastrophic failure of the aircraft.

Aircraft Depressurization

Decompression at altitude is categorized as "slow" or "rapid." Rapid cabin depressurizations are dramatic events. With a loud explosion and numerous master caution warning horns sounding in the cockpit, it is quickly obvious to everyone that a major emergency has occurred. When rapid cabin depressurization occurs, the occupants of the aircraft are immediately exposed to the dangers of hypoxia, decompression sickness, gastrointestinal expansion, and hypothermia. In addition, the cyclonic winds may lead to the loss of personnel or passengers through the aircraft opening that caused the depressurization, and a dense fog and icy temperatures quickly fill the cabin. The dissipation rate of the fog is dependent on the size of the aircraft. Larger aircraft will take longer to clear the fog. All commercial pilots are extensively trained and tested on rapid decompression and emergency descent procedures.

A slow decompression can occur when a small leak develops in a pressurized aircraft. This type of decompression is dangerous because of the insidious onset and undetectable loss of oxygen leading to hypoxia and death if uncorrected. In 1999, golf legend Payne Stewart died when his Lear 35 jet aircraft developed a small leak at 45,000′ and everyone onboard the aircraft died of hypoxia. The aircraft flew for 3 hours with all of the crew presumed dead before it crashed. Military chase planes observed frost built up on the inside of the windows, indicating that the aircraft was depressurized.

A review of data collected by the National Aeronautics and Space Administration shows that of all decompression events reported worldwide each year, generally 40 to 50 will be classified as rapid decompression. Of these, approximately 70% will occur above 30,000′. If a loss of cabin pressure occurs, a descent must be made immediately to a level at which cabin altitude can be maintained at or below 10,000′ and oxygen will be used by all occupants until the flight crew indicate it is no longer necessary. As previously mentioned, rapid cabin depressurization also dramatically affects the effective performance time and the time of useful consciousness—reducing these times by up to half.

Primary Forces That Act on an Aircraft

There are four primary forces that constantly act on an aircraft in flight: **lift**, **thrust**, **weight** (gravity), and **drag** **Figure 4-14**. Lift counteracts weight, and thrust opposes drag. Essentially, greater amounts of thrust and lift allow an aircraft to take off. Conversely, greater amounts of drag and weight must exist for an aircraft to land. In straight and level flight, thrust equals drag and lift equals weight. The forces of flight and how they relate to flight medicine were discussed in Chapter 3.

Primary Stressors of Flight

There are many stressors that flight crew members will experience during their career; however, some stressors may cause catastrophic outcomes. When flying in an airplane or a rotor-wing aircraft, rapid altitude changes are frequently experienced. When the body is exposed to significant altitude changes without the proper precautions, adverse outcomes may result as the body attempts to maintain homeostasis. As the body is exposed to altitudes of 8,000′ and above, the atmospheric pressure continuously decreases, making it more difficult to breathe. The difficulty in breathing is a direct result of a decrease in the amount of oxygen that is available in one breath. If the proper precautions are not taken, CCTPs, other crew, and patients may become hypoxic and begin to experience symptoms such as confusion, fatigue, visual impairment, headache, nausea, and euphoria.

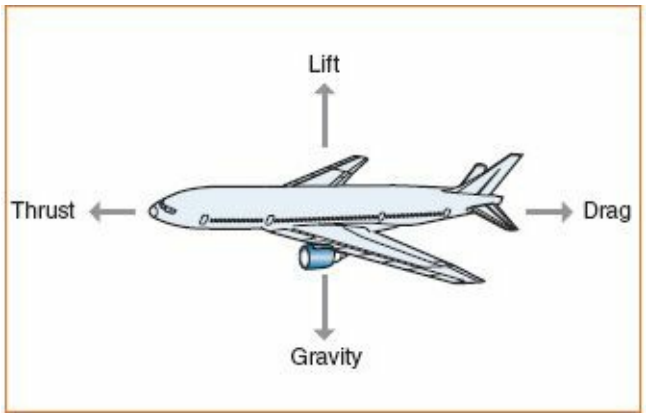


Figure 4-14 The forces of flight: weight, lift, drag, and thrust.

Decreased Levels of P_{O_2}

As mentioned previously in this chapter, decreased levels of P_{O_2} can quickly cause hypoxia in flight crew and passengers. At 15,000′, the barometric pressure is 429 mm Hg, and average values for a healthy patient would include an oxygen saturation of 80% (the body requires 87% to 97%) and a Pa_{O_2} of 44 mm Hg (the body requires 60 to 100 mm Hg) **Table 4-4**. Remember that these values are averages for a healthy person; they are not representative of critical care patients.

Barometric Pressure Changes

The greatest pressure change in an aircraft takes place from sea level to 5,000′. Therefore, problems associated with pressure must be considered even in nonpressurized aircraft that are not flying at altitudes requiring supplemental oxygen or cabin pressurization. During daylight hours for nonpressurized aircraft, 10,000′ of altitude is the ceiling at which crew members must begin to use oxygen. Because the loss of night vision is one of the first symptoms of hypoxia, the ceiling drops to 8,000′ for nighttime flight operations that exceed one hour in length. The alveolar P_{O_2} at 10,000′ is approximately 61 mm Hg, which produces the maximum acceptable degree of hypoxia allowed. The upper limit for the indifferent stage of hypoxia is 10,000′. However, deviation by a few thousand feet for a short time will have little difference. For this reason, certain military operations are allowed at 13,000′ as long as the duration of the flight is less than 3 hours. In addition to causing hypoxia and malfunctioning pacemakers, ambient pressures and increasing altitudes can cause discomfort in air-trapped organs and sinuses (for example, the ears and gastrointestinal system).

Thermal Changes

Flight crew members are subjected to a variety of thermal extremes ranging from the very cold to the very hot. These extremes in temperature affect the metabolic rate and demands on the body. Temperature changes increase the oxygen demands on the body and make the body less tolerant of the effects of hypoxia. Temperature changes can result in the effects of hypoxia at lower altitudes than normally expected.

Temperature declines with increases in altitude. Temperature decreases by 3°F to 5°F (1°C to 2°C) per 1,000′ gain in altitude, depending on humidity. (At altitudes from 35,000′ to 99,000′, the temperature remains relatively constant at –32°F [–50°C].) In helicopters, pilots often take advantage of the decline in temperature at higher altitudes during the summer to assist with cooling the aircraft cabin. If the air conditioner cannot keep the cabin cool because of high ambient temperatures, the aircraft can ascend and take advantage of the cooler temperatures at higher altitudes.

The flight crew is also subjected to hot temperatures because they frequently must operate outside at emergency scenes for extended durations during summer months. The flight crew must then work aggressively in the tight, hot confines of the aircraft to provide medical care to a patient in critical condition. Most flight crews wear fire-retardant flight suits **Figure 4-15**, which are an added weight and do not allow heat to rapidly dissipate. In addition, most rotor-wing flight personnel wear helmets, which do not facilitate cooling and can lead to increases in the core body temperature.

Table 4-4 Partial Pressures of Oxygen at Various Altitudes

Altitude	Atmospheric Pressure (mm Hg)	Pa_{O_2} (mm Hg)*	Pv_{O_2} (mm Hg)	Pressure Differential (mm Hg)	Blood Saturation (%)*
Sea level	760	100	40	60	98
10,000′	523	60	31	29	87
18,000′	380	38	26	12	72
22,000′	321	30	22	8	60
25,000′	282	7	4	3	9
35,000′	179	0	0	0	0

*These are average measurements for a healthy patient. Pa_{O_2}, partial pressure of oxygen (arterial); Pv_{O_2}, peak oxygen consumption (the highest amount of oxygen consumption during an exercise period).

Figure 4-15 A fire-retardant flight suit (Nomex).

Another problem encountered is that flight personnel are subjected to a wide range of ambient temperatures because of the range of their aircraft. Flight crew assigned to rotor-wing aircraft may experience a temperature of 80°F (26.7°C) at their base but fly less than an hour into mountainous areas where the temperatures can be less than 40°F (4.4°C). Flight crews assigned to fixed-wing aircraft may see an even broader range of temperatures as they start in a northern climate in the snow and end in the hot sun in a southern climate. A secondary hazard also is the greenhouse effect, which can result in an increase in the temperature by 50°F (10°C) to 59°F (15°C) in the cockpit or cabin of a small aircraft simply because of radiant solar heat.

Flight crews must be cognizant of the potential for heat stress. Core body temperatures that exceed 100°F (37.8°C) result in decreased short-term memory, degradation of motor skills, and a general decrease in performance. Heat stress also causes increased irritability and poor judgment, increases the propensity for motion sickness and hypoxia and potentiates the effects of gravitational forces, which are discussed later in this chapter.

Vibration

Vibration is noted in all helicopters, most propeller-driven aircraft, and, minimally, jet-engine aircraft. Studies have found that vibrations between 1 and 12 Hz can cause significant effects on the body. The low-frequency vibrations can cause several untoward effects, including body discomfort, pain (usually in the abdomen or chest), decreased vision, and, most notably, fatigue. Excess vibration is an issue that must be addressed with aircraft mechanics and manufacturers. However, there are some modifications that can be made to reduce the effects of vibration. Increasing the cushioning on seats is one of the most important modifications, as is proper use of shoulder and lap belts, which reduces the transmission of vibrations.

Prolonged effects of vibration can increase the core body temperature as the body constantly tries to fight the effects of vibration and movement. As mentioned, one of the most noted effects of vibration is fatigue. As the body becomes fatigued with prolonged exposure to vibration, it becomes more susceptible to the effects of vibration and, thus, continues in an unbroken cycle.

To combat crew fatigue due to vibration, there are restrictions regarding the amount of time a crew is allowed to fly on any given shift before resting. Preventive measures include prohibiting medical crew members from leaning against the airframe (to decrease vibration) and providing extra padding for patients so they are not leaning against the airframe.

Decreased Humidity

Humidity is the degree of moisture (water vapor) in the air and is expressed as a percentage. Humidity is relative to temperature. As temperature increases, so does humidity. Conversely, as temperature falls, so does the relative humidity. Because temperature falls with altitude, so does humidity. Because of the relatively low levels at which they fly, humidity levels in rotor-wing and propeller aircraft are higher than those of jet aircraft. Jet aircraft fly at high altitudes where there is low humidity. Any humidity in the air is quickly used on inspiration by the flight crew and passengers. Any other humidity that was trapped in the aircraft from wet clothes or perspiration is also used quickly until all of the moisture is lost. The air in a pressurized jet aircraft is constantly being recirculated through filters, and any moisture is drawn off by the system. In highspeed, high-altitude, long-range flights, less than 5% humidity remains after 2 hours of flight and less than 1% remains after 4 hours of flight.

During a long flight, the dry air can cause dry, cracked mucous membranes, chapped lips, and sore throats and can lead to dehydration. This lack of humidity is why many people feel fatigued or jet-lagged

after a long commercial flight. These problems can further be compounded in injured or ill patients, so it is important to ensure that patients are properly hydrated before and during the flight **Figure 4-16**. Patients who are receiving supplemental oxygen are at twice the risk of becoming dehydrated because oxygen causes moisture to dry up in the respiratory system. If available, humidified oxygen should be used during air transports. The dry air in the aircraft can also cause drying of the cornea in unconscious or intubated patients if their eyes are not protected by taping or using artificial tears. In addition, owing to the relatively dry environment, consideration should be given to increasing the rate of IV fluids or providing additional oral hydration, if possible. To combat the effects of decreased humidity, crew members are encouraged to increase their fluid intake.

Figure 4-16 Proper hydration for all air transport patients should be provided.

Noise

Noise is defined as any unwanted sound. Long exposure to noise can damage the soft tissue of the inner ear. The cells and nerves in the inner ear may be completely destroyed by continuous exposure to loud sounds. If enough cells are damaged, hearing is permanently damaged.

To determine how noise will affect hearing, three factors must be considered: loudness, pitch, and duration of exposure. The results of loudness and duration are inversely proportional. The louder the sound, the less time before damage occurs. One of the biggest problems with damage caused by noise involves the subtle symptoms of hearing loss. Hearing loss usually manifests as feelings of pressure or fullness in the ears, muffled speech, or ringing in the ears. These symptoms may go away within minutes, hours, or days after the exposure or, depending on the damage, may never go away. It was once thought that only extremely high sound levels create hearing loss; however, cumulative exposure to levels around 70 dBA without protection have been shown to lead to irreversible hearing loss **Table 4-5**. Sound at 140 dB causes pain and indicates danger of immediate hearing loss.

There are several proven negative consequences to exposure to noise. Noise causes a release of epinephrine, which causes increased myocardial oxygen demand, increased respiratory rate, and vasoconstriction. This vasoconstriction can cause a significant increase in blood pressure, especially in people who are susceptible to hypertension. High levels of noise are also proven to cause headaches, stomach ulcers, feelings of apathy, and feelings of a “headrush.” The US Environmental Protection Agency has established a link between exposure to high levels of noise for an extended period and low-birth-weight neonates and fetal birth defects. A study in Japan showed that a developing fetus is most susceptible to loud noises 14 to 60 days after conception. Because of the difficulty of studying fetuses in utero, research on the exact amount of noise and the duration to noise is questionable. Psychological effects of noise include sleep disturbance, increase in aggressive behavior, and overall increases in chronic stress levels. Noise has also been found to intensify the adverse effects of carbon monoxide and hypoxia, both of which are prevalent in the aircraft environment. Prolonged exposure to noise is also a

significant factor in causing fatigue.

TABLE 4-5 Noise Exposure Without Hearing Protection That Leads to Irreversible Hearing Loss

Sound	Decibel Level (dBA)*	Time of Exposure
Whispering	20	No limit
Normal conversation	60	No limit
Car	70	No limit
Diesel truck	100	2 h
Jackhammer	100	2 h
Helicopter	105	1 h
Live rock music	90-130	30 min to 8 h
Propeller aircraft	120	7 min
Jet aircraft	140	Imminent hearing loss at any length of exposure

*dBA refers to noise levels from A-weighted noise-monitoring equipment, used in noise studies to filter noises so that they are more similar to how they would be interpreted by the human ear.

Noise in the cabin of the aircraft can make it difficult for patients and care providers to communicate, which can lead to patient anxiety. The noise also makes it difficult for CCTPs to assess heart and lung sounds. Many monitoring errors and malfunctions can be attributed to the noise and vibrations associated with air medical transport. Pulse oximetry and hemodynamic monitoring are two technological advances that can help lessen these negative effects and permit early recognition of complications. To preserve the hearing of patients and accompanying family members, CCTPs must remember to provide them with appropriate hearing protection for the flight. In addition, CCTPs and flight crew members must protect their hearing. Many employers require aircraft personnel to wear hearing protection to limit the effects of noise in the aircraft.

A recent study showed that noise causes apathy and that people will instinctively attempt to get away from noise, even if it means ignoring the obvious. An example in the aircraft environment can be seen when loading or unloading a patient when the aircraft is running. Because of a desire to get away from the sound of the aircraft during loading, there may be a tendency to hurry and load the patient while omitting safety features such as fastening all seatbelts on the stretcher. This is a well-documented phenomenon of which flight personnel must be aware, especially when monitoring a patient.

There are several ways to prevent hearing loss in the critical care transport environment. One way is to make equipment modifications that reduce noise exposure. Several such modifications have been made to sirens in the ground transport environment to minimize the effects of the noise on the crew members, while maximizing the effect of the siren on traffic. Previously, siren speakers were mounted on the roofs of ambulances, but are now mounted in the front of the truck above the bumper. This modification was made to decrease the amount of noise to which the crew was exposed, and to also place the siren speaker closer to traffic level. Crew members who are frequently exposed to siren noise should also wear noise-attenuating headsets or ear plugs when the siren is in use. Many ambulances now are equipped with headsets that allow the crew to communicate while protecting them from the external noise of the siren.

Crew members who work in the aircraft environment must be very attentive to the potential for hearing loss. Crew members should be required to always wear hearing protection anytime that they are operating around an aircraft that is running. All crew members working inside a rotor-wing aircraft should always wear hearing protection. Often times this is accomplished by wearing a helmet with an internal communication system. Many crew members elect to wear soft hearing protection under their helmets as a secondary means of protection. For those rotor-wing programs that do not utilize helmets, the use of headsets should always be required. Not only do the helmets and headsets protect from noise, they also facilitate communication between crew members.

Crew members who operate on fixed-wing aircraft may or may not be required to have hearing protection inside the aircraft. Hearing protection is dependent on the type of aircraft (propeller vs jet) and the individual aircraft design. Generally speaking, larger aircraft will have better sound insulation and will not require the use of hearing protection inside the aircraft.

Fatigue

Fatigue is much more than simply the lack of sleep; however, the lack of sleep is the foundation for fatigue. Most physiologic problems encountered in the flight environment can cause significant fatigue. These physiologic problems can quickly become cumulative when combined with the lack of restful sleep. Many flight crew members begin their shift already tired. Studies have shown that greater than 50% of flight personnel are chronically fatigued because of long work hours. Studies conducted by the FAA and the National Transportation Safety Board find that crew fatigue is often times a mitigating factor in aviation accidents and near misses. Air medical crew members most commonly work 12- or 24-hour shifts. However, most pilots are limited by federal regulations to a maximum of 8 flight hours in any 24-hour period and are required to have at least 8 hours of uninterrupted rest. Most companies and flight crews do not rely on government agencies to police their duty time; instead, they impose more stringent requirements on themselves. In addition to lack of sleep, three other factors contribute to crew fatigue: jet lag, vibration of the aircraft, and poor diet, which often includes missed meals due to the very nature of air medical transport.

In the fixed-wing environment, jet lag becomes another fatigue factor. When flight crew travel across time zones, their bodies require time to adjust to the new time zone. Studies have shown that it takes 1 day for each hour of difference from the original time zone to reset the body's own circadian rhythm.

It is not uncommon for CCTPs to experience fatigue while flying, but the challenge arises when CCTPs are required to maintain competency while caring for critically ill or injured patients. Fatigue causes delayed reaction time, thus increasing vulnerability to error. The Commission on Accreditation of Medical Transport Services, discussed in Chapter 1, emphasizes crew rest and ensuring that duty days do not exceed 16 hours. To avoid beginning another flight in a fatigued state, CCTPs should immediately request "crew rest" as soon as they feel fatigued. This request must be granted and typically lasts for 4 hours, occurring between flights. At the end of the 4 hours, a CCTP is reassessed to determine fitness for duty.

The constant vibration that aircrew members experience during flight also leads to fatigue. In order to combat the fatigue from vibration that is often experienced during flight, crew members must be in good physical shape. Crew members can also gradually become resistant to vibration fatigue by increased flight time and the body's ability to compensate with repeated exposure.

The fourth contributing factor to crew fatigue is the 24-hour operational nature of air medical transport. Most air medical programs operate around the clock, and flight crews work varied shift schedules ranging from 8-hour shifts to 24-hour shifts. Crew members may work a varied shift schedule that frequently rotates between day shifts and night shifts, which does not allow for crew members to establish a natural circadian rhythm. There has been an increased focus within the air medical industry to

limit shifts to a maximum of 12 hours in length in order to prevent medical crew fatigue. Air medical programs are encouraged to allow crew members the ability to have crew rest periods incorporated into shifts in order to minimize fatigue.

Gravitational Forces

The body's response to **gravitational forces** is affected by the intensity of the impact of acceleration, its direction, the length of time the body is subjected to stress, the time it takes for gravity's effects to appear, and the individual's unique physical makeup. Although termed gravitational force, it is actually rapid acceleration or deceleration, not gravity, that affects the body. One unit of gravitational force (informally referred to as g force or as 10*g*, for example) is equal to the weight of the object. The gravitational force experienced is multiplied by the weight to determine the actual force on the body. For example, a 100-lb person who experiences 10*g* will have 1,000 lb of force on his or her body.

There are two types of gravitational forces that can be experienced. Negative gravitational forces result from a steep dive in an aircraft. Positive gravitational forces result from high-speed acceleration, climbs, or high-speed turns. Positive gravitational forces push blood away from the brain, whereas negative gravitational forces push blood toward the brain. Most humans can survive a positive gravitational force of 9*g* but are only able to tolerate a negative gravitational force of 2*g* or 3*g*. Most humans will become unconscious around gravitational forces of 6*g* to 8*g*.

Physiologic response to gravitational forces is influenced by the length of time a person feels the effects of acceleration. Acceleration "pushes" that last fewer than 0.2 seconds can be tolerated at a relatively high gravitational force as long as the duration is brief. People who are in car crashes can reach a gravitational force up to 12*g*, but very quickly. A person who experiences a sustained gravitational force of 12*g* will have a greater physiologic impact on the body as a result of the force. Exposure to a gravitational force that lasts longer than 0.2 seconds is considered sustained or prolonged. During prolonged exposure, organs shift and third spacing (discussed later in this chapter) can occur.

Gravitational forces can have significant impacts on the human body. The first physiologic sensation of gravitational force is the feeling of being pushed down in a seat or feeling weightless, depending on whether the aircraft is accelerating or decelerating. As the gravitational force increases, breathing becomes labored. Acceleration compresses the rib cage and lungs, making it difficult to draw in air or exhale. This can result in exhaustion and air hunger. Hypoxia arises when gravitational force causes blood to leave the brain (for example, during a high-speed acceleration or turn) and transfer to the lower extremities. The body must then work much harder to circulate blood to the brain. Hypoxia can result in a loss of peripheral vision, when the body is subjected to gravitational forces of 5*g* to 6*g*. As the gravitational force continues, tunnel vision develops, and, eventually, vision becomes gray (loss of color perception). Further exposure to acceleration stress will cause a person to experience blackout—a loss of all vision. During this time, the organs are displaced downward, significantly affecting blood flow. If the gravitational force continues, the flight crew member will lose consciousness (also called gravitational force loss of consciousness, or gravitational force–induced loss of consciousness). Other signs and symptoms include petechiae, rashes, and bruising; loss of consciousness with accompanying seizures, amnesia, and confusion; and cardiac arrhythmias (tachycardia and bradycardia), heart blocks, and stress cardiomyopathy.

Several factors affect tolerance to sustained exposure to gravitational force. The response is dependent on the person's conditioning, psychological stress level, and amount of muscular straining while experiencing the gravitational force. Because of these factors, military pilots are given extensive training in gravitational force maneuvers to improve their tolerance.

Several other factors are known to decrease tolerance to positive gravitational force during

acceleration. Age can play an important role in determining the extent to which a person may be affected by acceleration. People older than 60 years, for example, have a diminished tolerance to gravitational forces. Infection and illness, resulting in fever and dehydration, also increase the body's susceptibility to the effects of gravitational forces. A person with hypoglycemia will lose consciousness, under acceleration stress, 0.5*g* earlier than someone with a higher, normal blood glucose level. Alcohol consumption will cause a person to feel effects 0.1*g* to 0.4*g* sooner.

Hypoxia and heat stress, conditions that often arise during periods of acceleration, also alter the body's response to gravitational force. Heat stress causes the body's temperature to rise, resulting in a loss of moisture. A person suffering heat stress is more immediately at risk of blacking out. Hypoxia describes a decrease in oxygen levels within the blood, which in turn inhibits blood flow to the brain and eyes. This condition also decreases physical tolerance to positive gravitational force during acceleration. Furthermore, the stress of a situation may cause a person to hyperventilate, a physical response that will reduce the body's resistance to the effects of acceleration.

Spatial Disorientation and Illusions of Flight

Particular movements or situations in flight can make it difficult for a person to keep track of his or her orientation in relation to surroundings. This concept, spatial orientation, is important in studying flight physiology. In flight, a person may not be able to trust his or her senses to orient himself or herself. When this happens, a person experiences **spatial disorientation**—a condition in which he or she has an incorrect understanding of the body's position with respect to earth.

To maintain spatial orientation on the ground, there are three key components. Effective perception, integration, and interpretation of visual, **vestibular**, and **proprioceptive** sensory information are all essential to maintaining orientation. (Vestibular sensory information comes from the organs of equilibrium located in the inner ear. Proprioceptive sensory information comes from the skin, muscles, tendons, and joints.) The brain recognizes changes in linear acceleration, angular acceleration, and gravity and attempts to relate them with visual input. Spatial orientation in flight is difficult to achieve because of conflicting sensory input. When visual, vestibular, and proprioceptive sensory stimuli provide conflicting information, a sensory mismatch occurs and results in illusions and spatial disorientation. Of all of these senses and stimuli, visual reference provides approximately 90% of the information to maintain spatial orientation. Visual reference is a dominant source of input that will overpower conflicting sensations from other systems, and people seldom realize when the brain has received contradictory information. Learning not to rely on visual reference is an important factor for pilots who are flying by instruments. In fact, a great deal of time during instrument training is spent convincing students to trust the instruments and not their senses. Of note, even birds (with the exception of bats) must have vision to maintain orientation and safe flight. Like humans, birds become disoriented in clouds or in fog and are susceptible to spatial disorientation. Senses provide invalid information because the brain is convinced that "down" is the bottom of the aircraft, no matter the actual position or angle of the aircraft.

It is important to note that because all of the illusions discussed in this section result from vestibular ear disorders, these illusions can also cause significant nausea and vomiting, further debilitating the flight crew. Spatial disorientation is responsible for 5% to 10% of all general aviation accidents, and most of the time (90%), accidents resulting from spatial disorientation are fatal.

Types of Spatial Disorientation

Spatial disorientation has been studied for many years by military and civilian organizations. Spatial disorientation is divided into three types: type I, type II, and type III.

Type I

Type I spatial disorientation occurs when the pilot does not notice that spatial disorientation exists because the senses confirm that the pilot's experience is real. If a flight crew member does not sense danger, he or she will not respond to the disorientation. This is a potentially deadly type of disorientation, because it can lead to a crash. A pilot experiencing type I spatial disorientation may fly directly toward the earth without realizing that is where he or she is headed.

Type II

Type II spatial disorientation occurs when the pilot does not realize that he or she is experiencing spatial disorientation, but senses that something is wrong. Usually the pilot will misinterpret the problem as a malfunction of the controls and trust his or her own senses instead of relying on the instruments. For example, during gradual banking and descent, the pilot may sense the descent but not the bank and may attempt to gain altitude. The attempt to gain altitude actually increases the rate of the bank until the aircraft spirals out of control. This is called the graveyard spiral, but it is a rare occurrence.

Type III

Type III spatial disorientation occurs when the pilot is affected by the illusion of intense movement and is unable to reorient himself or herself. Usually when one person is affected this way, the other generally is not, and the correctly oriented person (the copilot) can steer to safety.

Since the John F. Kennedy, Jr, crash in 1999, the FAA has revised several rules regarding recognition of and training for spatial disorientation. Currently, even initial private pilot applicants must receive at least 3 hours of instrument training and be taught recovery techniques, and most instructors include the topic of spatial disorientation during recurrent training.

Visual Illusions

The visual system can give deceptive signals to the brain, especially during instrument meteorological conditions, such as when flying in clouds or low visibility. These illusions can lead to the misperceptions about location, altitude, distance away from other aircrafts or objects, the rate of speed as the aircraft closes in on other objects, and attitude. Flight crew members need to have a good understanding of assorted visual spatial disorientation illusions to prevent and recognize potentially disastrous situations. These situations are potentially lethal but extremely rare.

Third Spacing

Third spacing became a factor in aviation at the end of World War II. Third spacing is the loss of fluids from the intravascular space into the tissues. The German Luftwaffe encountered this phenomenon after they developed the first jet-powered aircraft. It became evident that the human body was not designed for rapid acceleration and deceleration or for high-speed turns. Third spacing became a factor during high-speed turns because of the addition of centrifugal force in concert with the acceleration or deceleration of the aircraft. These forces actually began to push fluids (primarily plasma) from the intravascular into the extravascular space, causing hypovolemia and potentiating hypoxia.

To counteract the effects of third spacing, the "g-suit" or military antishock trousers (MAST pants) were developed. The first g-suits developed actually were tightly laced suits that were similar to corsets. The aviators put these suits on and then tightened the laces to help counteract third spacing. The newer suits recognized high-gravitational-force maneuvers and automatically inflated to help prevent plasma from leaching into the extravascular space. Today, in most aircraft that will experience high-gravitational-force maneuvers, the flight team is equipped with pressure suits and is trained in techniques to prevent them from experiencing third spacing.

Flicker Vertigo

Flicker vertigo is defined by the Flight Safety Foundation as "an imbalance in brain cell activity caused by exposure to low-frequency flickering or flashing of a relative bright light." The effects of flicker vertigo may include nausea, vomiting, seizures, or fainting. These symptoms are usually mild and stop as soon as the source of the flickering is removed. This condition can be brought about by any bright light flickering at a frequency of 4 to 20 cycles per second (Hz). Helicopter personnel are most often affected by flicker vertigo when natural light or reflections of anticollision strobe lights are distorted by rotor blades. This same phenomenon is possible in fixed-wing propeller aircrafts. A rotating beacon can also cause flicker vertigo. If the propellers are the cause of flicker vertigo, simply changing the revolutions per minute of the engine can often eliminate the symptoms; if strobe lights while in clouds are the cause, they can be turned off until the aircraft is in clear skies.

Flicker vertigo can also manifest in patients who are susceptible to external noxious stimuli. For example, a patient with a severe head injury is being prepared for helicopter transport. As the medical crew loads the patient in to the aircraft, the pilot begins to increase the revolutions per minute of the engine. As the engine is warming up, the patient has a seizure. The medical crew instructs the pilot to delay departure so that they can manage the seizure. The patient is given an appropriate dose of an antiepileptic medication and the seizure stops. The medical crew advises the pilot that they are ready for departure. As the pilot speeds up the aircraft engine, the patient has another seizure. The flight crew quickly realizes that the patient's eyes are uncovered and the sunlight coming through the rotors is producing a strobelike effect, causing the seizure. The patient's eyes are covered and there is no more seizure activity during the flight.

Fuel Vapors

Flight crew members and patients are exposed to the noxious odors of fuel vapors. Most of the exposure occurs when the flight crew is loading or unloading a patient. The jet fuel odors can cause headaches and precipitate feelings of nausea, if prolonged. The flight crew is more susceptible to the effects than is the patient, because the crew undergoes more prolonged exposure.

Weather

Weather can be an additional stressor. For example, rapidly worsening conditions or inadvertent flight into conditions that require the use of instrument flight rules (IFRs) cause stress. Transport programs that operate by visual flight rules (VFRs) fly only in conditions that meet a visibility minimum of 3 miles and appropriate clearance from clouds. Transport programs rated for IFRs can fly in all but the worst of weather. The vast majority of rotor-wing programs operate under VFRs only because the pilots often must land the aircraft at unimproved sites (sites that are not paved). Pilots undergo extensive training to quickly handle changing weather conditions.

Anxiety

Anxiety has a pivotal role in flight operations but is rarely addressed or discussed; it is often assumed to have been covered in initial crew training. Several factors can cause anxiety. Numerous studies have shown catecholamine release in flight crew members and patients in rotor-wing operations. Even among experienced flight crew members, there is still a significant release of adrenaline, which is a catecholamine. This release of catecholamines can be advantageous to performance if it occurs in small quantities but detrimental if the release is in large quantities.

The experience and confidence of flight crew members helps determine the amount of adrenaline released. Critical care flight medicine is very different from critical care medicine within the confines of

a hospital. First, providers must realize that the patient always comes second. The safety of the flight crew and the operations of the aircraft always take precedence. Especially during takeoffs and landings, the pilot might require the assistance of medical crew members, and they must divert their attention from the patient. Second, medical flight crew members must not only learn to deal with a wide range of injuries and illnesses in critical care patients, but also must become intimately familiar with the safe operations of the aircraft and how to function as integral members of the aircrew. An excellent critical care practitioner may quickly be overwhelmed by the additional challenges of the flight environment.

There is an exceptionally steep learning curve for new flight nurses and paramedics. They must be adept at treating patients in the most critical of conditions—traumatic and medical—and ranging in age from birth to old age. Initially, all new flight crew members can be expected to have a high level of anxiety until they have gained experience transporting patients with a range of conditions during different types of flights.

Another stressor that can lead to anxiety is the confines of the aircraft cabin. Especially in rotor-wing aircraft, cabins are very small and usually filled with equipment and crew **Figure 4-17**. Even people who do not consider themselves claustrophobic may feel as though they are in a space that is simply too small. Flight crew members are trained in numerous advanced life-saving techniques but may quickly become frustrated when they realize that the confines of the aircraft prevent them from being able to use all of the needed skills.

Anxiety can occur in patients as well as the crew. Patients who express concern about the tight confines of the aircraft may be pretreated with antianxiolitics. Administration is based on individual protocols and patient presentation.

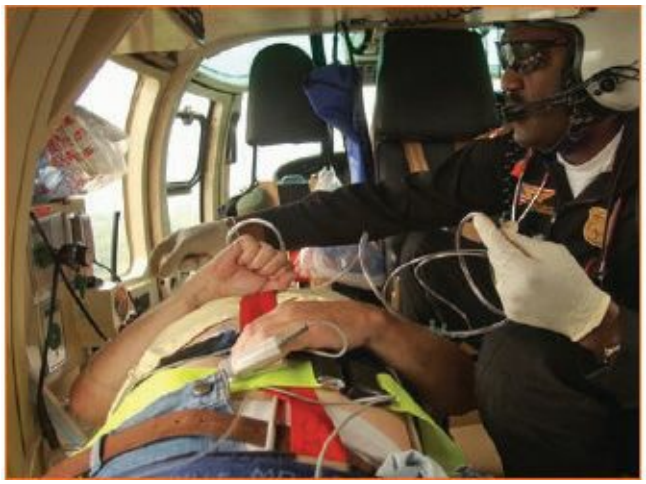


Figure 4-17 Aircraft cabins are very small, which can lead to anxiety on the part of patients and CCTPs.

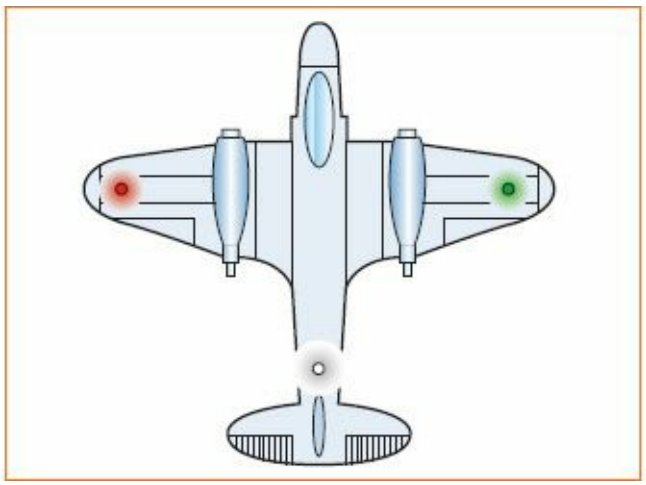


Figure 4-18 An aircraft's lights are set up in a standard format that makes it possible to determine the aircraft's direction of flight.

Night Flying

Crew members must be vigilant in assisting the pilot's scan for other aircraft when able, at night and during the day. Fortunately, due to lighting, aircraft are often easier to spot at night. Lights on airplanes indicate the direction of flight: from the seat of the aircraft being flown, the green light is on the right wing, the red light is on the left wing, and a white light is displayed to the rear. Because the position of lighting on all aircraft is standard, it is easy to determine the direction of flight at night **Figure 4-18**. For example, if a pilot in an aircraft sees another aircraft with a red light on the left, a green light on the right, and a white light in the middle, he or she knows that the aircraft is moving away. A crew member who identifies aircraft traffic on a potential converging course should notify the pilot at once.

Crew members should reduce use of white light that may affect the pilot's night vision. Red lighting inside the aircraft is preferred. The pilot and crew have various advantages and disadvantages. The crew has no outside visibility. The pilot has the following disadvantages:

- Limited field of vision (reduced 40% due to night-vision goggles, if used)
- Added weight stress from helmet
- Loss of depth perception
- Monochromatic vision
- A reduced sense of speed

Factors Affecting Tolerance of the Physiologic Stressors of Flight

The primary human factors affecting tolerance of the stressors of flight can be remembered by the mnemonic IM SAFE, which stands for Illness, Medication, Stress, Alcohol, Fatigue, and Emotion. In flight medicine, there are two "types" of altitude: physical altitude, which is represented in feet above ground, and physiologic altitude, which is the altitude *perceived* by the body. Although incapacitation to altitude does not begin to manifest until 10,000′, the body may perceive being at 14,000′ because of human factors.

Illness

Many illnesses, even a common cold, can significantly impair a crew member's performance. Nasal congestion can lead to severe headaches, vertigo, or nausea during pressure changes, such as during a rapid descent. All flight crew members should be cleared to fly by a physician during any period of illness.

Medications

The use of medications affects the tolerance of hypoxia. Over-the-counter (OTC) medications, although frequently considered benign, can result in incapacitation when their effects are combined with the effects of hypoxia. In addition, an underlying problem might go untreated because OTC medications can mask the problem. All flight crew members should follow approved lists for OTC medications before they fly or consult with a flight surgeon or a physician knowledgeable in flight medicine, such as an aviation medical examiner, before taking any medications when they are scheduled to work.

The FAA has a list of approved prescription and OTC medications that flight crew members are permitted to use. The FAA states in FAR part 61 that pilots assume responsibility for all medications that

they take and that if there is any doubt about the effect of the medication on the ability to fly, they must consult with a flight surgeon before flying. All flight crew members should be strongly cautioned against taking a medication for the first time when they are working because they are unaware of how the body will react to the medication.

Stimulants such as caffeine are frequently used and have a high potential for abuse. Caffeine can cause tachycardia, hypertension, increased urine production, increased excretion, and dehydration. Intake of caffeine greater than 250 mg (about two and a half cups of coffee) can cause problems in most of the population. Caffeine is also a strong diuretic: 1 cup of coffee can result in the elimination of 2 cups of water through urination. Tea has an additional diuretic (theophylline) that can cause an even greater loss of water by urination. Stimulants can also cause insomnia, tremors, indigestion, and nervousness.

Stress

Most pilots and flight crew cannot simply "leave stress on the ground." Everyday stresses such as work, financial, and family issues can affect performance negatively and can lead to distraction and poor judgment. If crew members are experiencing high levels of stress, they should be encouraged to discuss this with members of management at their program. High levels of stress can cause inattentiveness and may have adverse effects on performance, including catastrophic effects in the aviation environment. If crew members recognize high levels of stress in their coworkers, they should take action to ensure the safety and effective operation of the program.

Alcohol

Alcohol can be a toxin in the body and can result in histotoxic hypoxia, inhibiting the use of available oxygen by hemoglobin and delaying metabolism at the cellular level. Research indicates that the intake of 1 oz of alcohol is equivalent to 2,000′ of physiologic altitude. Simply drinking two beers can add between 4,000′ and 8,000′ of physiologic altitude. Alcohol works by depressing the central nervous system and inhibiting judgment and coordination, further amplifying the effects of altitude. Alcohol is also a potent diuretic and can lead to dehydration in the early stages of drinking. As the blood alcohol level increases nearing intoxication, the kidneys begin to produce less urine, actually leading to significant fluid retention. Some studies indicate that part of the reason for hangovers is the significant amount of dehydration followed by rapid fluid retention. FAR part 91 clearly addresses the topic of alcohol. It reads as follows:

a. No person may act or attempt to act as a crewmember of a civil aircraft—

(1) Within 8 hours after the consumption of any alcoholic beverage;

(2) While under the influence of alcohol;

(3) While using any drug that affects the person's faculties in any way contrary to safety; or

(4) While having an alcohol concentration of 0.04 or greater in a blood or breath specimen. Alcohol concentration means grams of alcohol per deciliter of blood or grams of alcohol per 210 liters of breath.

Most aviation companies have a 12-hour "bottle to throttle" policy; however, all policies are underscored by the caveat that people who feel under the influence of alcohol, regardless of the time from the last drink, should not fly. FAR parts 91 and 135 also state that all flight crew members are subject to random drug and alcohol tests at any time while on duty.

Regardless of maintaining 8 or 12 hours between drinking alcohol and flying, another significant hazard is the hangover, or postalcohol impairment, associated with drinking. The symptoms of hangovers include tremors, thirst, nausea and vomiting, diarrhea, sweating, heartburn, dizziness, and headaches.

Physical and mental performance are still markedly reduced 14 to 24 hours after the blood alcohol level has returned to zero. One of the most dangerous problems during the hangover phase is the propensity for hypoglycemia and significant fatigue. Despite the frequent claims made about OTC medications, there is no simple remedy for hangovers. Coffee, cold showers, and breathing 100% oxygen will not speed up the metabolism of alcohol.

Fatigue

Exhaustion and fatigue have an important role in one's ability to tolerate the effects of hypoxia. Flight crew members who provide air medical transport must work all hours. Shift work causes a tremendous disruption in the circadian rhythm, making it difficult for crew members to regulate sleep at work and at home. Shift work can lead to crew members starting their shifts exhausted, predisposing them to the effects of hypoxia. Many air medical crew members also work second jobs during their off time and must use caution to allow for plenty of rest before the start of their flight shifts. Exhaustion can lead to judgment errors, narrowed attention, uncharacteristic behavior, and falling asleep at work. Crew members who find that they need to use pharmacological sleeping aids need to first have the medication approved by a flight surgeon. Frequent use of sleeping aids should be considered a flight safety issue and discussed in depth with a flight surgeon. The constant vibration that aircrew members experience during flight also leads to fatigue.

Emotion

Certain emotionally upsetting events, such as major arguments, a death in the family, and divorce, can significantly impair pilot and crew performance. Any pilot or crew member who experiences an intense emotional event should not fly until satisfactory resolution occurs.

Tobacco

An aviator who smokes risks the effects of hypemic hypoxia, because carbon monoxide is 50 to 300 times more attracted to hemoglobin than oxygen. Research studies have found that smoking three cigarettes in rapid succession or smoking 20 to 30 cigarettes in a 24-hour period can saturate 10% of the hemoglobin in the body. At sea level, a regular smoker has a starting physiologic altitude of 3,000′ to 8,000′. Smoking also affects night vision; a regular smoker has already lost 20% of the night vision, even at sea level.

Hypoglycemia

A nutritious diet allows flight crew members to be much more tolerant to the effects of hypoxia. Poor diet and low blood glucose levels can cause nausea, headache, dizziness, shakiness, nervousness, and judgment errors. Also, not eating or eating foods high in sugar and fat or cooked in grease can precipitate the effects of motion sickness. A good way to prevent the effects of motion sickness is to eat several small meals or regularly snack on healthy foods during the shift. A lack of food in the stomach can cause feelings of nausea and exacerbate the feelings of motion sickness. It is a good idea to keep healthy snack foods such as trail mix or granola bars on the aircraft for situations when eating a meal is not possible.

It is important for flight team members to maintain a healthy weight. Obesity can cause several problems in the flight environment and can be detrimental to the overall safety and performance of the overweight flight crew member. In addition, most rotor-wing flight programs have a weight limit for crew members. A flight crew member taking diet supplements must first have the supplements approved by a flight surgeon.

Additional Stressors

In addition to the aforementioned stressors, there are several others. Age is an important factor; as the body ages, its ability to compensate to stress is diminished. Good physical shape is also important for flight crew members. Physical exertion during flight significantly lowers the altitude at which evolved gas disorders occur. Physical exertion increases oxygen demand, increasing the risk for hypoxia. Maintaining good physical conditioning will help to increase this threshold.

Dysbarism and Evolved Gas Disorders

Various disorders are directly related to altitude. Barotrauma can result from gases expanding and contracting in the body, which causes pain, usually in the digestive tract, sinuses, teeth, middle ear, or lungs. Some illnesses, such as decompression sickness, are not fully understood. Other illnesses, such as **dysbarism**, are directly related to the effects of altitude as described by the various gas laws. A dysbarism is a syndrome resulting from a difference between the barometric pressure and the pressure of gases within the body. As gases expand at altitude, they can cause pain in closed cavities.

Barotitis Media

Barotitis media affects the middle ear and is one of the most common trapped-gas problems. A flight team member who flies with a head cold can experience substantial pain. Barotitis media results from failure of the middle ear space to equalize pressures when going from low to high atmospheric pressure. Pressure in the middle ear becomes increasingly negative and a partial vacuum is created. As the pressure increases, the tympanic membrane is depressed inward and becomes inflamed and a petechial hemorrhage develops. Blood and tissue fluids are drawn into the middle ear cavity. The eardrum may rupture if the pressure does not equalize.

As the barometric pressure in the aircraft cabin decreases during ascent, the air trapped in the middle ear begins to expand, pushing the eustachian tube open. Air escapes through the nasal passages, and the pressure is equalized **Figure 4-19**.

Normally, the eustachian tubes can be opened by swallowing, yawning, tensing the muscles in the throat, and pinching the nose and attempting to blow through the nostrils. On ascent, expanding trapped air usually escapes easily, with the eardrums equalizing pressure, which is usually felt as “popping” in the ears. On descent, it becomes much more difficult for the negative pressure inside the middle ear to equalize. As the negative pressure continues to build, loss of hearing and pain are noted. Any type of respiratory infection can make equalization in the eustachian tube difficult or impossible. If the eustachian tubes are unable to equalize pressure on descent, profound pain may be felt, along with the possible occurrence of hemorrhage, indicating that the eardrum is about to burst or has already ruptured. To relieve the symptoms, the pilot should ascend until the pain is relieved and equalized, then take a very slow descent so the ears can slowly equalize.

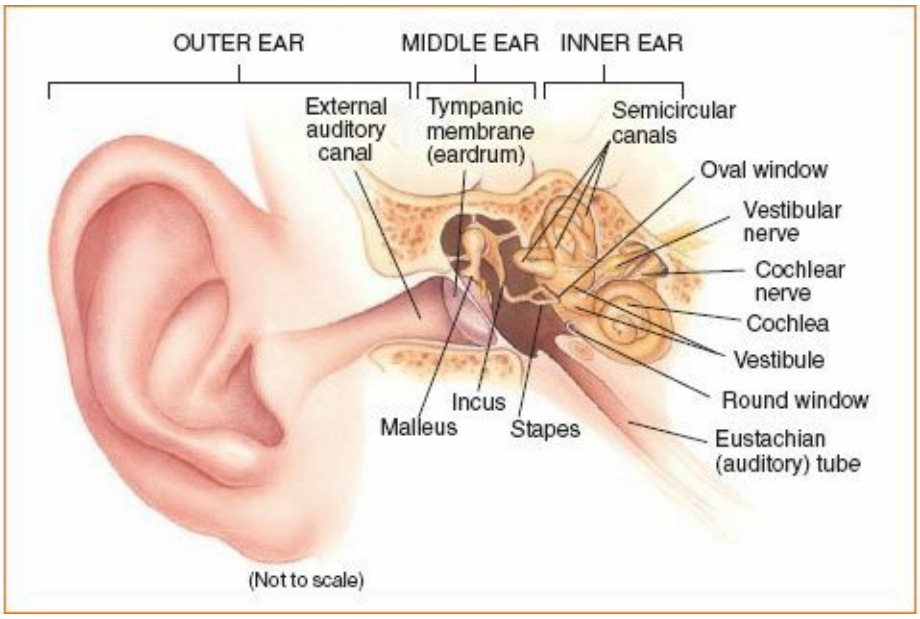


Figure 4-19 The inner ear, including the vestibular system. The otolith organs within the vestibular system are responsible for sensing equilibrium.

Decompression Sickness

Although **decompression sickness** is not the most frequent dysbarism, it is the most commonly known. Decompression sickness is explained by Henry's law and occurs by the formation of inert nitrogen gas bubbles at one or more locations in the body. The manifestation of symptoms depends on the location in the body in which the nitrogen bubbles form.

Normally, the tissue and fluid of the body contain from 1 to 1.5 L of dissolved nitrogen, depending on the barometric pressure of the atmosphere. As altitude decreases, nitrogen leaves the body in an attempt to reestablish equilibrium. Normally, the excess nitrogen diffuses into the capillaries in solution and is eliminated through venous circulation. If a human body is subjected to a rapid decrease in atmospheric pressure, the capillaries become supersaturated and nitrogen begins to leave as a gas instead of in a solution. Nitrogen bubbles begin forming in the tissue and in the blood. Fat is able to dissolve nitrogen five to six times more readily than blood, so tissues having the highest fat content are more likely to become saturated with nitrogen. Decompression sickness can cause circulation problems and, in severe cases, even death because of the potential for nitrogen bubbles in the arterial circulation causing an arterial gas embolism. Symptoms of decompression sickness are listed in **Table 4-6**.

TABLE 4-6 Symptoms of Decompression Sickness
Shortness of breath
Chest pain
Cough
Joint pain
Numbness and tingling of an arm or leg
Partial paralysis
Loss of speech
Loss of hearing

Vertigo
Visual disturbances (blind spots or the sensation of flashing or flickering lights)
Rashes
Itching of the skin

Summary

CCTPs must have a thorough knowledge of flight physiology, not only for their patients, but also for themselves and their fellow flight team members. Even if CCTPs are ground-based, they must still have a strong knowledge of the implications of flight because a large number of their patients will be prepared for flight or be received from a flight. Because the majority of patients that CCTPs will encounter have comorbid conditions or have multiple traumatic injuries, the effects of altitude will be far more pronounced. The most important aspect of flight medicine is safety. A thorough and comprehensive understanding of the implications of flight will help to keep the flight team members and their patients and passengers safe during the duration of the flight.

Case Study

YOU AND YOUR PARTNER ARE WORKING FOR A SMALL, RURAL AIR MEDICAL TRANSPORT SERVICE located in southeastern West Virginia that operates an American Eurocopter EC-135. The terrain that you are accustomed to is extremely mountainous with elevations more than 5,000′ above sea level. Owing to the geographic layout of your service area, many of the flight legs are completed in mountainous terrain with limited signs of human population.

It is 5:00 PM, and you are about half way through your 24-hour shift when you are dispatched to a small community hospital 45 nautical miles from your base. The requesting facility is asking your team to transport a patient via rotor-wing aircraft to a university hospital that is 2 hours away by ground and 25 minutes away by helicopter. Your pilot-in-command checks the local weather, and the decision is made to accept the flight. Once your pilot-in-command calls your operations center to confirm that you can accept the flight, you head to the aircraft and prepare for departure.

As soon as cruise altitude and speed are reached, your communications center informs you that your patient is a 64-year-old man with respiratory failure because of bilateral pneumonia and exacerbation of COPD. The patient was admitted to the hospital 2 days ago with acute shortness of breath and required immediate ventilatory support, including endotracheal intubation. The patient's condition has been stable during the hospital stay, but the family is requesting transport to the university hospital for a second opinion. The patient is receiving a propofol infusion for sedation, normal saline, and ceftriaxone. The patient is being mechanically ventilated with the following ventilator settings: tidal volume, 750 mL; synchronized intermittent mandatory ventilation rate, 12 breaths/min; and F_{IO_2}, 70%.

When you reach the patient in the outlying hospital intensive care unit, you are met by the staff nurse who provides you with a more detailed patient report. During this report, the nurse tells you that the local pulmonologist has been weaning the patient's ventilator settings with the goal of extubation. The results of recent laboratory studies, including blood gases, are reviewed by you and the staff nurse. The most recent set of vital signs are as follows: blood pressure, 124/64 mm Hg; pulse rate, 84 beats/min; respiratory rate, 16 breaths/min (patient is breathing 4 breaths/min on his own); and pulse oximetry, 94%. The nurse tells you that a copy of the patient's chest film and medical chart are in a folder for you to review and asks that you take them to the accepting hospital. Once the patient assessment has been completed and the

medications are transferred to your transport pumps, you secure the patient to your stretcher and prepare him for transport. You and your partner are concerned about his respiratory status owing to the bilateral pneumonia and are worried that he may not tolerate your transport ventilator as well as he did the hospital ventilator. You and your partner decide to perform a trial with the patient on your ventilator for at least 10 minutes before departing for your aircraft. The trial goes smoothly, and you proceed toward the aircraft.

During the initial phase of your flight to the university hospital, the patient remains adequately sedated and his vital signs remain unchanged. About 12 minutes into the flight, you notice that the patient's oxygen saturation readings are beginning to drop into the low 90% levels. After checking the patient and troubleshooting the ventilator and oxygen connections, you determine that ventilator settings are appropriate. You contact online medical command, provide a brief report, and ask permission to adjust the F_{IO_2}. Orders are received to increase the F_{IO_2} to 100% to maintain pulse oximetry readings of at least 97%. Within minutes of making this change, the pulse oximetry readings return to normal, at 99%. The remainder of the flight is unremarkable, and a full patient report is given to the nurse in the intensive care unit on your arrival at the university hospital.

1. What is the most likely cause of your patient's hypoxia that was experienced during flight?
2. What type of changes would you expect to see, as a result of the barometric pressure changes, with the IV solutions that you are infusing through the transport pump?
3. What are some common medical conditions that are typically worsened by altitude changes?

Analysis

It is important to first understand the critical state of a patient who has been diagnosed with bilateral pneumonia and has an underlying lung disease. Patients with these conditions often experience desaturation rather quickly with any change in position, altitude, or ventilator settings. In this particular case, flying at 6,000′ above sea level over very mountainous terrain was required to get to the university hospital. In situations like this one, it is very important to understand Henry's law, which states that the F_{IO_2} should be adjusted upward proportionate to the change in barometric pressure in the helicopter cabin. Because the barometric pressure in the hospital and the barometric pressure in the air are rarely available to CCTPs in advance, awareness is essential that increasing the F_{IO_2} en route may be necessary as altitude increases.

The second question asks what type of changes would be expected as a result of barometric changes when IV solutions are infused through the transport pump. The answer is none! As long as the infusions are being maintained on a pump, the barometric changes will not affect the flow rates. However, if the patient is receiving an infusion via gravity, the infusion rate will increase as the barometric pressure decreases, which occurs with increasing altitudes.

Pneumonia is one of the most common medical conditions that might require additional oxygen therapy to prevent worsening hypoxia. Other medical conditions that are adversely affected by altitude changes are COPD, asthma, coronary artery disease, pneumothorax, shock, and blood loss. Close attention should be given to patients with these conditions, and medical consultation should be obtained before departing the referring facility to discuss adaptive therapies that might be needed throughout the transport.

Prep Kit

Ready for Review

- CCTPs must have a thorough understanding of flight physiology and the implications for patients and for themselves because the medical environment during flight is complex and extremely dynamic and the changes in barometric pressures and the forces experienced in flight can have a significant impact on disease pathophysiology.
- There are several distinct layers of the atmosphere. The first layer is called the troposphere, followed by the tropopause (which together constitute the lower atmosphere). Above the tropopause are the stratosphere, stratopause, mesosphere, and thermosphere, respectively. Critical care transports would not usually fly above the tropopause.
- The atmosphere is divided into three distinct zones that directly correlate with a human's response to hypoxia. These zones are the physiologic zone (which extends from sea level to 10,000′ and contains the oxygen and has the barometric pressure needed for a healthy person to live), the physiologically deficient zone (from 10,000′ to 50,000′, where the barometric pressure begins to decrease and protective equipment, supplemental oxygen, and pressurized aircraft are necessary), and the space equivalent zone (from 50,000′ to 120 miles, where pressure suits and sealed cabins are required).
- Barometric pressure, also called atmospheric pressure, is a direct result of the weight of air and is also related to air density, air temperature, height above the earth's surface, and weather.
- CCTPs need to have a thorough knowledge of the seven gas laws, Boyle's law, Charles' law, Dalton's law, Fick's law, Gay-Lussac's law, Graham's law, and Henry's law, that have integral roles in aviation medicine and flight physiology.
- Boyle's law states that as altitude increases, atmospheric pressure decreases, and the gases inside the body expand. Boyle's law also applies to the expansion of any trapped gas within the body, in such places as the chest, skull, middle ear, sinus, stomach, and intestines, and increasing tension and pressure. Medical equipment and supplies can also be sensitive to an increase or decrease in barometric pressure, including IV fluids, nasogastric and orogastric tubes, and colostomy bags.
- Charles' law states that the higher the altitude, the lower the pressure and the colder it becomes, which puts patients and crew members at risk for hypothermia.
- Dalton's law states that the total pressure of a gas mixture is the sum of the pressures of the individual gases in the mixture, and at higher altitudes, oxygen molecules are less densely distributed, which means that at higher altitudes, an insufficient number of oxygen molecules can lead to hypoxia.
- Fick's law states that the rate of diffusion is affected by atmospheric pressures, the surface area, and the thickness of the membrane. This is the primary gas law for diffusion of oxygen across the alveolar membrane, which means that a patient with COPD and pneumonia will have decreased gas exchange.
- Gay-Lussac's law states that as pressure decreases, temperature decreases, and, therefore, it is important to keep patients warm.
- Graham's law states that if an existing permeable or semipermeable membrane separates regions of unequal pressure, the gas at higher pressure will diffuse through the membrane into the region of lower pressure, meaning that carbon dioxide will diffuse more readily into the bloodstream than oxygen.
- Henry's law states that as the pressure of gas over a liquid decreases, the amount of gas dissolved in the liquid will also decrease, meaning that as the barometric pressure decreases, nitrogen, which normally saturates the tissues, will form gas bubbles that travel to the lungs.
- A main hazard in aviation is hypoxia or oxygen deprivation. In addition to the fact that hypoxia mimics fatigue and hypoglycemia, making it difficult to recognize, one of the earliest effects of hypoxia is impaired judgment, which also limits an aviator's ability to recognize it.

- The length of time a person can function with an inadequate level of oxygen is called the effective performance time, with the time of useful consciousness defined as the period between an individual's sudden deprivation of oxygen at a given altitude and the onset of physical or mental impairment to the point at which deliberate function is lost.
- Hypoxic hypoxia, which can be due to increased altitude, results from a lack of oxygen or an inability of oxygen to diffuse into the bloodstream, symptoms of which begin to manifest at heights above 5,000′.
- Histotoxic hypoxia is the cell's inability to adequately use oxygen, even though there is plenty of oxygen available, because the tissues are not able to accept it or because the oxygen cannot offload from the hemoglobin.
- Stagnant hypoxia is the failure to transport oxygenated blood, and in flight medicine, the reduction in the flow of blood can result from venous pooling during acceleration maneuvers. Stagnant hypoxia, with blood pooling in the lower extremities of patients and crew members sitting in aircraft for extended periods, increases the risk of deep venous thrombosis.
- Hypemic hypoxia, also known as anemic hypoxia, occurs when there is a reduction in the ability of the blood to carry oxygen to the tissues due to a lack of hemoglobin or red blood cells.
- The symptoms of hypoxia are divided into stages (the indifferent stage, the compensatory stage, the disturbance stage, and the critical stage), which are directly related to the altitude, approximate barometric pressure, and the oxygen saturation of the blood.
- The indifferent stage is experienced between sea level and 10,000′ but can occur at altitudes as low as 5,000′. At this stage, night vision begins to deteriorate; electrocar-diographic changes, including tachycardia, are common; a slight increase in alveolar ventilation may be noted; and the oxygen saturation varies from 98% to 87%.
- In the compensatory stage, which occurs between 10,000′ and 15,000′, the body is able to provide short-term physiologic compensation against the effects of hypoxia, such that the respiratory rate and depth may increase and the cardiac output increases. At this stage, hemoglobin saturation varies from 87% to 80%.
- In the disturbance stage, which occurs between 15,000′ and 20,000′, the physiologic compensatory mechanisms are no longer able to provide adequate oxygenation to the tissue, and respirations, senses, mental processes, personality manifestations, and psychomotor functions may all be affected by hypoxia. At this stage, hemoglobin saturation varies from 79% to 70%.
- The critical stage, occurring above 20,000′, is the last stage of hypoxia. Within 3 to 5 minutes of hypo-oxygenation, judgment and coordination deteriorate, and mental confusion is quickly followed by incapacitation, unconsciousness, and death, if the hypoxia is not corrected. At this stage, hemoglobin saturation is less than 65%.
- Recovery from hypoxia is rapid, and full mental abilities can be recovered within 15 seconds after receiving high-flow oxygen. Prevention and avoidance of hypoxia are key to safety, but if hypoxia is detected, immediate use of supplemental oxygen and descent to below 10,000′ are necessary.
- Pressurizing an aircraft, which is accomplished by increasing the barometric pressure inside the aircraft above the ambient pressure outside, is the most effective method of protecting people from the physiologic effects of reduced barometric pressure. The two primary methods used for pressurizing an aircraft are the isobaric system (which is most frequently used and maintains the aircraft cabin at a constant pressure between 5,000′ and 80,000′) and the differential control method (which is used primarily by the military and does not maintain a constant cabin pressure, but varies the pressure with altitude; supplemental oxygen and pressure suits must be available and used at certain altitudes).

- Aircraft decompression at altitude is categorized as slow (as when a small leak develops) or rapid (a dramatic event that occurs with a loud explosion). As a result of decompression, the occupants of the aircraft are exposed to the dangers of hypoxia, decompression sickness, gastrointestinal expansion, and hypothermia.
- The four primary forces that act on an aircraft in flight are lift, thrust, weight (gravity), and drag, with lift counteracting gravity and thrust counteracting drag. In flight, thrust equals drag and lift equals weight, but to take off, a greater amount of thrust and lift are needed, whereas a greater amount of drag and weight are required to land.
- There are many stressors that can cause hypoxia in crew members, and if the proper precautions are not taken, CCTPs may begin to experience symptoms of hypoxia such as confusion, fatigue, visual impairment, headache, nausea, and euphoria.
- A decrease in the PO_2 can quickly cause hypoxia in flight crew members and passengers.
- The greatest change in barometric pressure occurs from sea level to 5,000′, and, therefore, problems associated with pressure must be considered, even in nonpressurized aircraft that are not flying at high altitudes at which supplemental oxygen or cabin pressurization is required.
- Flight crew members are subjected to a variety of extreme temperature changes, ranging from the very cold to the very hot as a result of changes in air temperature and altitude, which, therefore, affect the metabolic rate and the oxygen demands on the body, and at the same time make the body less tolerant of the effects of hypoxia. Temperature changes can cause the effects of hypoxia to be seen at lower altitudes than would normally be expected. Flight crews must be aware of the potential for heat stress and also the greenhouse effect, in which the temperature in the cockpit or cabin of a small aircraft is raised by 50°F (10°C) to 59°F (15°C) simply by radiant solar heat.
- Vibration is noted in all helicopters, in most turboprop aircraft, and, minimally, in jet-engine aircraft. As the body becomes fatigued with prolonged exposure to vibration, the person becomes more susceptible to pain, decreased vision, and fatigue. Preventive measures to decrease vibration include not leaning against the airframe and providing extra padding for the patient.
- Humidity is the degree of moisture in the air and is relative to temperature. Humidity is decreased during a long flight, and the dry air in the aircraft can cause cracked mucous membranes, chapped lips, and sore throats and can lead to dehydration. Crew members should increase fluid intake, and all patients should be properly hydrated.
- Noise in the cabin of an aircraft can make it difficult for patients and care providers to communicate and for CCTPs to assess heart and lung sounds. Monitoring errors and malfunctions can be attributed to the noise and vibrations associated with air medical transport; however, pulse oximetry and hemodynamic monitoring are two technological advances that can lessen these negative effects and help identify complications.
- Most physiologic problems encountered in the flight environment can cause significant fatigue and, coupled with the lack of restful sleep, quickly become cumulative. Jet lag in the fixed-wing environment becomes another fatigue factor. While caring for critically ill or injured patients, CCTPs should immediately request crew rest, typically 4 hours, the minute they feel fatigued.
- Gravitational forces can have a significant impact on the human body, including the feeling of being pushed down in the seat or feeling weightless, depending on whether the aircraft is accelerating or decelerating. As the gravitational force increases, other complications are difficulty breathing, hypoxia, loss of vision, loss of consciousness, petechiae, rashes, bruising, seizures, amnesia, confusion, and cardiac arrhythmias.

- Spatial disorientation is the state of having an incorrect understanding of the body's position in relation to earth and can cause significant nausea and vomiting. The three components to maintaining spatial orientation on the ground are effective perception, integration, and interpretation of visual, vestibular (from the organs of equilibrium in the inner ear), and proprioceptive (from the skin, muscles, tendons, and joints) sensory information.
- Third spacing is the loss of fluids from the intravascular space into the tissues, causing hypovolemia and potentially hypoxia, as a result of high-speed turns in conjunction with acceleration or deceleration of the aircraft.
- Flicker vertigo is defined as an imbalance in brain cell activity caused by exposure to low-frequency flickering or flashing or a relative bright light that occurs when sunlight flickers through the rotor blades or propeller, at night by the anticollision lights reflecting off the clouds, or by rotating beacons or strobe lights, resulting in nausea, vomiting, seizures, or unconsciousness.
- Exposure to the noxious odor of fuel vapors occurs mostly when the flight crew is loading or unloading a patient and can cause headaches and nausea if prolonged.
- Weather, reduced visibility, and night flying can be additional flight stressors.
- Additional factors affecting tolerance of the stressors of flight can be remembered by the mnemonic IM SAFE, which stands for Illness, Medication, Stress, Alcohol, Fatigue, and Emotion. Although incapacitation to altitude does not begin to manifest until 10,000′, the body may perceive being at 14,000′ because of these factors.
- Age and physical condition are additional factors related to one's ability to tolerate the effects of hypoxia.
- Illnesses such as dysbarism (a condition that results from the difference between the barometric pressure and the pressure of gases within the body) are directly related to the effects of altitude as described by various gas laws. As gases expand at altitude, they can cause pain in closed cavities. Barotrauma can result from gases expanding and contracting in the body. This expansion causes pain, usually in the digestive tract, sinuses, teeth, middle ear, or lungs.
- Barotitis media results from the failure of the middle ear space to equalize pressures when going from low to high atmospheric pressure. As the pressure increases, the tympanic membrane is depressed inward and becomes inflamed and petechial hemorrhage develops. Blood and tissue fluids are then drawn into the middle ear cavity, and the eardrum may rupture if the pressure does not equalize.
- Although decompression sickness is not the most frequent dysbarism, it is the most commonly known.

Vital Vocabulary

atmosphere Gases that extend from the earth's surface to space; composed primarily of nitrogen, oxygen, argon, and trace gases.

barometric pressure The weight per unit area of all of the molecules of the gases above the point at which the measurement was taken.

barotitis media Inflammation and possible petechial hemorrhage in the middle ear and possible rupture of the eardrum that results from the failure of the middle ear space to ventilate when going from low to high atmospheric pressure; also known as ear block.

Boyle's law States that the volume of a gas is inversely proportional to the pressure to which it is subjected. Gases trapped in body cavities will expand with increases in altitude and will contract with

decreases in altitude.

Charles' law States that when pressure is constant, the volume of a gas is very nearly proportional to its absolute temperature. Thus, the volume is directly proportional to the temperature when it is expressed on an absolute scale where all other factors remain constant.

compensatory stage A stage of hypoxia in which the physiologic adjustments that occur in the respiratory and circulatory systems are adequate to provide defense against the effects of hypoxia. Factors such as environmental stress and prolonged exercise can potentiate certain effects of hypoxia.

critical stage The stage of acute hypoxia in which there is almost complete mental and physical incapacitation, resulting in rapid loss of consciousness, seizures, respiratory arrest, and death.

Dalton's law States that the total pressure of a gas mixture is the sum of the individual or partial pressures of the gases in the mixture; also referred to as the law of partial pressure.

decompression sickness A condition resulting from exposure to low barometric pressure, causing inert gases normally dissolved in body fluids and tissue to come out of physical solution and form bubbles.

disturbance stage A stage of hypoxia in which physiologic responses are inadequate to compensate for the oxygen deficiency, and hypoxia is evident.

drag The resistance of an airplane to forward motion, directly opposed to thrust.

dysbarism A condition resulting from the effects (excluding hypoxia) of a pressure differential between the ambient barometric pressure and the pressure of gases within the body.

effective performance time The amount of time an individual is able to perform useful duties in an environment of inadequate oxygen; also known as expected performance time.

Fick's law States that the net diffusion rate of a gas across a fluid membrane is proportional to the difference in partial pressure, proportional to the area of the membrane, and inversely proportional to the thickness of the membrane.

flicker vertigo An imbalance in brain cell activity caused by exposure to low-frequency flickering or flashing light. Light flickering from 4 to 20 times per second can precipitate reactions, including nausea, migraines, unconsciousness, and seizures.

flight surgeon A physician who specializes in flight medicine and has been trained extensively in various aspects of aviation and the effects of flight on the human body; also specializes in working with pilots and flight crew members.

Gay-Lussac's law States that the pressure of a gas when volume is maintained constant is directly proportional to the absolute temperature for a constant amount of gas. Simply stated, as pressure increases, volume increases.

Graham's law States that the rate at which gases diffuse is related inversely to the square root of their densities.

gravitational forces Force changes that occur with acceleration and deceleration.

Henry's law States that the amount of gas dissolved in solution is directly proportional to the pressure of the gas over the solution.

histotoxic hypoxia Hypoxia caused by the inability of the tissues to use oxygen, usually as a result of poisoning by toxins such as carbon monoxide and cyanide.

humidity The degree of moisture in the air, expressed as a percentage.

hypemic hypoxia Hypoxia caused by a decrease in the oxygen-carrying capacity of the blood due to a

reduced amount of hemoglobin in the blood or a reduced number of red blood cells; also known as anemic hypoxia.

hypoxia A state of oxygen deficiency in the body, which is sufficient to cause an impairment of function. Hypoxia is caused by the reduction in partial pressure of oxygen, inadequate oxygen transport, or the inability of the tissues to use oxygen.

hypoxic hypoxia Hypoxia caused by a decrease in the amount of oxygen in the blood due to a reduction in oxygen pressure in the lungs, a reduced gas exchange area, exposure to high altitude, or lung disease.

indifferent stage The stage of altitude hypoxia in which the body is able to compensate for the hypoxia induced by low barometric pressures.

isobaric pressurization system A system in which the aircraft cabin is pressurized and the cabin altitude is maintained at a constant pressure even as ambient pressure decreases. This type of pressurization is found in most cargo planes and passenger aircraft.

lift The upward force created by the wings moving through the air, which sustains the airplane in flight.

oxygen paradox A condition that results when a hypoxic person rapidly breathes in 100% oxygen; he or she may experience sudden dizziness, which is quickly resolved followed by complete restoration of function. The condition is possibly due to the sudden constriction of dilated arteries in the brain.

partial pressure of oxygen (arterial) (Pa_{O_2}) The amount of the total pressure in the blood contributed by oxygen; a value measured when analyzing arterial blood gas level.

physiologically deficient zone The zone that extends from 10,000′ to 50,000′. Noticeable physiologic deficits occur above 10,000′. A decrease in barometric pressure results in oxygen deficiency, causing hypoxic hypoxia. In this zone, the manifestation of trapped and evolved gases occurs. The use of pressurized aircraft and/or supplemental oxygen is necessary in this zone.

physiologic zone The zone that extends from sea level to 10,000′ and is the area of the atmosphere to which humans are well adapted. The barometric pressure is sufficient in this zone to facilitate adequate oxygenation. The changes in pressure encountered with rapid ascents or descents within this zone can produce ear or sinus trapped-gas problems.

pneumomediastinum The collection of air within the mediastinum (the space within the chest that contains the heart, major blood vessels, vagus nerve, trachea, and esophagus; located between the two lungs).

pneumoperitoneum The collection of air within the peritoneum (the membrane in the abdomen encasing the liver, spleen, diaphragm, stomach, and transverse colon).

proprioceptive Referring to information that comes from receptors located in the skin, muscles, tendons, and joints; this information helps a person know the position of his or her body.

rapid decompression Occurs when a large leak or hole develops in a pressurized aircraft; can result in hypoxia and injury to people inside the aircraft and catastrophic failure of the aircraft.

space equivalent zone The zone that begins at 50,000′. In this zone, 100% oxygen is not sufficient to prevent hypoxia without the use of a pressurized aircraft or suit. Unprotected personnel may experience boiling of body fluids at a level above 66,500′.

spatial disorientation A state characterized by an erroneous sense of one's position and motion relative to the earth's surface. This condition results from a person's inability to determine his or her position, altitude, and motion in relation to the surface of the earth or to a significant fixed object during flight.

stagnant hypoxia Hypoxia caused by a malfunction of the circulatory system resulting in a decrease in

blood flow.

third spacing A loss of fluids from the intravascular space into the tissues caused by an increase in intravascular pressures and/or increased permeability of the cell membranes. Physical stressors of flight such as temperature, vibration, and changes in gravitational force can cause or aggravate this condition.

thrust The force exerted by the aircraft engine, which pushes air backward with the objective of causing a reaction of the airplane in the forward direction.

time of useful consciousness The time between a person's sudden deprivation of oxygen at a given altitude to the point at which deliberate function is lost. With the loss of effective performance during flight, a person is no longer capable of taking proper corrective or protective actions.

tropopause The space between the troposphere and the stratosphere. It rises to 60,000′ at the equator owing to heated air masses that expand and sinks to about 30,000′ at the poles owing to contracting cold air masses.

troposphere A portion of the earth's atmosphere that extends from the surface of the earth to 5 to 10 miles high depending on the relation to the equator and the poles. This layer is characterized by the presence of water vapors, a constant decrease in temperature with increasing altitude, and large-scale vertical currents.

Type I spatial disorientation The pilot is unaware of becoming disoriented.

Type II spatial disorientation The pilot realizes that a problem exists but does not recognize it as disorientation.

Type III spatial disorientation A sudden incapacitating form of loss of positional awareness.

universal gas law States how a hypothetical gas should act if there are no variables affecting it, such as temperature or pressure. Also known as the ideal gas law.

vestibular Describes organs of equilibrium located in the inner ear.

weight The downward force due to the weight (gravity) of the airplane and its load; directly opposed to lift.

Bibliography

Air and Surface Transport Nurses Association. Holleran RS, ed. *Air and Surface Patient Transport: Principles and Practice.* 3rd ed. New York, NY: Mosby; 2003.

Better Hearing Institute. *Your Guide to Better Hearing.* Washington, DC; 1998.

Bolen RL Jr. Flight physiology. Duke *LifeNet.* 2004;12(2):4–5. Available at: http://trauma.dukehealth.org/wysiwyg/downloads/LifeNet_V12_2.pdf. Accessed November 20, 2006.

Dysbarisms and altitude sickness. Transport Canada Civil Aviation. Available at: http://www.tc.gc.ca/CivilAviation/Cam/tp13312-2/section2/dysbarisms.htm. Accessed January 6, 2007.

Dysbarisms and other selected health effects. US Department of Health; Occupational Safety and Health Administration. Available at: http://www.osha.gov/SLTC/commercialdiving/more.html. Accessed January 6, 2007.

The effects of space flight on the human vestibular system. NASA Exploration Systems, Mission Directorate Education Outreach. Available at:

http://weboflife.nasa.gov/learningResources/vestibularbrief.htm. Accessed February 7, 2007.

Egger AE. Earth's atmosphere. *VisionLearning*. 2003. Available at: http://www.visionlearning.com/library/module_viewer.php?mid=107&l=&c3. Accessed November 28, 2006.

Fischer P. High Altitude Respiratory Physiology. Available at: http://www.brooks.af.mil/af/files/fsguide/HTML/Chapter_02.html. Accessed November 26, 2006.

Fischer PW. United States Air Force Flight Surgeon's Guide. Available at: http://www.brooks.af.mil/af/files/fsguide/HTML/Chapter_02.html. Accessed December 19, 2006.

Flight Physiology. Available at: http://www.safetydata.com/manual/poeg18.htm. Accessed November 24, 2006.

Gases: Graham's laws of diffusion and effusion. Department of Chemistry, Texas A&M University. Available at: http://www.chem.tamu.edu/class/majors/tutorialnotefiles/graham.htm. Accessed November 26, 2006.

George VJ. Visual illusions. Indian Society of Aerospace Medicine Web. Available at: http://www.isam-india.org/essays/visual.shtml. Accessed February 7, 2007.

Inflatable trauma pants. Available at: http://everything2.com/index.pl?node_id=1345712. Accessed November 26, 2006.

Lee S. The effects of G-forces on humans. Electronic Aviation. October 13, 2004. Available at: http://www.electronicaviation.com/articles/General/142. Accessed February 2, 2007.

National Aeronautical and Space Administration. Available at: http://liftoff.msfc.nasa.gov/academy/space/atmosphere.html. Accessed November 10, 2006.

Oxygen. Mountain Flying. Available at: http://www.mountainflying.com/oxygen.htm. Accessed November 29, 2006.

Physiology of flight. US Marine Corps. Available at: http://www.iiimef.usmc.mil/medical/FMF/FMFE/FMFEref/fs_man/CHAPTER%201.html. Accessed November 26, 2006.

Reinhart RO. *Basic Flight Physiology*. New York, NY: TAB Books, a division of McGraw-Hill; 1992.

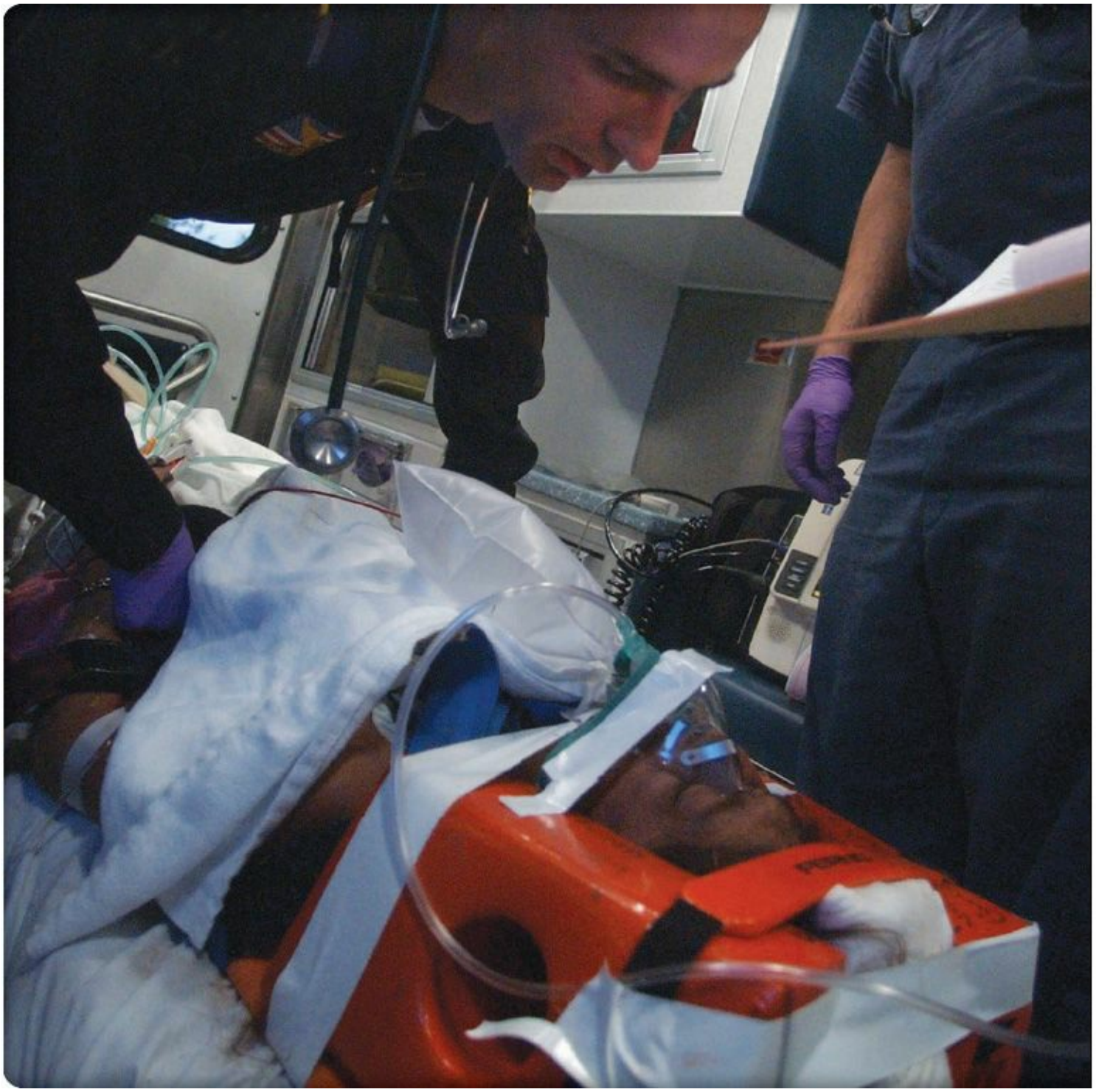
Sharing information for safer amusement ride thrills: G-force primer. Available at: http://www.saferparks.org/safety/dynamic_force/index.php. Accessed February 2, 2007.

South African Civil Aviation Authority. Disorientation. Available at: http://www.caa.co.za/resource%20center/av.%20medicine/docs/Disorientation.htm. Accessed January 6, 2007.

Spence C. Flicker vertigo: know it to avoid it. Available at: http://www.iflyamerica.org/flicker-vertigo.asp. Accessed January 6, 2007.

Voshell M. High acceleration and the human body. Available at: http://csel.eng.ohio-state.edu/voshell/gforce.pdf. Accessed February 3, 2007.

Wynbradt J. Spatial disorientation: confusion that kills. *Safety Advisor*. AOPA Online. Available at: http://flighttraining.aopa.org/student_pilot/maneuvers/pdfs/SA17_Spatial_Disorientation.pdf. Accessed November 21, 2006.



Patient Assessment

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Objectives

1. Understand the advantages and disadvantages of prehospital and in-hospital assessment models when used in the critical care transport setting (p 102).
2. Define differential diagnosis and how it is used in the critical care transport setting (p 102).
3. Compare assessment at a scene transport with assessment at an interfacility transport (p 103).
4. Describe the evaluation of assessment information and the process for assessment at a scene transport (p 103–104).
5. Discuss the considerations for packaging a patient at a scene transport (p 104).
6. Describe the evaluation of assessment information and the process for assessment at an interfacility transport (p 104).
7. Discuss the steps for packaging a patient at an interfacility transport (p 110).
8. List the components of a hospital transfer sheet (p 105–109).
9. Define normal and abnormal parameters of the critical care assessment (p 110–119).
10. Describe how to gather the necessary information for a critical care transport (p 103, 104, 105).
11. Describe and perform the assessments required for critical care transports, including the following:
    - General appearance
    - Cardiovascular assessment
    - Respiratory assessment
    - Neurologic assessment
    - Gastrointestinal assessment
    - Genitourinary assessment
    - Musculoskeletal assessment
    - Psychosocial and emotional assessment (p 110–119)
12. Discuss how to determine patient condition utilizing assessment skills (p 110–119).
13. Discuss transferring a patient to a receiving facility appropriately, including proper communication and documentation (p 120).
14. Understand how to interpret patient complaints, signs, and symptoms to determine patient condition and anticipate changes in condition (p 105, 110–119).

Introduction

Patient Assessment in the Critical Care Environment

Assessment of critical care patients presents unique challenges and, at times, dilemmas. During these

transports, patients with complex medical conditions are removed from the security of the intensive care unit, which may destabilize their condition.

Therefore, the need for a thorough understanding of the clinical scenario is a high priority for CCTPs, especially given that the time to obtain and absorb the data may be brief. CCTPs must, therefore, use sharp skills in clinical assessment. Interpersonal communications and diplomacy are also essential when relating to personnel at the sending and receiving hospitals and to patients. A balanced approach to assessment and treatment, applied in the light of modern critical care techniques, produces an effective blend of field and in-hospital principles.

As in all areas of health care practice, critical care intervention is based on the condition of the patient and response to therapy. For this reason, patient assessment is one of the most important processes for CCTPs to understand. Fortunately, nurses and paramedics are quite familiar with assessment processes through their initial training, their observation of other practitioners, independent reading, continuing education courses, and personal interactions and experiences with patients. Nevertheless, it is important to establish an assessment process that ensures the discovery and appropriate management of the patient's critical care needs to limit **morbidity** and **mortality** and improve patient outcomes.

Although we expect a patient in critical health status to have been assessed by prehospital care and hospital personnel in many cases, it is important to be familiar with the assessment process that established the initial care plan and also because critically ill patients change so quickly. Initial plans of care used in nursing and medicine are premised on a review of pertinent data, including assessments, laboratory and radio-logic studies, and other diagnostic tests, to arrive at one or more diagnoses. Paramedics use similar assessment skills and perform limited field diagnostic tests but often do not have the time or resources to achieve hemodynamic stability in a patient or the range of data needed to proceed any further than assembling a list of potential, or differential, diagnoses. When prehospital providers give initial care, they treat according to assessment findings and, using the limited data acquired, often formulate a **projected diagnosis**.

A useful tool mentioned throughout this book is the **differential diagnosis**. The differential diagnosis is a list of all possible diagnoses, aside from the projected diagnosis, that could be causing a patient's symptoms. The clinician, by using diagnostic testing and treatment, excludes or includes each differential diagnosis. In the critical care transport setting, it is useful for CCTPs to have an advanced understanding of pathophysiology and the full range of potential differential diagnoses for patients to assist in making treatment decisions and assessing the effects of treatments.

There are advantages and disadvantages to the paramedic and medical or nursing assessment models. Paramedics treat major problems as the symptoms are found, using a standardized assessment approach based on sequencing the most immediate threats to life and patient survival. The medical and nursing models of assessment are not always attuned to providing treatment during the assessment process. The wealth of information assembled using a medical or nursing model of assessment can be very beneficial to providers and patients by targeting treatment toward a known list of problems. Targeting treatment to underlying problems can be more effective than treating symptoms alone without knowing their causes. A combination of the paramedic and medical or nursing models of assessment is most ideal in the critical care transport environment.

Bridging Environments and Disciplines

The systems assessment presented later in this chapter comprises the detailed physical exam; however, if at any time the patient experiences a sudden deterioration in status, the CCTP should revert to the prehospital assessment and intervention strategies for short-term management (resuscitation).

The prehospital "find a life threat—fix a life threat" mindset is drastically different from the

problem-solving assessment more typically used in clinical practice. Although the prehospital assessment model works well in acute emergency care, it may not be the best choice for critical care. Fortunately, EMS practitioners need not reinvent the wheel. Critical care nursing and critical care medicine have for several decades developed and refined processes, techniques, and interventions unique to critical care patients that we can borrow and refine for use in the evolving field of critical care transport.

A critical care patient is usually being treated for problems involving one or more systems and frequently has interrelated, multisystem complications. CCTPs have for some time used a systems assessment approach for continuing patient evaluation, identifying subjective and objective criteria and performance indicators to measure physiologic status. This approach may be as simple as a neurologic assessment that notes a minor behavioral change or the more sophisticated measurement of intracranial pressure (ICP) with an ICP monitor.

Critical care is not solely driven by technology. Historically, the digital bells and whistles have received significant emphasis, but there has been a philosophical shift in the last decade, a return to bedside clinical care (that is, treating the patient, rather than the machine). The new approach involves looking at and listening to patients and families and making decisions and solving problems by balancing personal and clinical observations with technology. Astute assessment skills can provide data not given by machines or technology. Often, technology used in the hospital intensive care unit is unavailable in the field; therefore, excellent assessment skills are required in the transport setting. This chapter explores a systems approach using clinical observation and technological parameters for the continuing assessment of patients in critical condition.

Scene vs Interfacility Transport

Scene Transport

A CCTP called to an emergency scene will often need to interact with other providers caring for the patient, in some ways not unlike interactions needed on an interfacility transport. It is important to acknowledge and recognize the care provided before CCTP arrival and expeditiously assess, treat, package, and transport the patient to definitive care. Although paramedics may be accustomed to providing care and transport based only on a brief focused exam and limited diagnostic information, medical and nursing CCTPs need to be aware that more comprehensive assessment and sophisticated treatments are best deferred until the patient arrives at a definitive care facility.

Interfacility Transport

Unlike scene transports, voluminous patient information is sometimes available before arrival for interfacility transports. CCTPs should take the time to review all available data and patient information before arrival to reduce the need for referring staff to review or repeat information already transmitted when requesting the transport and to more intelligently interact with staff when receiving the patient for transport. In stark contrast with scene transports, CCTPs should gain a comprehensive, overall picture of the patient as much as possible before initiating transport. This activity includes performing any procedures or diagnostic tests necessary to reduce the likelihood of adverse events occurring during transport.

It is important for the transfer team to have a general understanding of the patient's situation. It can be challenging even for a health care provider to describe in 5 minutes conditions that may have been evolving for 15 days. When preparing for an interfacility transport, the hospital staff needs to understand that the service is not there just to transport the patient from point A to point B. In fact, this period of transfer of an acutely ill or injured patient may become the most critical point in the continuum of total care. An alert CCTP will prompt for and inquire about needed information. A professional manner should be exhibited to maintain good relations with hospital staff. The transport team should respect the nurse-

patient dynamics in the critical care unit because often they have developed a therapeutic relationship that can lead to separation anxiety and increased stress as a result of the transfer.

Anticipated transport time is a critical variable in differentiating "need to know" from "helpful to know" information. The longer the transfer, the more information that may be necessary to provide appropriate assessment and intervention.

The hospital medical record may not contain all the data listed in **Table 5-1**, but it typically includes several special sections based on the patient's diagnostic and medical care needs:

- Admission orders
- Advance directives
- Operative notes
- Postoperative notes
- Progress notes
- Consultation notes
- Preoperative notes
- Procedure notes
- Discharge summary
- Lab reports
- Medication administration records
- Nurses' notes and flow sheets

Scene Transports

Evaluating Available Information

At the beginning of a scene transport, in addition to eliciting patient assessment findings from scene personnel, it is important to ascertain details from the scene that might not be available to providers treating the patient at the receiving facility. These details include the same information that prehospital care providers routinely record and convey, including mechanism of injury details such as speed, distance of fall, type of weapon, and duration of environmental exposure **Figure 5-1**. CCTPs accustomed to practicing in the prehospital environment routinely observe and record pertinent details from the scene and may even include digital or instant photographic images with the patient record.

Current Interventions

Before leaving the scene, it is imperative to obtain as much detail as possible about interventions provided before arrival of the CCTP. These interventions include medications administered, procedures performed, and any other care provided.

Interventions provided on the scene and during transport are recorded using the data collection system of the transport service. At any given moment, a CCTP should be able to reference or immediately recall the total volume of fluids administered, the total doses of each medication administered, and the major procedures performed.

Figure 5-1 CCTPs should observe details from the scene when arriving at a scene transport.

Responses to Interventions

Patient outcomes are largely described by response to interventions. Intervals between reassessment vary and are based on patient acuity. Nevertheless, observing patient response to each intervention and recording these data cannot be overemphasized.

Patient Status

Numerous scoring systems and patient status descriptors have been developed and are in use in in-hospital and prehospital settings. The most useful terms for describing patient status in general are objective and measurable in nature. Subjective terms such as stable, unstable, no acute distress, mild distress, moderate distress, and in extremis are left to interpretation of the caregiver. The Glasgow Coma Scale (GCS) score is one prehospital scoring system that is useful and that facilitates communication between providers and with receiving facilities.

Process for Patient Assessment at the Scene

Patient assessment by CCTPs for scene transports is no different from patient assessment conducted as a field provider except that CCTPs may have available and use more sophisticated patient monitoring devices and technologies on scene and during transport **Figure 5-2**.

Patient Packaging for Transports

Packaging for on-scene transports is dependent on multiple variables, including the physical location of the patient, care in progress on arrival, temperature and weather conditions, and the type of CCTP transport vehicle. Often one CCTP will obtain a patient report while a second CCTP applies the monitoring equipment, conducts a cursory physical assessment, performs any necessary procedures such as obtaining venous access or securing the airway, and bundles the patient and equipment for transfer to the transport unit stretcher.

Figure 5-2 A critical care transport ambulance.

Local practice protocols and procedures dictate the sequence of packaging and moving the patient for transport and the methods used to secure lines, tubes, and equipment before and during transport. The most important component of patient packaging for scene transports is expediency. Delays are not in the best interests of patients, the credibility of CCTPs, or the reputation of the critical care transport service with the field providers on scene.

Interfacility Transports

Evaluating Available Information

Like a CCTP with extensive prehospital experience, a seasoned critical care transport nurse or physician can often scan the "scene" around a hospitalized critically ill patient and instantly gain considerable insight. Drips infusing, ventilator settings, numbers and types of lines, drains, and hemodynamic monitoring values all provide vital information about a patient's condition.

Although actual physical assessment of a patient is focused on the diagnosis or presenting problems following the outline later in this chapter, assessment of ventilator settings, medication infusions, and monitoring equipment in use must be detailed and thorough. Ventilator settings need to be replicated using the transport ventilator, drip fluids may need to be remixed or transferred to different infusion pumps, and monitoring devices and equipment need to be replaced with the critical care transport monitors. To ensure patient safety, CCTPs must assess each therapy before and after transfer to the transport equipment **Figure 5-3**.

Figure 5-3 Assess each therapy the patient is undergoing before and after making transfers to the transport equipment.

Current Interventions

Unlike scene responses, current and previous interventions on interfacility transports can be extensive. Although the history may be contributory, it is important to maintain a focus on the current problem necessitating the need for transport because subsequent care will be predicated on previous interventions. In addition to the therapies presently in place, CCTPs need to learn the other interventions initiated to address the problem at hand, when they were attempted, and their outcomes.

As at scene responses, CCTPs should continually maintain an awareness or be able to immediately access the total volume of fluids administered, including blood and blood products, total doses of each medication administered, and the major procedures performed within the previous 24 hours.

Responses to Interventions

Response to treatments in the interfacility arena tends to be more clear-cut than in the prehospital setting. Often, the reason for critical care transport is to access services not available at the current facility. This supports the principle of getting patients to the most appropriate facility from the beginning of their illness. Sometimes going to the closest in-territory facility actually adversely affects patient outcome.

It is extremely useful to inquire about treatments and interventions that had been tried previously (for example, a phenylephrine infusion administered yesterday that failed to increase the patient's blood pressure or repeated platelet transfusions that did not result in an increased platelet count). Such information is not always easily elicited from the medical record but can benefit patient care during transport and provide helpful information for the receiving facility. In addition, CCTPs should inquire about patient-specific responses to current treatments, recognizing that individuals respond differently to medications and treatments. A critical care nurse caring for a patient can likely provide the precise increment to increase or decrease a pressor infusion in response to changes in blood pressure. Nurses are also probably well versed in the precise amount and type of sedation and/or analgesia needed to manage breakthrough pain or agitation in a specific patient. Obtaining this information before assuming patient care promotes a safer and smoother transition of care.

Patient Status

Interfacility transports encounter patients in two types of conditions: stable and unstable. Standardized scoring systems (ie, APACHE [Acute Physiology and Chronic Health Evaluation]) and other tools to

describe patient status are helpful for charting, billing, and coding but have little value for hands-on patient care. Although much of the assessment information used during an interfacility transport is obtained from the medical record or verbal report from providers at the bedside, determination of patient status must be done by CCTPs.

As with patient responses to interventions, critical care providers at the bedside can be most helpful in outlining anticipated problems during transport. CCTPs should ask about known issues with equipment, specific patient behaviors, and suggested responses to problems that have previously been observed. Knowing, for example, that an endotracheal tube has a small cuff leak and requires 0.1 mL of air every 2 hours, that an intra-aortic balloon pump alarms rapid gas loss whenever the patient coughs, or that a patient's routine runs of ventricular tachycardia are readily converted with 50 J of biphasic energy can save considerable trial and error during transport and make the voyage safer for the patient.

One imperative before initiating any interfacility transport is learning whether there are any advance directives in place. If so, CCTPs should be certain of the specifics contained in these documents and have copies of them during the transport.

Process for Patient Assessment at the Transferring Facility

As with scene transports, CCTPs often split duties, with one provider obtaining report from the transferring facility staff and the other conducting a patient assessment and applying the transport unit monitors, infusion pumps, ventilators, and other equipment. Because substantial assessment data are obtained during report rather than direct assessment, it may be more appropriate in many situations for all CCTPs to listen to a bedside report or to receive report while simultaneously conducting an assessment and transfer of care. "Bedside report" is a national standard in hospitals to improve efficiency, decrease errors, decrease overlooked information, and include patients in the plan of care.

Assessment should begin on entry to the sending facility with an overview of the surroundings to ensure that hallways, elevators, and routes of travel will accommodate the patient, equipment, and personnel to the transport unit. After or while obtaining report from the bedside care providers, CCTPs transfer the patient's monitoring to the transport monitoring equipment, making certain that readings are reliable and replicate those previously observed on the hospital's monitors. By using the report received from the transferring staff, CCTPs conduct an appropriate assessment using the outline presented in Table 5-1. As previously mentioned, although CCTPs have the advantage of more available time for assessment during interfacility transports, it is never necessary to perform every assessment outlined; CCTPs should choose the assessments pertinent to the patient condition and reason for transport.

To better appreciate the balanced, holistic approach to the patient and the situation, when performing an interfacility transport it is best to begin with a general overview that includes items routinely noted in the medical record. Typically, CCTPs will receive this general patient overview from the primary critical care nurse during the patient transfer report and, perhaps, also in written form on the hospital transfer sheet. This brief synopsis should review applicable elements from the following categories:

- Admission data
- History
- Allergies

TABLE 5-1 General Patient Overview for Interfacility Transport

Category	Elements	Examples
Admission data	Name Date Time Admitting diagnosis Sex Age Height Weight Baseline vital signs Mode of arrival Location from which the patient was admitted Attending physician of record Family physician or primary care provider	N/A
History	Chief complaint History of present illness Family medical history Social history Past medical history Hospitalizations Past surgeries and dates	Diabetes Cancer Bleeding tendencies Heart disease Hypertension Stroke Seizures Lung disease Hepatic or renal disorders Other
Allergies	Medications Foods Dye Environment Latex Anesthesia • Prior experience • Any reactions Past blood transfusion • Reaction Types of reactions for all	N/A
Medications	Home In hospital Last use During transport Route, dosage, indication, actions, adverse effects, contraindications, and so forth	N/A
Radiologic films and findings, diagnostic reports	Recent chest film CT scans, cardiac catheterization films, MRI • Copies (printed or electronic) of pertinent CT scans and cardiac catheterization films for transport 12-lead electrocardiogram (copies in transfer record)	Confirms correct placement of ET tube, gastric tube, central lines, chest tubes Unusual ECG patterns
Laboratory and pathology findings	Electrolyte levels ABGs Complete blood cell count Pathology reports (copies in medical record)	Sodium, potassium, and glucose suitable for transport Stable ABG levels Potential transfusion or clotting factor needs
General health	Perception and maintenance patterns Description of general state of health (patient's words) Patient description of own mental and emotional state Greatest concerns and expectations Recent major life changes Cultural beliefs affecting health Anticipated length of hospitalization Tobacco use and amount Recreational drug use, types, and amounts	Medications Dressings and bandages Appliances (such as braces, artificial limbs, dentures, and hearing aids)
Activity	Bed rest (confined) Ambulatory • Independent • Requires assistance Chair Specialty care bed in use	Type of specialty care bed in use
Integumentary system	Wounds	

Integumentary system	Wounds Pressure ulcers or skin breakdown Description of each • Location • Appearance • Drainage (color, amount) Dressing (condition, for example, dry and intact)	N/A
Cognitive/perceptual status	Review of systems, physical assessment Affect and communication style and ability	GCS Alert Agitated Confused Oriented (person, place, and time) Restless Other Language other than English Interpreter
	Eyes: vision	Normal Blind Diplopia Other
	Pupils • Size in millimeters • Reaction to light	No reaction Brisk Sluggish Hippus
	Hearing	No difficulty Hearing impairment Deaf Pain Other
Cardiopulmonary status	Skin	Normal Jaundiced or pale Dry Cool Diaphoretic Warm Within normal limits Edematous or dehydrated Intact Broken
	Heart sounds	S_1 S_2 S_3 S_4 Clicks Murmurs Gallops
	Dyspnea	At rest On exertion Orthopnea
Cardiopulmonary status (continued)	Lung sounds	Clear and equal Crackles or rales Rhonchi Wheezes Stridor
	Chest pain	History

		Site Onset Severity Radiation of pain
	Respiration	Within normal limits Excursion Dyspnea Cheyne-Stokes Agonal
	Edema	Pattern Nonpitting Pitting
	Peripheral pulses	Dorsalis pedis, right and left Radial, right and left
Gastrointestinal and genitourinary status	Diet	Appetite New diagnosis of diabetes or renal or cardiac disease Diagnoses of malnutrition, eating disorder, multisystem trauma Need for nutritional education Need for tube feeding, enteral nutrition, or total parenteral nutrition 10-lb weight loss or gain in past month Dysphagia
	Bowel movement	Normal Last bowel movement Rectal bleeding (color)
	Urination	No difficulty Hematuria Catheter Frequency Urgency
	Female reproductive system	No difficulty Last menstrual period Birth control Hysterectomy Other
	Mouth	Moist Cracked mucosa Sores or lacerations Odors Other
	Bowel sounds	Present Absent Other
	Ostomy	Location
	Abdomen	Soft Firm Flat Distended Tenderness or pain and the location Radiation of pain
	Genitalia	Tender
Musculoskeletal system	Limitations in daily living Weakness and its location Gait, within normal limits, assistance devices, other Orthopaedic appliances History of pain and its location Deformity	N/A

	Deformity Contracture and the location Hand grasp, equal or other Fall prevention protocol	
Personal belongings	Eyeglasses Contact lenses, right and left Intraocular lenses Artificial eye Dentures, upper and lower Hearing aid Electrical appliance Jewelry Prosthesis Other	N/A
Advance directives	Patient and family understanding	State laws Living wills Durable power of attorney for health care Guardianship DNR (do not resuscitate)/DNI (do not intubate) Organ donor Consent form signed Medical order signed
Patient and family understanding and educational needs	Orientation to environment Disease process Diagnostic tests Medication Treatment and therapies Diet and nutritional screening Activity and exercise Equipment and special training Transfer reason Orientation to critical care transport Social service Rehabilitation Other	N/A

Abbreviations: ABG, arterial blood gases; CT, computed tomography; ECG, electrocardiographic; ET, endotracheal; GCS, Glasgow Coma Scale; MRI, magnetic resonance imaging; N/A, not applicable.

- Medications
- Radiologic films and findings, and diagnostic reports
- Laboratory and pathology findings
- Health perception and maintenance pattern
- Activity
- Integumentary system
- Cognitive-perceptual status
- Cardiopulmonary status
- Gastrointestinal and genitourinary status
- Musculoskeletal status
- Personal belongings
- Advance directives
- Patient and family understanding and educational needs

Table 5-1 provides a sample of elements from each of these categories. History taking and physical examination can be overdone, so the elements unique to the patient's needs should be used to develop an individualized critical care plan and documentation designed to meet those needs.

Institutions, critical care transport programs, and states all have varying policies with regard to the transfer of narcotic or controlled substance infusions from one facility or provider to another. It is important to be aware of these policies, if they exist; and to be flexible with regard to remixing, altering, or discontinuing sedation infusions if a hospital policy does not allow you to remove medication infusions from the facility. Involvement of the transferring or receiving physician and/or online medical control may be indicated for patients who will require significant changes in their sedative management for transfer.

Patient Packaging for Transports

Skill Drill 5-1 shows steps for packaging patients for interfacility transports, as follows:

1. Once monitoring equipment is in place, transfer any infusions to the transport unit infusion pumps **Step 1**.
2. Be certain to:
   - Read labels of bags **Step 2**.
   - Trace infusion tubings completely between the bag and the connection to the patient **Step 3**.
   - Label the distal end of the tubing with the infusion name **Step 4**.
3. Once all infusions have been transferred and are operational, reassess for hemodynamic stability and proceed to transfer the patient to the transport unit stretcher. Recheck the integrity of the endotracheal tube and other invasive lines after each transfer.
4. Once the patient is comfortably positioned on the transport stretcher, transfer any other necessary equipment (such as ventilators, continuous positive airway pressure units, and intra-aortic balloon pumps) **Step 5**. Ideal patient position may not be supine. It is important to consider variables such as cervical spine, pain, shortness of breath, and the presence of an endotracheal tube. If a patient is intubated, the ideal position is with the head of the stretcher elevated at least 30°. This helps prevent aspiration pneumonia.
5. Secure all equipment to the transport unit stretcher or carrier, bundle the patient as appropriate for weather conditions, and reassess to ensure that no changes in condition have occurred **Step 6**.
6. Before leaving the patient care area, be certain that all necessary equipment adapters and connectors are with the patient, ensure that family members have directions to the receiving facility, and provide transferring staff with contact information should they want to follow up on the transport of the patient. Show your appreciation for the care and assistance provided by the staff in packaging and transferring the patient to your care.

The Critical Care Systems Assessment

As previously stated, critical care assessment is typically systems based, using observational skills and clinical parameters of physiologic status. The need for technologic monitoring is to support observations or provide greater specificity and/or differentiation to the findings. With this in mind, the following sections review:

- General appearance
- Cardiovascular system assessment
- Respiratory system assessment
- Neurologic system assessment
- Gastrointestinal system assessment
- Genitourinary system assessment
- Musculoskeletal system assessment
- Psychosocial and emotional system assessment

The next sections are not absolute or exhaustive but simply provide a functional framework to

discuss the various elements of the critical care systems assessment. The depth of a systems assessment is dictated by the patient condition. For example, a patient with a subarachnoid hemorrhage would require a detailed neurologic exam at least hourly. Further, limitations of assessment during transfer (noise and turbulence) would lead a prudent CCTP to conduct the assessment prior to leaving the scene. This is especially true during flight transport.

It is important to note early on in the study of physical assessment that the assessment and the data obtained must be individualized to patients and their conditions. Generalization of assessment findings may lead a CCTP astray and impact patient care, as well as CCTP credibility. For example, a newborn with cyanosis of the lips should illicit a different response from a clinician than an adult with the same. The ability of CCTPs to obtain, interpret, and include or exclude data based on a patient's differential diagnoses is referred to as critical thinking. The basic premise of critical thinking can be taught, but only after many years of experience in application can the skill be honed.

In addition, providers must realize that the subjective information obtained from the patient and the family is as important, if not more important, than the objective information obtained during the actual exam. The subjective history includes chief complaint, review of systems, and history of past and present illnesses. These findings should be recorded in the patient's record as direct quotes from the patient and/or family. This information should guide the provider to ask other questions or to examine objective data to qualify the information.

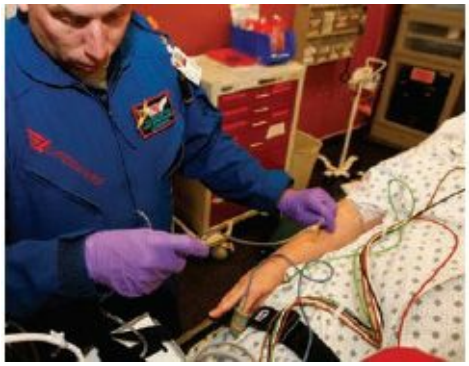
Furthermore, what appears in the beginning of the assessment learning process to be cumbersome will with time occur without an intentional effort. With experience, much of the critical care assessment can be completed with just casual encounters with the patient. For example, if while being placed on a stretcher a patient asks for four pillows, an alert CCTP will inquire more about the need for the pillows. For example, the CCTP should identify orthopnea, or shortness of breath while lying flat, often because of left ventricular dysfunction. The CCTP may then ask, "How long have you been sleeping on four pillows?" in an attempt to ascertain worsening of the condition. This exploration of the system will lead to other assessment parameters such as shortness of breath with activity. Merely listening to patients and their families will provide a myriad of information that assists CCTPs in assessing and treating patients.

Skill Drill 5-1

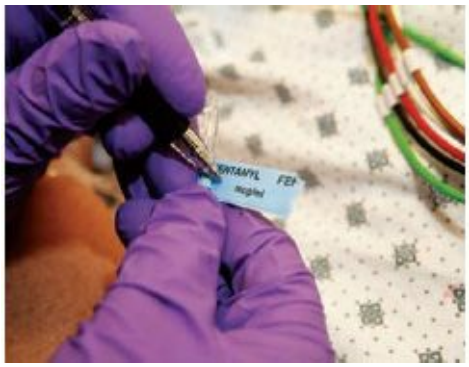
Packaging Procedures for an Interfacility Transport

1 Once monitoring equipment is in place, transfer any infusions to the transport unit infusion pumps.

2 Be certain to read labels of bags.



3 Be certain to trace infusion tubings completely between the bag and the connection to the patient.



4 Label the distal end of the tubing with the infusion name. Once all infusions have been transferred and are operational, reassess for hemodynamic stability. Proceed to transfer the patient to the transport unit stretcher.

5 Once the patient is comfortably positioned on the transport stretcher, transfer any other necessary equipment.

6 Secure all equipment to the transport unit stretcher or carrier, bundle the patient as appropriate for weather conditions, and reassess to ensure that no changes in condition have occurred. Before leaving the patient care area, be certain that all necessary equipment adapters and connectors are with the patient. Ensure that family members have directions to the receiving facility. Provide transferring staff with contact information for follow-up. Show your appreciation for the care and assistance provided by the staff.

Assessment of General Appearance

The evaluation of general appearance includes an assessment of the patient's apparent health status based on data from the medical records and determination of parameters such as the apparent age relative to chronological age, the level of consciousness (LOC), and skin findings. CCTPs should also note the presence or absence of gross deformity, stature, posture, gait (if the patient is ambulatory), presence and degree of edema, skin lesions, and state of the fingertips and nail beds. Position of comfort is another piece of data that can be used to diagnose disease process. For example, the tripod position in toddlers can signify cardiovascular or respiratory disease. Whenever feasible, it is prudent to allow the patient to assume the position of comfort during transfer.

Skin temperature is another important factor to consider. A cool temperature may indicate vasoconstriction, and a warm or hot temperature may indicate fever. Whether the skin is wet or dry is also of interest. **Turgor**, rapid or sluggish, is an indicator of fluid status. Turgor should be assessed over the sternum or forehead. Use of the hand to assess turgor is often misleading, especially in elderly people and people with friable skin.

Cardiovascular Assessment

Inspection

A general overview or inspection of the patient provides data lending support to an acute diagnosis or gives a clue to a chronic health condition. Just by looking at the patient, an overall sense of the patient's baseline health can be surmised. Is he or she morbidly obese? Cachectic? The patient's skin color, central and peripheral, should be assessed. Either pallor or cyanosis can indicate hypoxia. Cyanosis of the nail beds may indicate acute oxygen insufficiency or, when combined with clubbing **Figure 5-4**, an angle of more than 160° between the nail and nail bed, may indicate long-term hypoxia such as associated with chronic obstructive lung disease.

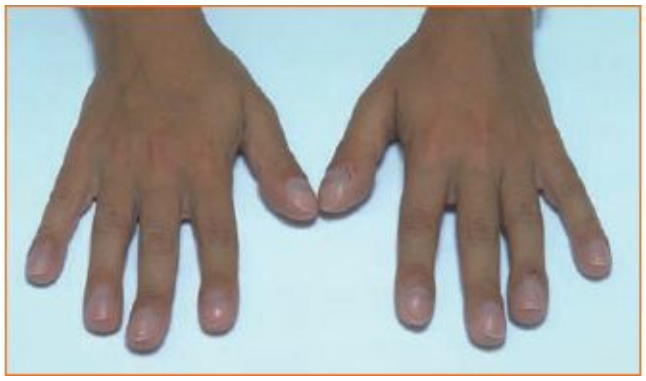


Figure 5-4 Clubbing.

Special Populations

Peripheral vascular disease (PVD) affects 10% to 40% of the population and is more common in patients with diabetes and in older people. Although 70% to 80% of persons with PVD are asymptomatic, the occlusive nature of PVD can have profound influences on assessment findings. Most significant PVD affects the lower extremities. This can lead to diminished peripheral pulses in the lower extremities and may require a Doppler device to detect arterial flow when pulses are not palpable. Significant PVD in one or both upper extremities can lead to falsely low arterial or cuff blood pressures in the affected arm. Verification of unusual blood pressures with measurement in a different extremity can help avoid unnecessary treatment.

When edema is present, the location and the severity should be noted. Pedal edema is usually graded on a scale from trace to 3+ or 4+ pitting depending on the scale used. Pedal edema should be evaluated by pressing on the skin behind the medial malleolus, over the shin, and over the dorsum of each foot with the thumb and index finger for at least 5 seconds. Indentation can be noted by running the pads of the fingers over the area pressed. A slight indentation that disappears in a short time is termed trace edema. Grade 3+ or 4+ (depending on the scale) is deep pitting that does not disappear readily. The location of the edema and the usual position of the patient should be noted and is used to differentiate between dependent and nondependent edema. In ambulatory and seated patients, edema develops in their lower extremities, whereas patients confined to bed may have more edema in the sacral area.

Caution should always be utilized with subjective scales such as those used to grade pulses and edema. The reason for this is that discrepancy between caregivers can be limb threatening. It is important to perform bedside assessments with the nursing staff prior to leaving the bedside.

Interpretation of the ECG rhythm is essential. As mentioned earlier, if a 12-lead ECG has been recorded, a copy should accompany the transfer record. Questions to consider include the following: What is the underlying or baseline rhythm? What is the usual monitoring lead and morphology? What arrhythmias has the patient experienced? What effects did the arrhythmia have on the patient and was any treatment used? Has the patient ever required defibrillation or cardioversion? If the patient has a pacemaker and/or implanted cardiodefibrillator, a record of the current settings should accompany the transfer record.

Although it is comforting to a health care professional to have invasive monitoring equipment, many times, a skilled health care professional can obtain the same data by using assessment skills. For example, central venous pressure or pressure in the right atrium, which is an indicator of fluid status, can be obtained by using an invasive triple lumen catheter or just by observing for jugular venous distention **Figure 5-5**.

A useful assessment technique that uses inspection is testing the **hepatojugular reflex**. This technique is performed by applying midabdominal pressure while observing for jugular venous distention. If noted, the test result is positive. Pressure on the abdomen displaces fluid from an engorged liver, indicating volume overload.

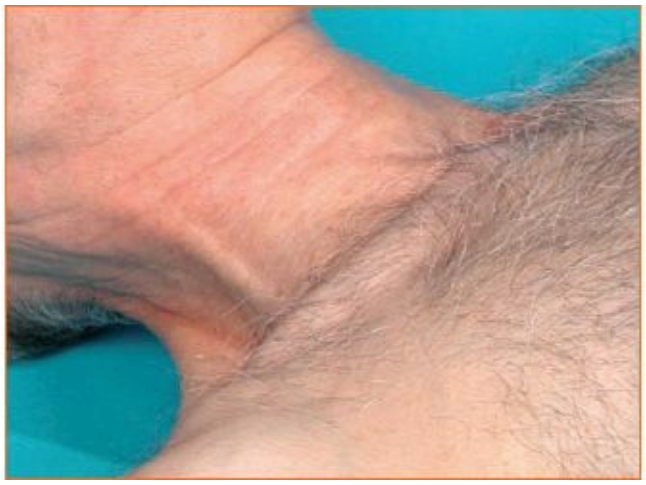


Figure 5-5 Jugular venous distention.

Assessing Pulse

Assessment of peripheral pulses provides valuable information. The carotid, radial, brachial, femoral, popliteal, posterior tibial, and dorsalis pedis should be assessed bilaterally for presence, strength, and pattern. Carotid pulses should be palpated one side at a time and without a massaging-type action. Failure to follow this guideline may result in a vagal response and loss of consciousness or embolization of carotid plaque deposits.

Often, trending of pulse assessment findings over time is more valuable than one-time assessments. For example, it is prudent to have two persons assess the strength of the pulses in a patient with a lower extremity crush injury before being transported to a trauma center from a tertiary center. Both should agree and record that, for example, the dorsalis pedis pulses are present bilaterally before transport, but in transit, reassessment shows the pulses still present but slightly weaker. This is a significant finding to be addressed immediately. This is a good example of when a CCTP and the nurse should assess circulatory status together at the bedside. Simultaneous assessment by the nurse and CCTP certainly is not necessary for the entire assessment of all patients, but when life or limb is compromised, it is prudent practice.

Patterns of pulsations are also of interest. In some ECG rhythms, not all electrical beats are perfused. For example, a patient may have ventricular bigeminy with a heart rate of 80 beats/min, but if only 40 of these beats are perfused, further assessment will be needed to determine hemodynamic stability. (A quick method of assessing how many beats are perfused is to compare the rate on the ECG monitor with the heart rate on a pulse oximetry display.) Similar scenarios can occur with sinus or atrial arrhythmias. Furthermore, a noted decrease in pulse rate with inspiration may occur with left ventricular failure.

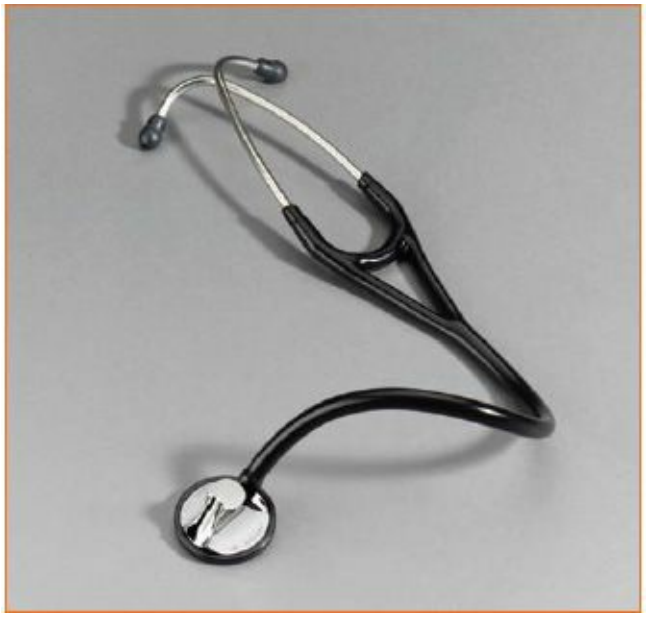


Figure 5-6 Stethoscopes with a diaphragm and bell, single-lumen tubing, and short tubing are ideal.

Auscultation

Auscultation of heart sounds is an art that requires years of practice to become proficient. The heart should be auscultated at the aortic, pulmonic, tricuspid, and mitral valve locations with the diaphragm and bell of the stethoscope. Furthermore, a quality stethoscope should be used. Stethoscopes with a diaphragm and bell, single-lumen tubing, and short tubing are ideal **Figure 5-6**. Newer stethoscopes incorporate the features of the bell into the diaphragm. When the operator (or CCTP) applies pressure over the diaphragm, it produces the acoustics of a bell. Conventional bell and diaphragm stethoscopes are rapidly being replaced by these newer, easier to use models. Stethoscopes with microphones are not recommended because of the adventitious sounds that they can create.

A thorough understanding of anatomy and physiology is critical to understanding auscultation. Each heart sound is evaluated for pattern, intensity, quality, and pitch. This includes not only listening for the normal heart tones to be sure that they are in fact normal, but also listening for adventitious sounds that can be critical to your patient. Chapter 13 discusses heart sounds in greater depth.

Finding a new murmur may be significant. A systolic murmur appearing after an inferior myocardial infarction is much more significant than a diastolic murmur that a patient has had since turning 90 years old. Also, generally speaking, diastolic murmurs are more concerning than systolic murmurs.

Auscultation over the carotid, renal, and femoral arteries is another assessment parameter to consider in critically ill patients. Detection of bruits and/or loud, harsh sounds indicates blood flow through a narrowed artery and, particularly in the carotid arteries, can be quite significant even in the absence of symptoms.

Blood pressure is a common but often underemphasized clinical parameter. Normal blood pressure in critically ill patients is defined by the individual patient, not a textbook. Indeed, a systolic blood pressure of 90 mm Hg in a 20-year-old may be normal, but this pressure would be abnormal in a patient with a recent myocardial infarction and a history of hypertension. Before transport, trends in blood pressure over time should be noted, especially in response to cardiogenic medications and interventions. When applying the transport unit monitoring equipment, CCTPs should be certain to compare measurements obtained with trends reported and investigate any differences before packaging the patient for transport. Blood pressure should be reassessed at intervals based on patient condition or local protocol. Typically, blood pressure is reassessed every 5 minutes in acutely ill critical care patients and up to every 15 minutes in stable critical care patients. Obviously, more frequent measurements may be

warranted to assess the results of medications or other interventions or if it appears that the patient's condition has changed. When titrating pressors, 5-minute intervals would be the maximal safe interval for blood pressure measurement. Orthostatic blood pressure was previously used in the critical care environment to assess the need for fluid resuscitation. The practice had obvious limitations because it required sitting and standing measurements of blood pressure, which are often impractical. **Passive leg raising (PLR)** is presently used to assess fluid responsiveness in patients with suspected volume depletion. PLR is performed with the patient in a supine position by a provider raising both of the patient's legs to a 45° angle **Figure 5-7**. The blood pressure is then assessed again while the patient's legs are raised. Within 30 seconds, PLR results in a reversible autotransfusion of some 150 to 300 mL of blood into the central circulation. Improvement in blood pressure using PLR suggests fluid-responsive volume depletion. Lack of improvement suggests that no benefit would be derived from volume administration.

Figure 5-7 Passive leg raising is useful for assessing the need for fluid resuscitation. If blood pressure improves while the legs are raised, the patient will benefit from fluid resuscitation.

As the art and science of physical assessment is studied, the question arises, "In what order do I perform assessment parameters?" Again, CCTPs must understand that not all parameters are assessed on all patients, nor are all parameters checked on the same patient at each assessment. A generally accepted critical care standard is that a head-to-toe assessment should be completed no less frequently than every 4 hours and more often when required, based on patient acuity. It is also important that all health care professionals develop an assessment sequence with which they are comfortable. Following the same pattern all the time will prevent inadvertently leaving out an assessment parameter. Adhering to this strategy also improves the time management skills of CCTPs.

Respiratory Assessment

Inspection

Inspection involves the visual examination of the patient with emphasis on the chest. CCTPs should assess the patient and inspect the chest for the following:

- Mental status
- Skin color and temperature; dry or diaphoretic skin
- Presence of an artificial airway (endotracheal or tracheostomy tube)
- Breathing spontaneously vs need for mechanical ventilation
- Equal chest expansion with each breath

- Use of accessory muscles such as scalene, sternocleidomastoid, and/or intercostals
- Work of breathing; labored or unlabored Presence of chest tubes, central lines, and dressings Presence of signs of injury (such as bruising, laceration, and penetrating wounds)

The shape of the chest wall provides evidence of trauma, congenital anomalies, and chronic obstructive pulmonary disease (barrel chest or increased anterior-posterior diameter). Respiratory patterns may lend a clue to an underlying problem as outlined in Chapter 6.

Work of breathing is assessed by inspection of the use of accessory muscles, intercostal retractions or bulging, and nasal flaring. Increased work of breathing requires great metabolic reserve and can be tolerated only for a short period before respiratory decompensation occurs. *Children with signs and symptoms of increased work of breathing are in grave condition and require immediate aggressive interventions*.

Special Populations

Children with signs and symptoms of increased work of breathing are in grave condition and require immediate aggressive interventions.

Palpation

Palpation can also yield information about the skin and subcutaneous tissues. Is the skin warm and dry or cool and clammy? Is there crackling when the skin and tissues are palpated? Feeling a "snap, crackle, pop" during palpation indicates the presence of subcutaneous emphysema, indicating that air is escaping from the pleural space and is dissecting through the subcutaneous tissues. Although subcutaneous emphysema is itself benign, the underlying cause may be life threatening.

A few critical care respiratory assessment parameters worth mentioning include palpation of the trachea for alignment, palpation of the chest wall for bilateral chest excursion, and palpation for subcutaneous emphysema. Each of these parameters has unique considerations. A deviated trachea may indicate the presence of a solid tumor (trachea moves away from tumor) or pneumothorax (trachea moves toward affected lung in a simple pneumothorax but deviates away in a tension pneumothorax). One way of remembering this is that the trachea deviates to the area of least resistance.

Chest excursion should be equal and symmetrical bilaterally. In other words, the chest should rise and fall with inspiration and expiration. A good method of determining where the chest excursions are equal bilaterally is to look at the patient while standing at the patient's feet rather than at the side. By standing at the feet, even small changes in excursion patterns, right vs left, can be detected. Unequal or asymmetrical chest excursion may indicate disease such as a pneumothorax.

Finally, subcutaneous emphysema is a useful indicator of disease states such as a ruptured **bleb**. In a patient with a chest tube, subcutaneous emphysema may indicate an air leak. A small amount of subcutaneous emphysema at the insertion site of a chest tube is a normal variant. A critically ill patient who requires mechanical ventilation and in whom subcutaneous emphysema develops during transport may be receiving too much tidal volume or positive end-expiratory pressures from mechanical ventilation. Another possibility is that the chest tube could have dislodged during transfer from bed to stretcher. A good rule to follow is to assess not only endotracheal tube placement, but also all line positions before and after any transfer.

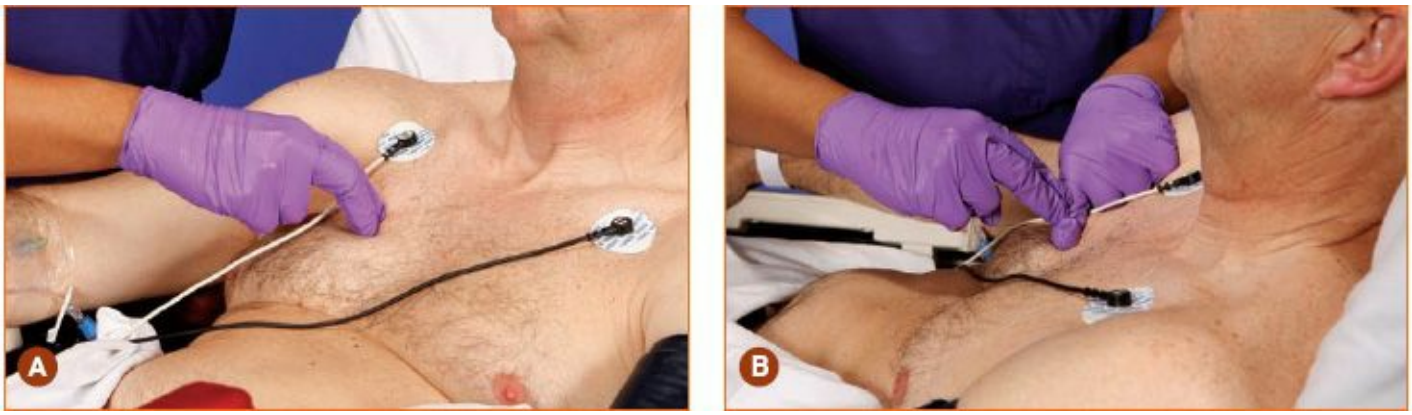


Figure 5-8 **A.** One-handed percussion. **B.** Two-handed percussion.

Percussion

This skill, although often underused, is valuable but requires CCTPs to have a sound knowledge of anatomy, physiology, and percussion technique. This technique is particularly useful when radiography is not available, although it requires a less noisy environment than often found in an intensive care or critical care transport environment. The technique of percussion is performed using one or two hands. The one-handed technique involves using the index and middle fingers to rapidly strike the skin over the lung fields **Figure 5-8A**. The two-handed technique involves striking the index finger of the free hand as it sits over the lung field to be examined **Figure 5-8B**. Attention is given to the sound made by percussion.

Normal lung fields produce **resonance** during percussion. If dullness or flatness is noted, fluid or solid tissue (tumor) may be present. **Hyperresonance** produced during percussion may indicate an air-filled emphysematous lung. Differentiating percussion sounds comes with practice and experience. These sounds are summarized in **Table 5-2**.

Auscultation

The importance of auscultation over all lung fields cannot be overemphasized. Lungs should be auscultated anteriorly, posterially, and medially at the midaxillary lines. All lung fields should be assessed for the presence of normal breath sounds, such as tracheal, bronchial, vesicular, and bronchovesicular sounds, and for adventitious sounds, such as crackles, rhonchi, stridor, and wheezing.

TABLE 5-2 Summary of Percussion Sounds

Sound	Intensity	Pitch	Example
Tympany	Loud	High	Air-filled stomach
Hyperresonance	Loud (louder than resonance)	Low	Emphysematous lung
Resonance	Loud	Low	Normal lung
Dullness	Medium	Medium	Liver, muscle, bone
Flatness	Soft	High	Thigh

The choice of auscultation sites affects the sound heard. One side of the chest wall should be compared directly with the opposite side. Bony landmarks should be used to ensure listening to the same site on each side of the chest, to compare bilaterally. Normally, sounds heard in a particular part of the chest on the right side should sound the same on the corresponding location on the left side. Each of the five lung lobes should be auscultated for a baseline assessment. This is accomplished by listening in as few as six places on the anterior chest wall; the specific locations are shown in Chapter 6 with an in-

depth discussion of abnormal breath sounds.

Normal breath sounds are only normal when heard over the area at which the sound is considered normal. For example, tracheal sounds heard past the midline would not be normal. Notation should be made to the anatomic location, the phase of respiration, and the presence of adventitious sounds. Crackles indicate air passing over fluid in small airways and accompany conditions such as atelectasis and fluid congestion. Rhonchi indicate air moving through large airways containing secretions. Wheezing indicates air moving through narrowed airways. Inspiratory wheezing occurs in upper airway obstructions, and expiratory wheezing occurs with asthma attacks.

When transporting a critically ill patient who is mechanically ventilated, it is important to note current ventilator settings and parameters. Baseline arterial blood gas measurements are essential as well. Maintaining the airway and adequate ventilation is of highest priority during transport. It is important to remember that more oxygen is not always better. CCTPs should always be prepared to invasively protect the airway. The patient's response to the transport ventilator, which may be different from response to a nonportable ventilator, should be assessed. CCTPs need to be prepared to take corrective measures if necessary. Adjustment of inspired oxygen levels to maintain saturations between 94% and 98% is probably appropriate for most patients. It is important to remember that oximetry reflects the percentage of hemoglobin saturated with oxygen. In the presence of very low levels of hemoglobin, even 100% saturated hemoglobin may not be sufficient to meet tissue oxygen needs.

Neurologic Assessment

Neurologic assessments are often the most challenging for CCTPs because of the complexity of the neurologic system. The focus of this discussion is a general neurologic assessment. The neurologic exam is somewhat different from the other system exams because it tends to be more focused to the individual needs of the patient.

Before critical care transports, a neurologic exam is necessary to establish the patient's baseline status for comparison with later findings to determine changes in the patient's condition. CCTPs must have a thorough knowledge of the range of normal responses to determine whether the patient's condition is improving or worsening.

The diagnosis and immediate clinical history of the patient guide CCTPs as to the detail needed for a proper neurologic examination. The LOC is the most important parameter in evaluating a patient with a central nervous system injury.

The GCS is the focal point of assessing a critical care patient. The scale assesses the LOC by evaluating eye opening, verbal, and motor responses to stimulus. This information is scored as shown in **Table 5-3**, and a total value in the range of 3 to 15 is assigned to the patient. The GCS was developed to standardize observations for the objective and accurate assessment of the LOC. A score of 3 indicates deep coma, and a score of 15 is normal. As the score falls, the morbidity and mortality of the patient increases. Medications such as anxiolytics, narcotics, paralytics, and the like all affect GCS. Therefore, where possible the GCS should be assessed both while the patient is sedated and not sedated. As with pulse and blood pressure, trending the GCS scores for a patient over time is especially helpful in the presence of neurologic insult.

In order to illicit a response from the patient, the least noxious stimulus required to illicit a response should be utilized. Deep nail bed pressure is a good starting point.

Decerebrate and decorticate posturing are ominous signs of upper brain stem or deep cerebral injury. Decerebrate posturing **Figure 5-9A** is characterized by hyperextension of the neck, stiff and extended extremities, and palmar flexion of the hands. This posturing results from a lesion in the brain stem. Decorticate posturing **Figure 5-9B** is characterized by arm flexion and adduction with fists clenched and

internal rotation of extended legs. This posturing is the result of a lesion at or above the upper brain stem.

Changes in the GCS score are critical to note because they may indicate increasing ICP. Often the first sign of increased ICP is a change in the LOC, perhaps restlessness or confusion. One must not confuse the effects of medications (such as for pain or sedation) with pathologic neurologic deterioration. For example, if a patient were medicated with lorazepam before transport, CCTPs should associate this with a decreased LOC. Late signs of increased ICP include elevated systolic blood pressure, profound bradycardia, and abnormal respirations (Cushing triad).

TABLE 5-3 Glasgow Coma Scale

Response	Reaction	Score
Eye opening response	Open spontaneously	4
	Open to speech	3
	Open to pain	2
	No response	1
Verbal response	Oriented to person, place, and time	5
	Confused	4
	Inappropriate	3
	Makes incomprehensible sounds	2
	No response	1
Motor response	Obeys verbal command	6
	Localizes pain	5
	Nonpurposeful movement	4
	Flexion: abnormal, decorticate posturing	3
	Extension: abnormal, decerebrate posturing	2
	No response	1

TABLE 5-4 Pupil Response

Pupil Finding	Possible Etiology
Unilaterally dilated, fixed, and nonreactive	Increased intracranial pressure May be normal in some patients Herniation of brain tissue Impending death
Bilaterally dilated, fixed, and nonreactive	Hypoxia Severe brain damage at the level of the midbrain
Bilaterally pinpoint and nonreactive	Narcotic use Severe brain damage at the level of the pons Ischemia

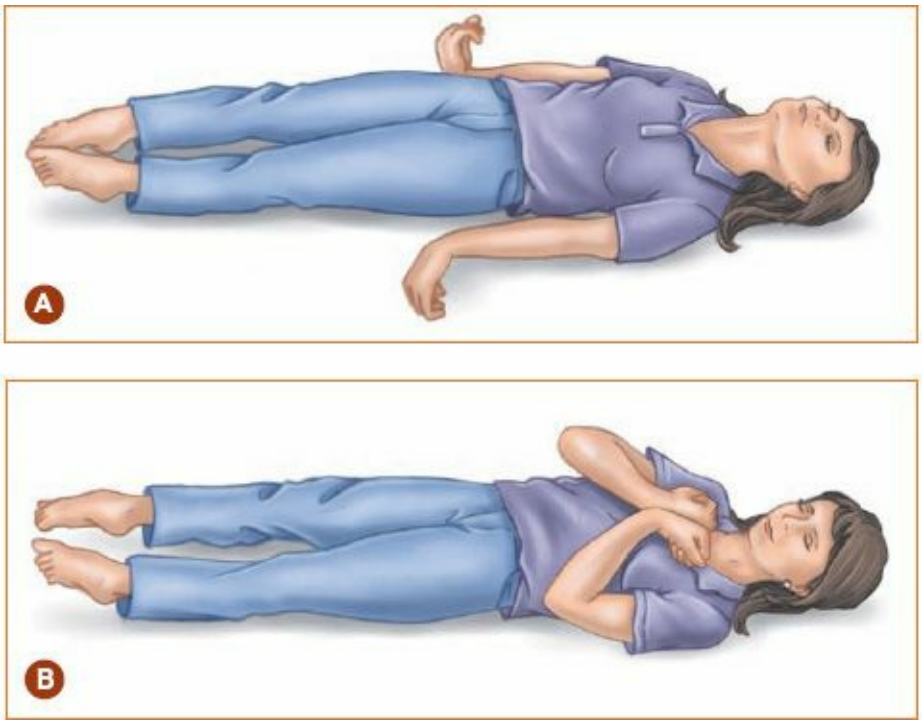


Figure 5-9 Posturing indicates significant ICP. **A.** Decerebrate posturing. **B.** Decorticate posturing.

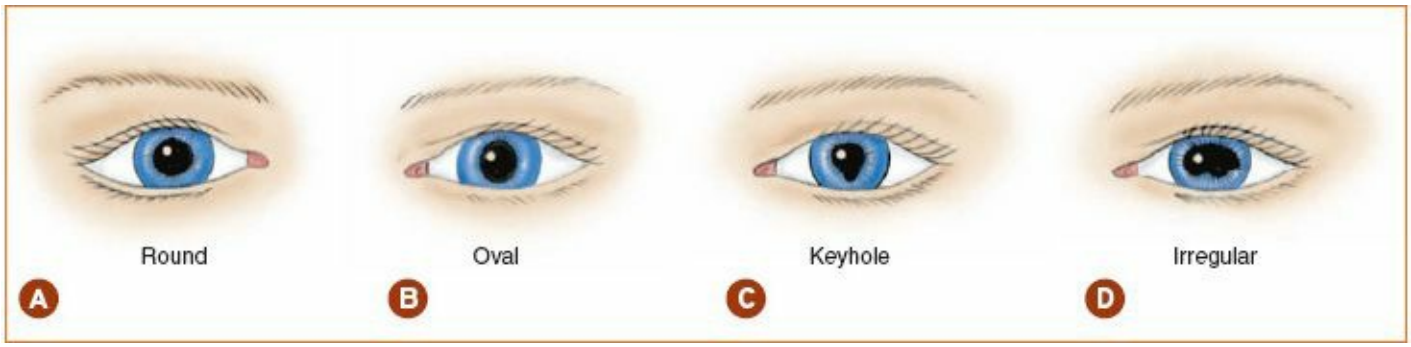


Figure 5-10 The four pupil shapes. **A.** Round. **B.** Oval. **C.** Keyhole. **D.** Irregular.

Another neurologic indicator of great concern is the pupil assessment. Pupils are assessed bilaterally for size (1 to 6 mm), reactivity to light (direct and consensual), and shape. **Table 5-4** lists common pupil findings related to size and reactivity and their possible causes.

Pupil reactivity to light is described as brisk, sluggish, or nonreactive. Occasionally, a CCTP will note a pattern of response in which there is rapid constriction of the pupil to light followed by dilation. This is called the **hippus phenomenon** and may be a variant of normal, but it is also seen with compression of cranial nerve III. **Anisocoria**, unequal pupil size bilaterally, is a normal variant and is seen in about 17% of the population.

There are four common shapes of pupils: round, oval, keyhole, and irregular **Figure 5-10**. Round is normal and the most common. An oval pupil is indicative of increased ICP and is often seen just before herniation of brain tissue. A keyhole pupil (resembling a keyhole in a door) is seen after iridectomies and usually reacts sluggishly. An irregular pupil is jagged looking and occurs most often after orbital trauma.

Unexplained changes in pupil assessment findings should be reported immediately. Consideration must be given to medications the patient is receiving (such as narcotics or cholinergics), anxiety, and other extraneous influences on pupils.

Finally, a cranial nerve assessment is valuable in a patient with a neurologic insult. Subtle changes may indicate impending herniation. Cranial nerve assessment is covered in detail in Chapter 11. Cranial

nerve III is especially helpful in assessing the patient with a neurologic insult. Changes in the extraocular movements and papillary response controlled by the nerve are often the first changes seen with an increase in intracranial pressure.

Controversies

The value of cranial nerve assessment is often downplayed. For many providers, assessment of cranial nerves is limited to preparation for certification or licensing examinations. In practice, cranial nerve assessment is an invaluable tool for localizing sources of sensory or motor dysfunction. Cranial nerve dysfunction can be an early warning sign for otherwise subtle but potentially significant neurologic deterioration but will be detected only if the CCTP is familiar with cranial nerve assessment.

Gastrointestinal Assessment

Inspection

A general overview of the oral mucosa and abdominal areas gives CCTPs general information about the gastrointestinal system. For example, the lips and mucous membranes should be moist and without lesions. Dry mucous membranes may indicate dehydration. A large abdomen may indicate ascites, especially if abdominal wall veins are noted. Hernias or masses may be visible. Bruising over the abdominal wall may indicate trauma or bleeding into the abdominal cavity. Abdominal girth is another assessment parameter used to document enlarging of organs or swelling from internal bleeding. The measurement should be taken at the same location each time. It is important to note that a chief complaint of "abdominal pain" is a major source of emergency department litigation claims.

Critical care patients often have gastrointestinal bleeding as a secondary stress response to their illness. Stools should be assessed for color, consistency, and a foul odor. Upper gastrointestinal bleeding is characterized by dark melena stools and lower gastrointestinal bleeding by stools mixed with bright red blood. Either of these findings should be reported promptly.

Auscultation

Important to note in gastrointestinal assessment is that auscultation of the abdomen occurs before palpation or percussion of the abdomen. The rationale is that one may get a false-positive assessment for bowel sounds if manual manipulation of the intestines occurs first. Bowel sounds should be present in all four quadrants of the abdomen. Bowel sounds may be hypoactive or hyperactive in various disease states. It is important to note, however, that there is great variation in bowel sounds with clinical conditions such as an illeus.

Percussion

Percussion is useful in establishing the size and location of the liver and sometimes the spleen. Percussion can be used to determine hepatomegaly. To percuss the liver, the CCTP should percuss upward on the midclavicular line from the level of the umbilicus. The tympany of the stomach will turn into dullness at the lower liver border. Percussion upward will then turn to resonance at the level of the lung. The distance between these two landmarks indicates liver span. If one performs percussion at the lowest left intercostal space and the sound is dull, the spleen is enlarged; this area is normally resonant.

Palpation

A CCTP typically uses palpation to document tenderness or rebound tenderness. Tenderness occurs with simple palpation; rebound tenderness is when the pain is reported as the pressure of palpation is

removed. Rebound tenderness suggests peritoneal inflammation. If a pelvic fracture is suspected, the pelvis should not be assessed for stability by every caregiver. The reason for this is that any hematoma that has formed to maintain hemostasis may be disrupted, causing internal hemorrhage to the patient.

Murphy's sign is noted when there is severe right upper quadrant pain on deep palpation, often exacerbated by deep inspiration and associated with cholecystitis. Deep palpation is performed by applying firm pressure to the abdominal wall. The liver may also be palpated by placing the left hand behind the patient under the liver and applying firm pressure upward. The fingers of the right hand are placed on the right upper quadrant of the patient's abdomen, and the patient is asked to take a deep breath. The edge of the liver can be felt as it comes down to meet the examiner's fingers. Next, the applied pressure is lightened and nodules or tenderness is noted as the liver slips under the fingers **Figure 5-11**. Nodules of the liver may be related to a malignancy. The spleen is generally not palpable. The right kidney may be palpated, but this examination is not of much clinical value. The kidney should never be palpated if there is a history of polycystic kidney disease.

Genitourinary Assessment

Assessment of the genitalia includes the mammary, testicular, and prostate glands. These assessments are limited to patients with specific needs, such as those with spinal cord injuries or trauma.

Proper kidney function is vital because the kidneys excrete the majority of toxins from the human body. The best indicator of kidney function is the serum creatinine level. The more nephrons (the functioning unit of the kidney) that have been destroyed, the higher the creatinine level.

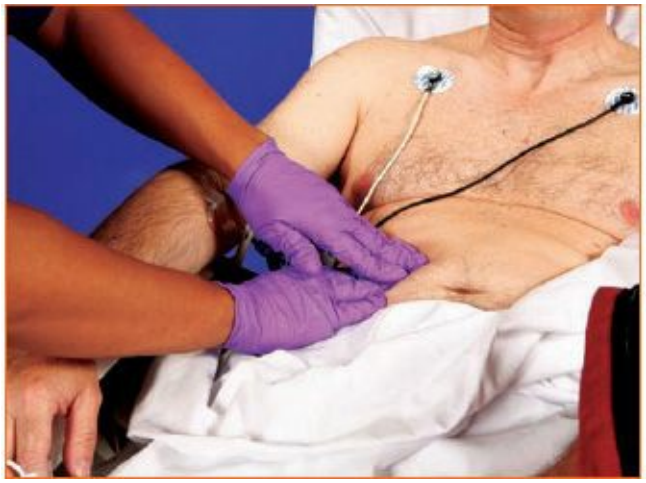


Figure 5-11 Palpation of the liver.

The **blood urea nitrogen (BUN)** value is more an indicator of hydration than of renal function. The BUN-creatinine ratio is important because it helps determine whether a patient has any type of renal failure. A normal ratio is in the range of 10:1 to 20:1. A normal creatinine level is typically 0.6 to 1.2 mg/dL; and a normal BUN level, 8 to 23 mg/dL. Before signs and symptoms of renal failure occur, 75% of the nephrons of the kidney can be destroyed.

Most often in critical care, renal insufficiency or renal failure is a secondary diagnosis. Because the kidney cannot clear the by-products of metabolism, patients with renal insufficiency or renal failure are prone to metabolic acidosis, congestive heart failure, and electrolyte imbalances. They are also prone to anemia because a diseased kidney does not produce erythropoietin, a hormone that stimulates red blood cell production. Monitoring of lab values, levels of arterial blood gases, fluid intake, and urine output is crucial in a patient with renal impairment.

Some patients are especially prone to renal failure because of burn and crush injuries. The reason is

that the release of large protein molecules from damaged tissue destroys nephrons. The patients often require large amounts of fluid to flush the molecules through the kidney.

In end-stage renal disease, patients may require peritoneal dialysis (PD) or hemodialysis. PD involves a catheter being placed in the peritoneum, where the peritoneum serves as the filter to remove waste from the peritoneal cavity using a dialyzing solution. The patients are especially prone to peritonitis. Therefore, an aseptic technique is important when assessing or performing PD. Patients undergoing hemodialysis will have an arteriovenous (AV) shunt, usually placed in the arm, that is attached to a machine that acts as a mechanical kidney to remove wastes from the body. This AV site should be assessed for a thrill (palpable) and a bruit (audible), which indicate a patent and normally functioning shunt. An absence of one or both of these may indicate clotting of the shunt. AV sites should be used only for hemodialysis. In patients who have a central line and tunneled line access for dialysis, these lines should not be accessed except in dire emergency and only while maintaining as strict sterile technique as possible. Additionally, these lines are typically heparin locked at the completion of treatments. Lines that contain heparin should not be flushed prior to aspiration of a significant amount of waste (minimum of 10 mL per port).

Musculoskeletal Assessment

A CCTP's main concern regarding the musculoskeletal system is maintaining stability of the joints and assessing for neurovascular compromise of distal extremities. Many times, a musculoskeletal concern in a critical care patient is a secondary diagnosis, such as in the case of a multisystem trauma patient.

Maintaining stability of a joint is done with a variety of splints or soft and hard casts. The joint should be maintained at the anatomic degree of flexion or extension found at rest or as ordered by the physician. CCTPs should assess for neurologic changes and vascular compromise related to the immobilizing device. Assessments should be completed bilaterally routinely. A common misconception is that only the operative or injured extremity should be assessed. Complete neurovascular checks include assessment of the distal skin color, nail bed color, skin temperature, mobility of distal joints, pulses, pain, and sensation.

Psychosocial and Emotional Assessment

If a patient has been hospitalized for more than 24 hours, it is likely that a complete psychosocial, emotional, and cultural needs assessment was completed. Information from this assessment should be transferred with the patient because it may be helpful to staff at the receiving facility.

Pertinent aspects relative to CCTPs would be a previously diagnosed psychiatric disorder, significant coping needs related to the present illness, and the level of anxiety the patient is experiencing. Psychiatric disorders may manifest in unsafe behaviors requiring physical or chemical restraint during transport. Significant coping issues or high levels of anxiety may interfere with adequate analgesia and/or require administration of anxiolytics or benzodiazepines.

A CCTP should not discount the therapeutic value of compassionate and empathetic interactions with a patient, even in the face of critical illness. Patients thought to have been deeply unresponsive, heavily medicated, or otherwise uncommunicative will often later recall providers who handled them gently, took the time to explain the care being provided, held their hand, and offered reassurance during a time of utmost stress and anxiety.

Communication and Documentation

Documentation of assessment findings should occur when the patient is first encountered, routinely as

patient condition warrants, and on arrival at the destination. Prudent CCTPs should diligently document findings to provide for continuity of care and patient safety and to protect themselves in case legal issues arise.

Communication should be ongoing with the transferring hospital, which is responsible for care of the patient until arrival at the receiving hospital. Changes should be called in immediately, orders obtained and implemented, and the response of the patient assessed and documented. Staff at the receiving hospital should also be made aware of events occurring en route so they are prepared to receive the patient.

Summary

Assessment skills are vital to CCTPs. Patients are classified as critical because they require frequent if not continuous monitoring. CCTPs are integral members of the health care team, and the continuum of care should be consistent with that provided in the acute care setting.

Assessments are done thoroughly and as frequently as warranted by the patient condition. When examined independently of one another, signs or symptoms hold little meaning. CCTPs must learn how to integrate the pieces of the puzzle into a large holistic view. Subtle changes in a patient or subtle changes in a finding over time may be significant. These subtle changes, if detected, could improve the patient's outcome, including saving the patient's life.

Finally, all assessment findings should be documented accordingly in the medical record. From a legal viewpoint, assessment and care that was not documented was not done.

Case Study

YOU AND YOUR CRITICAL CARE TRANSPORT TEAM are responding to an interfacility transport of a 48-year-old man who presented to the emergency department with severe chest pain. The patient was diagnosed with acute myocardial infarction and an intra-aortic balloon pump was inserted. The patient was admitted to the intensive care unit (ICU) awaiting transport to the specialty heart hospital. En route to the hospital, your crew receives an update on the patient's condition. You and your crew are informed that the patient's vital signs are as follows: blood pressure, 120/75 mm Hg; pulse rate, 110 beats/min; respiratory rate, 30 breaths/min; and pulse oximetry reading, 93%. The patient is on 4 L of oxygen by nasal cannula. He continues to complain of chest pain, rating his pain an 8 on a scale of 1 to 10. He is also complaining of severe back pain.

On your crew's arrival, you find the patient in the ICU. You and your team receive a report from the ICU nurse. She reports that the patient presented to the emergency department this morning around 8:30 complaining of severe chest pain. He was given an aspirin, started on a nitroglycerin drip, and administered a total of 5 mg of morphine. Blood was drawn for lab analysis. The ICU nurse hands you the lab results. You review the results and report the findings to the rest of your crew. The lab results were as follows:

- Calcium: 9.5 mg/dL (normal range, 8.2 to 10.2 mg/dL)
- Chloride: 100 mEq/L (normal range, 96 to 106 mEq/L)
- Magnesium: 2.0 mEq/L (normal range, 1.3 to 2.1 mEq/L)
- Potassium: 4.0 mEq/L (normal range, 3.5 to 5.0 mEq/L)
- Sodium: 142 mEq/L (normal range, 136 to 142 mEq/L)
- Hemoglobin: 14.4 g/dL (normal range, 14.0 to 17.5 g/dL)
- Hematocrit: 45% (normal range, 41% to 50%)

- White blood cell count: 10,400/μL (normal range, 4,500 to 11,000/μL)
- Platelet count: 210 × 10^3/μL (normal range, 150 to 350 × 10^3/μL)
- Creatine kinase-MB: 3.5 ng/mL (normal range, 0 to 7 ng/mL)
- Creatine kinase: 100 U/L (normal range, 40 to 150 U/L)
- Cholesterol: total, 198 mg/dL (normal range, 200 to 239 mg/dL); high-density lipoprotein, 278 mg/dL (normal range, < 40 mg/dL); low-density lipoprotein, 32 mg/dL (normal range, > 160 mg/dL); triglycerides, 205 mg/dL (normal range, < 160 mg/dL)

The arterial blood gas measurement results were as follows:

- pH: 7.30 (normal range, 7.35 to 7.45)
- $Paco_2$: 55 mm Hg (normal range, 35 to 45 mm Hg)
- HCO_3^-: 25 mEq/L (normal range, 21 to 28 mEq/L)
- Po_2: 93 mm Hg (normal range, 80 to 100 mm Hg)

An electrocardiogram (ECG) revealed sinus tachycardia with right axis deviation. As you enter the patient's room, you find the patient lying supine with 4 L of oxygen being delivered via nasal cannula. The patient is attached to the bedside cardiac monitor and the intra-aortic balloon pump (IABP). The cardiac monitor reveals a pulse rate of 130 beats/min, a blood pressure of 145/96 mm Hg, and a pulse oximetry reading of 93%. He is awake, oriented, and follows all commands. His skin is pale. His respiratory rate is 30 breaths/min.

Patient assessment reveals the following:

- **Cardiopulmonary assessment:** The patient's airway is patent, with a slight decrease in chest rise on the right side. Breath sounds are present bilaterally, but slightly diminished on the right side. No wheezes, crackles, or rhonchi were noted. Respiratory effort is labored. No subcutaneous emphysema is noted on palpation. Skin is pale and diaphoretic. Capillary refill is 4 s. No murmur, rubs, friction, or gallops were noted on auscultation of the heart. Pulses are equal in pattern, intensity, and quality in all four extremities. The ECG reveals sinus tachycardia with a rate of 133 beats/min. No pitting edema is noted to extremities.
- **Neurologic assessment:** The patient has a Glasgow Coma Scale score of 15. He is able to move all four extremities without difficulty. His pupils are equal, round, and reactive to light. All cranial nerves are intact.
- **Gastrointestinal assessment:** No ascites or bruising is noted on inspection of the abdominal area. The abdomen is soft, with no tenderness or distention on palpation. No masses or hernias are noted. No rebound tenderness is present. Auscultation of the abdomen reveals hyperactive bowel sounds. The patient denies having vomited blood or blood with a coffee-ground appearance and also denies having had dark, tarry stools.
- **Musculoskeletal assessment:** The patient is able to move all four extremities without difficulty. All reflexes are intact. No swelling to extremities is present. Pulses are present in all extremities.

1. What complications could be occurring with this patient?
2. What do the lab values, arterial blood gas results, and ECG reveal?
3. What is your general impression of this patient?
4. What additional information should you request from the hospital? Would you request any other test(s) prior to transport of this patient?

Analysis

The patient presented with severe chest pain that started 2 hours prior to his arrival at the emergency department. The patient stated that the pain occurred suddenly and nothing he could do relieved it. He described the pain as sharp, and originally rated it a 10 on a scale of 1 to 10. The emergency department physician made a diagnosis of acute myocardial infarction based on borderline lab values. An IABP was inserted and arrangements were made to transport the patient to a specialty heart hospital. The transport crew received the update that the patient was having severe back and chest pain.

The crew's first thoughts while en route to the patient included dissection of the aorta, which is a common complication after an IABP insertion. Classic presentation of an aortic dissection is chest and back pain. The crew discovered that a chest radiograph had not been obtained and asked for an updated chest radiograph. A chest radiograph should be performed before and after IABP insertion.

The lab results and ECG in this case did not confirm the physicians' diagnosis of a myocardial infarction. More testing was needed to confirm an accurate diagnosis. The general impression of this patient is that he is in respiratory distress. Further investigation is warranted because of the possibility of heart failure, respiratory infection, or pneumothorax. Assessment reveals the possibility of a pneumothorax, which must be investigated further, for example with the chest radiograph already requested.

In addition to the chest radiograph, the CCTP should consider requesting a determination of the patient's troponin level. Troponin is a cardiac enzyme specific to injuries of the cardiac muscle. Physicians diagnose myocardial infarction based on several factors, including an ECG, lab values, and patient presentation.

In terms of assessment and treatment, the patient has diminished breath sounds in the right side of the chest. He has labored respirations, and arterial blood gas determinations revealed respiratory acidosis, which are all classic signs of a pneumothorax and possible tension pneumothorax. In situations in which you believe more testing should be done to determine the diagnosis, the critical care team should discuss their assessment findings and ask whether a radiograph or other relevant test can confirm their assessment. Professional and nonconfrontational communication patterns are expected. Respectful behavior on the part of the CCTP, regardless of the situation, helps to promote positive working relationships, while disrespect or negativity may result in a referring facility no longer calling that particular CCT service. If the physician disagrees with your team, a team member should contact the medical director and have him or her contact the transferring physician to help determine the best course of action.

In this case, on the crew's arrival to pick up the patient, the relieving attending physician stated that chest radiography revealed a pneumothorax. After chest tube insertion, the patient was transported to the specialty facility and was evaluated. No cardiac problems were found.

Prep Kit

Ready for Review

- The assessment process ensures proper discovery and management of a patient's critical care needs and improves outcomes.
- CCTPs should be familiar with the assessment process used to establish the initial care plan and have an understanding of pathophysiology and differential diagnoses to be able to make treatment decisions and assess the effects of treatments.

- The assessment process should combine the paramedic model of assessment, which treats major life-threatening problems during the assessment process, and the medical or nursing model of assessment, which targets treatment to underlying problems.
- The systems-based assessment approach must recognize that critical care patients are usually receiving treatment for problems involving multiple systems and interrelated complications.
- Patient assessment should include the use of subjective and objective data and performance indicators to measure physiologic status.
- Assessment ideally includes bedside clinical observations, listening to patients and families, and balancing personal and clinical observations with findings through the use of technology.
- The assessment process differs for scene and interfacility transports.
- At scene transports, CCTPs should ascertain as many details from the scene as possible, interact and communicate with other providers, and defer complex assessment and treatments until arrival at a care facility.
- CCTPs should note interventions provided before their arrival and clearly document patient response to these interventions.
- Multiple variables such as patient location, care in progress on arrival, temperature and weather conditions, and the type of transport vehicle inform the process of patient packaging.
- Local protocols and procedures dictate the sequence of packaging and moving a patient for transport and the methods used to secure lines, tubes, and equipment before and during an interfacility transport.
- For interfacility transports, assessment begins on entry to the sending facility with an overview of the surroundings and routes of travel to ensure safe and feasible transport of the patient, equipment, and personnel to the transport unit.
- CCTPs should review all available data and patient information before their arrival and use anticipated transport time as a consideration for differentiating necessary information from information that is helpful but nonessential.
- In interfacility transports, nurse-patient dynamics should be considered. A patient may experience separation anxiety and increased stress during the transfer.
- CCTPs should scan the scene at the transferring facility for obvious signs of the patient's condition, note medication infusions, and document settings of ventilators and monitoring equipment.
- Patient assessment should include evaluation of each therapy before and after transfer to the transport unit.
- At the transferring facility, CCTPs should inquire about treatments and previous responses to current treatments, determine patient status (for example, stable or unstable), and learn if there are any advance directives in place.
- Patients requiring interfacility transport have often undergone extensive prior interventions; it is important to keep treatment focused on the principal concern that necessitated transport.
- CCTPs must determine if it is necessary to divide tasks (for example, one provider obtaining a report from the transferring facility and the other conducting a patient assessment).
- To help maintain a balanced, holistic approach to the patient and the situation, CCTPs should request a general patient overview and medical record.
- Critical care assessment uses observational skills and clinical parameters of physiologic status to assess general appearance: cardiovascular, respiratory, neurologic, gastrointestinal, genitourinary, and

musculoskeletal systems; and psychosocial and emotional state.

- Assessment of each system and the data needed must be individualized to each patient and his or her specific condition.
- In addition to the exam, CCTPs should always assess and validate differential diagnoses and obtain subjective assessment data from the patient and family.
- Evaluation of general appearance includes an assessment of the patient's overall apparent health status, apparent age relative to chronological age, and level of consciousness.
- Cardiovascular physical assessment includes consideration of the patient's skin color (central and peripheral), making note of any pallor or cyanosis of the lower extremities, redness, cyanosis of the nail beds, clubbing, or edema.
- Cardiovascular assessment must include ECG rhythm interpretation and consideration of heart rate, rhythm, and a history of arrhythmia.
- Invasive monitoring equipment, while helpful, may not be necessary, because skilled providers can obtain substantial data by using assessment skills.
- Carotid, radial, brachial, femoral, popliteal, posterior tibial, and dorsalis pedis pulses should be assessed bilaterally for presence, strength, and pattern.
- CCTPs should consider trending of pulse assessment findings over time and patterns of pulsations.
- In the cardiovascular assessment, the heart should be auscultated at the aortic, pulmonic, tricuspid, and mitral valve locations with the diaphragm and bell of the stethoscope.
- Auscultation should be performed over the carotid, renal, and femoral arteries for bruits.
- Blood pressure can be a valuable clinical parameter, but it is important to remember that normal blood pressure is defined by the individual patient. Further, be cautious with the overuse of machines to take blood pressure. There is a margin of error with these machines, especially in hypovolemic states.
- Before transport, trends in blood pressure over time should be noted, especially in response to cardiogenic medications and interventions.
- Vital sign frequency is dictated by the condition of the patient and may range from continuous to every 15 minutes.
- Passive leg raising should be used to assess fluid responsiveness in patients with suspected volume depletion.
- Although not all parameters are assessed on all patients, it is an accepted critical care standard that a head-to-toe assessment be completed at least every 4 hours.
- A CCTP should develop a comfortable and familiar assessment sequence to reduce omissions of parameters and improve time management.
- Assessment of the respiratory system involves visual examination of the patient with emphasis on the chest and evaluation of work of breathing by consideration of use of accessory muscles, intercostal retractions or bulging, and nasal flaring.
- Palpation may yield important information about the skin and subcutaneous tissues.
- Additional respiratory assessment parameters include palpation of the trachea for alignment, palpation of the chest wall for bilateral chest excursion, and palpation for subcutaneous emphysema.
- When radiography is unavailable, percussion may be a valuable assessment tool, although it requires strong knowledge of anatomy, physiology, and technique.

- The ability to differentiate percussion sounds comes with practice and experience. Normal lung fields produce resonance; providers should note dullness or flatness (which may indicate fluid or tumors) or hyperresonance (which may indicate an air-filled, emphysematous lung).
- Lungs should be auscultated from the front and back, at the right and left mid-axillary lines, and over the trachea and bronchi.
- All lung fields should be assessed for the presence of normal breath sounds, such as tracheal, bronchial, vesicular, and bronchovesicular, and for adventitious sounds, such as crackles, rhonchi, stridor, and wheezing.
- When possible, one side of the chest wall should be compared directly with the opposite side.
- When transporting a patient who is mechanically ventilated, it is important to note current ventilator settings and parameters.
- Before critical care transport, a neurologic exam is necessary to establish the patient's baseline status for comparison with the findings of later examinations.
- Neurologic evaluations should be repeated frequently, noting the level of consciousness, Glasgow Coma Scale score, cranial nerve assessment, and pupil assessment, including size, reactivity to light, and shape.
- Neurologic assessment should also include evaluation of posturing and of sensory and motor function in each extremity, comparing both sides.
- Gastrointestinal assessment begins with a general overview of the oropharynx and abdominal areas, noting lesions, swelling, discoloration, and girth.
- Providers should assess stool for color, consistency, presence of blood, and odor.
- Unlike evaluation of other systems, in gastrointestinal assessment, auscultation of the abdomen precedes palpation and percussion.
- Percussion is useful in establishing the size and location of the liver and spleen.
- Palpation can determine tenderness and rebound tenderness.
- Genitourinary assessment includes assessments of the mammary, testicular, and prostate glands. These are generally limited except in the case of trauma.
- The focus of musculoskeletal system assessment is maintaining stability of the joints and considering neurovascular compromise of distal extremities.
- Psychosocial, emotional, and cultural assessment may have been completed before arrival of CCTPs and includes information such as previously diagnosed psychiatric disorders, significant coping needs related to the present illness, and levels of anxiety.
- Regardless of the patient's conditions, CCTPs should always strive for compassionate and empathetic interactions with patients and families, even in the face of critical illness.
- Assessment findings before transfer, en route, and after arrival (based on the acuity of the patient) should be documented.
- The medical record should include documentation of all assessment findings; even subtle changes in an individual patient or an individual finding may become significant.

Vital Vocabulary

anisocoria A condition in which the pupils are not of equal size.

bleb A blister or bladderlike structure that may be filled with fluid.

blood urea nitrogen (BUN) A test used to measure urea, which is a biomarker for adequate kidney function.

differential diagnosis A list of all possible diagnoses, aside from the projected diagnosis, that could be causing a patient's symptoms.

hepatojugular reflex A reflex tested in assessment in which midabdominal pressure is applied while observing for jugular venous distention; if noted, the test result is positive and indicates volume overload.

hippus phenomenon A pattern of pupil response to light in which there is rapid constriction of the pupil followed by dilation; can be normal or signify compression of cranial nerve III.

hyperresonance An abnormal respiratory sound that is exaggerated or increased beyond normal and low in pitch.

morbidity Number of nonfatally injured or disabled people; usually expressed as a rate, meaning the number of nonfatal injuries in a certain population in a given period divided by the size of the population.

mortality Deaths caused by injury and disease; usually expressed as a rate, meaning the number of deaths in a certain population in a given period divided by the size of the population.

Murphy's sign Pain when pressure is applied to the right upper quadrant of the abdomen in a specific manner; helps detect gallbladder problems.

passive leg raising (PLR) A technique used to assess fluid responsiveness in patients with suspected volume depletion, performed by raising both of the patient's legs to a 45° angle and then taking the blood pressure; improvement in blood pressure suggests that the patient will benefit from fluid administration.

projected diagnosis An assumed decision on the medical condition of a patient from preliminary investigation; assists in the provision of initial treatment and projection of further diagnostic testing requirements.

resonance Normal lung sound heard with percussion.

turgor The elasticity of the skin.

tympany A loud, high-pitched, abnormal lung sound.

Bibliography

Alspach J. *Instructor's Resource Manual for the AACN Core Curriculum for Critical Care Nursing,* 5th ed. Philadelphia, PA: WB Saunders Co; 2001.

Basile C, Buchda V, Dietrich TR, et al. *Mastering Advanced Assessment.* Springhouse, PA: Springhouse Corp; 1993.

Bickley LS. *Bates Guide to Physical Examination and History Taking,* 8th ed. Philadelphia, PA: Lippincott; 2003.

Boulain T, Achard JM, Teboul JL, et al. Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. *Chest.* 2002; 121; 1245–1252.

Grimes J, Burns E. *Health Assessment in Nursing Practice,* 2nd ed. Boston, MA: Jones and Bartlett Publishers; 1987.

Hickey JV. *The Clinical Practice of Neurological and Neurosurgical Nursing,* 4th ed. Philadelphia, PA: Lippincott; 1997.

Respiratory Emergencies and Airway Management

Authors

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Objectives

1. Summarize the anatomy and physiology of the respiratory system (p 128–134).
2. Define the five requirements for normal ventilation and oxygenation (p 134).
3. Discuss basic airway management strategies (p 141–145).
4. Differentiate between obstructive and restrictive diseases (p 135).
5. Assess a respiratory cycle (p 139).
6. Identify and interpret normal and abnormal breath sounds (p 130).
7. Identify abnormal respiratory patterns (p 137–139).
8. Review the breath sound assessment technique used in critical care transport patients (p 137).
9. Define tidal volume, vital capacity, and peak flow (p 134).
10. Describe how pulse oximetry, capnometry, and capnography can be used to monitor respiratory function (p 140).
11. Identify the parameters measured in arterial blood gas monitoring and identify which parameter reflects the effectiveness of ventilation and oxygenation (p 140).
12. Describe particular clinical events that alter the functioning of the respiratory system in the critical care patient (p 135).
13. Describe how ventilation and perfusion abnormalities affect blood gas values (p 131–133).
14. Perform a basic respiratory assessment for adequacy of ventilation and oxygenation using inspection, auscultation, palpation, and noninvasive monitoring (p 137, 139).
15. Differentiate invasive and noninvasive ventilation (p 174).
16. Differentiate positive- and negative-pressure ventilators (p 173).
17. Differentiate pressure, volume ventilators, and flow- and time-cycled ventilators (p 173–174).
18. Define the parameters of tidal volume, fraction of inspired oxygen, respiratory rate, peak flow, ratio of the length of expiration to inspiration (I:E) ratio, mode, and positive end-expiratory pressure (PEEP) (p 134, 136).
19. Identify the components of a ventilator circuit (p 171).
20. Define the various modes of mechanical ventilation (p 175).
21. Explain the use of PEEP (p 174).
22. Troubleshoot low pressure (disconnect), high pressure, power failure, and other common alarms (p 174).

Introduction

There is no more important or controversial skill associated with prehospital care than airway management. No other skill has the potential for such an impact, positive or negative, on patient outcome. The ability to assess and manage the airway at the BLS level is the starting point in airway management. As ALS providers, it is a standard to fall back on. Assessment of airway and breathing is the critical first step in the ABCs of resuscitation. This assessment will guide you in developing a treatment plan that may be as simple as providing supplemental oxygen to a spontaneously breathing patient or as complex as placing an artificial airway device, or providing adequate ventilation with a bag-mask device or ventilator.

Ensuring the adequacy of ventilation and oxygenation in critically ill patients is one of the primary treatment goals of the CCTP. Patient history, visual inspection of the chest, vital signs, auscultation, and the blood gas measurements all provide key information about the functioning of the respiratory system. A thorough understanding of respiratory function and assessment is essential in order to meet this goal. Without these basic skills, you will be unable to provide the necessary intervention when a patient's respiratory status deteriorates during transport. When respiratory compromise occurs in your patient, time is critical, and the CCTP must be prepared to perform a rapid, accurate respiratory assessment followed by the appropriate intervention.

Anatomy and Physiology of the Respiratory System

An understanding of airway anatomy and physiology is essential to successful airway management.

Upper Airway Structures

Important airway structures of the upper airway include the nose, mouth, pharynx, and larynx, which contains the epiglottis.

Nose

The nose is a cartilaginous, bony structure in the midline of the face that warms and humidifies inspired air. The nose is lined with coarse hairs in the vestibular area, which act as a filter to trap small inspired particles. Olfaction (sense of smell) also originates here via the first cranial nerve (olfactory nerve). The nasal cavities connect to the four sinuses: frontal, ethmoidal, sphenoidal, and maxillary. These four sinuses are hollow chambers lined with membranes that secrete mucus into the nasal cavities. The nasal cavity subsequently opens into the nasopharynx.

The nose, which is extremely vascular (Kisselbach's plexus), can be a significant source of epistaxis, which may complicate airway patency and management.

Mouth

Primarily designed for phonation and mastication, the mouth begins at the lips and ends with the oropharynx. The size of the oral cavity (the larger the better) can affect airway management **Figure 6-1**. The mouth contains the tongue, which is attached to the mandible, and the teeth. Both of these structures may make airway management more difficult if edema of the lingual or sublingual spaces is present. Additionally, prominent central incisors (buck teeth) may also make airway management more difficult, as this may interfere with insertion of the laryngoscope blade. Salivary glands continuously secrete saliva, which may make topical anesthesia and visualization of airway structures difficult. Remember, the tongue is the most common cause of airway obstruction in the unconscious patient. A simple jaw thrust maneuver may temporarily alleviate airway obstruction and allow effective bag-mask ventilation.

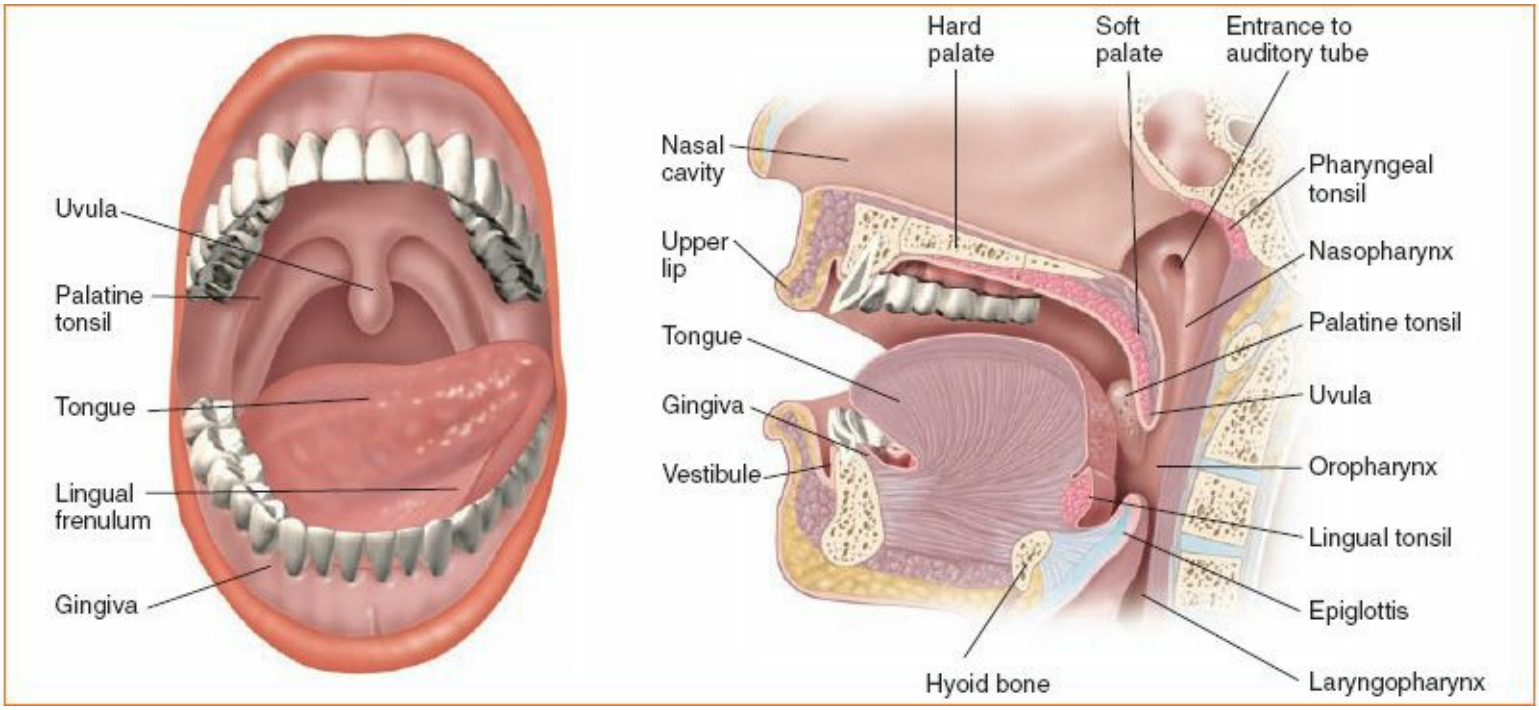


Figure 6-1 The oral cavity.

Pharynx

The pharynx is a U-shaped tube that begins at the base of the skull and extends to the lower border of the cricoid cartilage near the esophagus **Figure 6-2**. It is composed of three parts: the nasopharynx, the oropharynx, and the hypopharynx (or laryngopharynx):

- Nasopharynx: Extends from the posterior choane (the posterior portion of the nasal cavity) of the nose to the soft palate and contains the adenoid tissue and the eustachian tubes.
- Oropharynx: Extends from the soft palate to the vallecula. It is the portion that is visible when the mouth is opened.
- Hypopharynx: The portion of the pharynx inferior to the epiglottis.

The normal resting muscle tone of the oropharynx maintains upper airway patency. The ninth cranial nerve, the glossopharyngeal, provides sensory innervation to many of the structures in this area (posterior tongue, valleculae, and parts of the epiglottis). The inferior portion of the pharynx ends with the entrance to both the trachea and the esophagus.

Larynx

Structures contained within the larynx are the thyroid cartilage, aryepiglottic folds, epiglottis, vallecula, and arytenoid cartilages **Figure 6-3**. The larynx is the final structure before entering the trachea. The vagus nerve, a branch (superior laryngeal) of the 10th cranial nerve, provides strong sensory innervation to the larynx. Stimulation of the larynx may cause significant sympathetic stimulation and result in significant increases in heart rate and blood pressure. The cricothyroid membrane is also located here, extending from the lower surface of the cricoid cartilage to the upper border of the thyroid cartilage. In the adult patient, the cricothyroid membrane is approximately 6 to 8 mm from superior to inferior border.

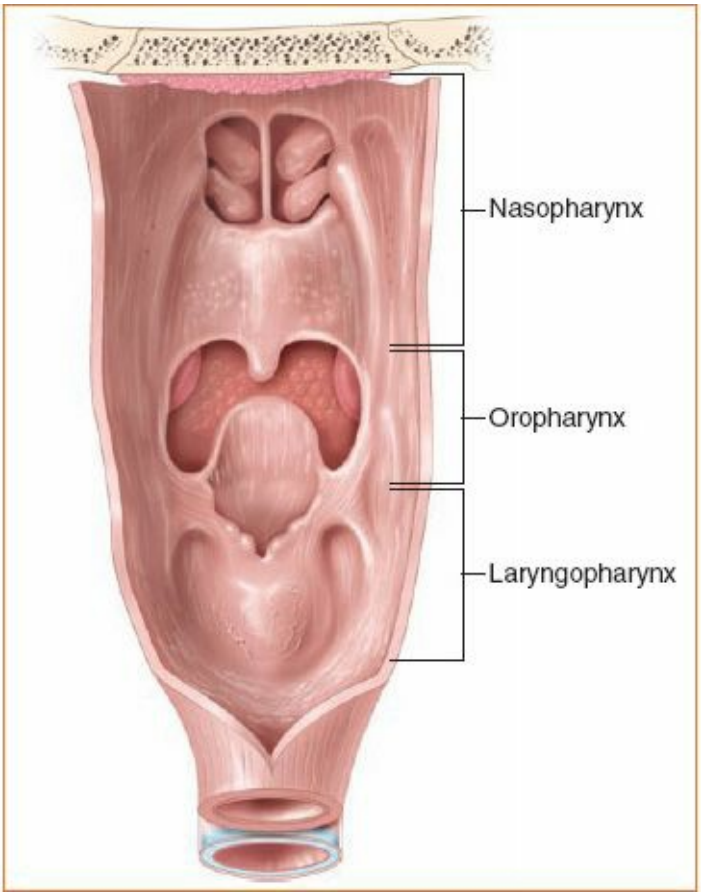


Figure 6-2 The pharynx.

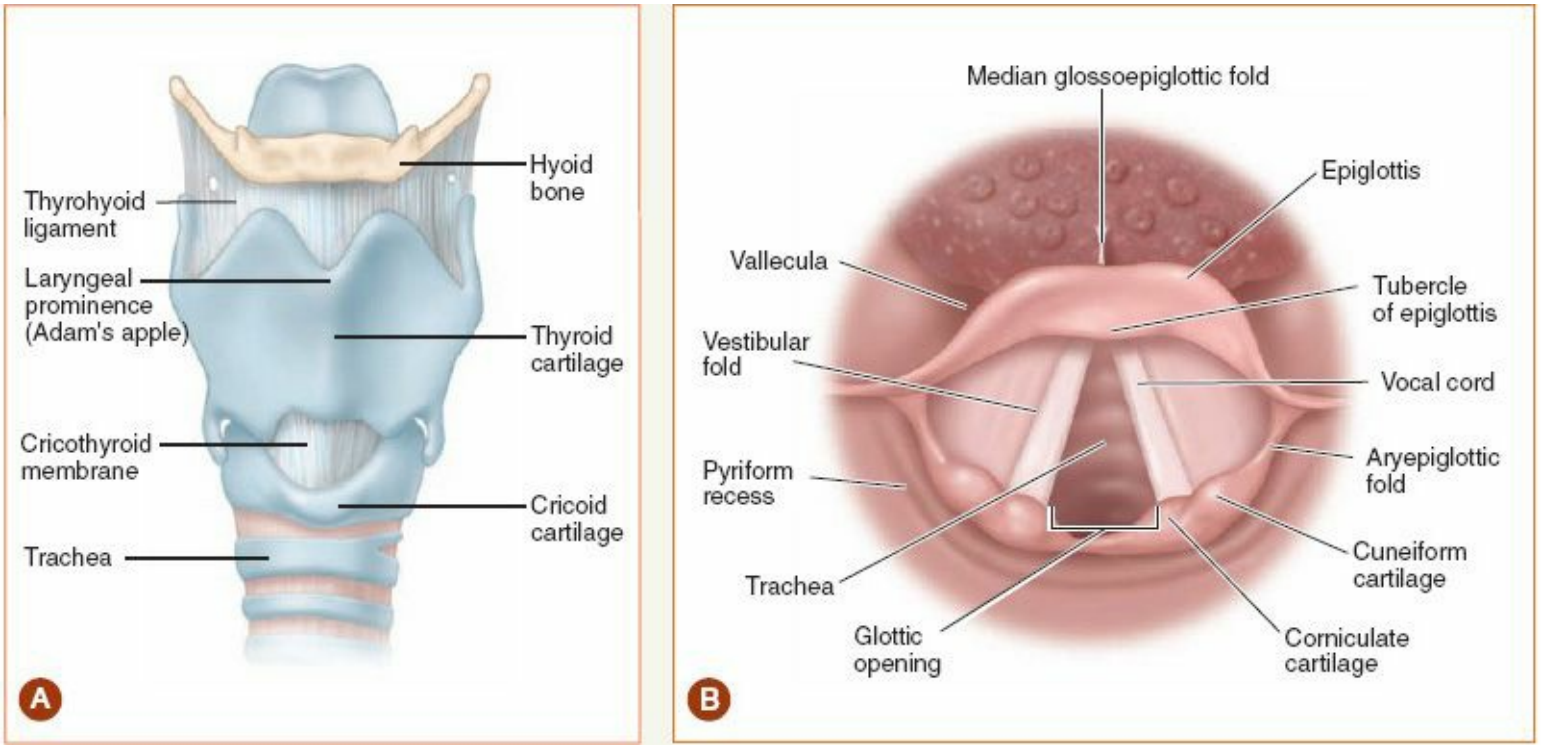


Figure 6-3 **A.** The larynx. **B.** The glottis and surrounding structures.

Lower Airway Structures

Important structures of the lower airway include the trachea, right and left main stem bronchi, bronchioles, alveolar ducts, and sacs.

Trachea

Beginning at the inferior border of the cricoid ring and ending at the carina, the trachea is approximately 9 to 15 mm in diameter and approximately 12 to 15 cm long. Not quite cylindrical, it is composed of 16 to 20 C-shaped cartilaginous rings on the anterior portion, with the posterior portion being fibrous tissue and muscular fibers. These rings help maintain luminal patency to allow unimpeded air flow. The trachea is larger in adult males than in adult females.

Lungs

The trachea divides into the right and left mainstem bronchi. These bronchi lead to the right and left lobes of the lungs **Figure 6-4**. The right lung is divided into three lobes; the left lung has two lobes. The bronchi continue to divide into smaller bronchi, which then divide into progressively smaller branches called bronchioles. After a total of 23 divisions into smaller and smaller branches, the bronchioles end at the alveolar ducts. These alveolar ducts lead to the alveoli, the area where gas exchange occurs. The adult lung contains about 300 million alveoli, each one in contact with a pulmonary capillary. This interface is referred to as the alveolar capillary (AC) membrane. An alveolus is made up of type I and type II squamous epithelial cells. Type I cells are involved in gas exchange, whereas type II cells manufacture surfactant. Surfactant is an important phospholipid that reduces the surface tension within the alveolus, preventing collapse and making it easier for the alveoli to expand during inhalation. Without surfactant, the work of breathing would be significantly more difficult. A lack of surfactant is commonly found in premature neonates and is a leading cause of respiratory issues in this patient population. Another type of cell, the alveolar macrophage, is also present; its function is to help defend the body by ingesting inhaled particles.

The blood supply in the lung arises from the right ventricle. The pulmonary trunk then splits into the right and left pulmonary arteries. The pulmonary arteries contain deoxygenated blood that is high in carbon dioxide (CO_2). The arteries subdivide into the pulmonary capillaries. This vasculature is a low-pressure system. The pressure in the normal pulmonary artery is approximately 25/10 mm Hg as compared with a normal systemic pressure of approximately 120/80 mm Hg. In order for gas exchange to occur, there must be blood flow in contact with the alveoli. This is defined as the $\dot{V}/\dot{Q}$ **ratio**. The normal ratio is 0.8 for the entire lung. This means that for every 4 L of air ($\dot{V}$), there must be a blood flow ($\dot{Q}$) of 5 L. Gravitational effects cause perfusion to be better in areas that are positioned lower (dependent areas) and ventilation to be better in other (nondependent) areas. Changes in the $\dot{V}/\dot{Q}$ ratio are one of the most common causes of hypoxemia. Disruptions can include changes in gravity, pulmonary artery pressure, alveolar pressure, airway obstruction, and lung compliance. Many diseases can cause these changes, such as chronic obstructive pulmonary disease (COPD), pulmonary embolism, heart failure (HF), and pneumonia, to name a few.

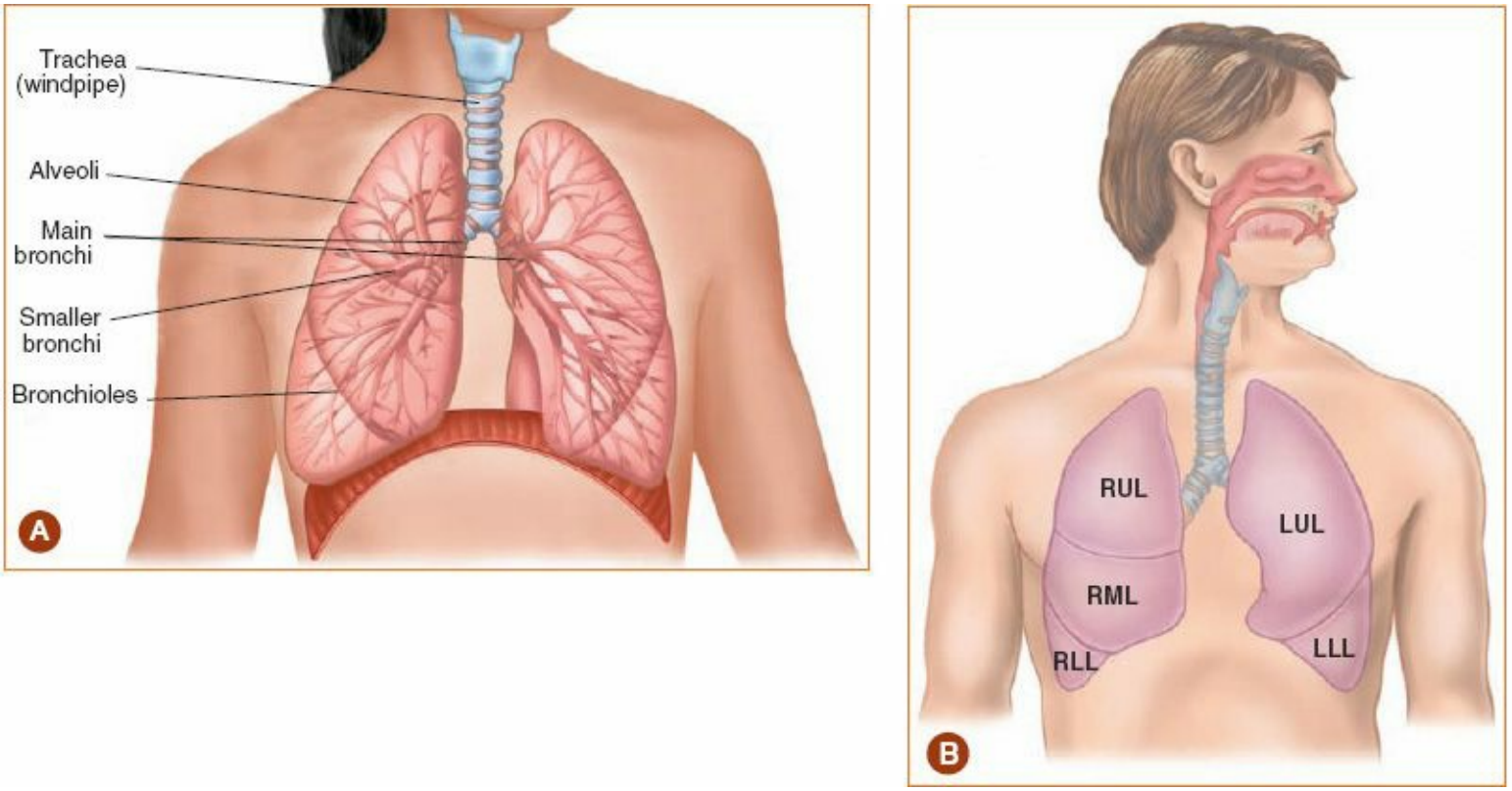


Figure 6-4 **A.** The respiratory system. **B.** The lobes of the lungs: RUL, right upper lobe; RML, right middle lobe; RLL, right lower lobe; LUL, left upper lobe; LLL, left lower lobe.

Pediatric Considerations

There are several variations in airway anatomy specific to the pediatric patient. Newborns and infants have a proportionally larger head with a prominent occiput. This may cause flexion of the airway and difficulty in visualizing laryngeal structures unless proper positioning is maintained (the use of a towel under the infant's shoulders to raise the rest of the body and straighten the airway usually improves airflow). Infants are obligate nose breathers, and congestion may result in respiratory distress. The tongue occupies a much larger proportion of the mouth in pediatric patients compared with adults. This difference in tongue size may result in airway obstruction in the unresponsive pediatric patient and make visualization of the glottis more difficult when performing laryngoscopy. The glottic opening is more cephalad and anterior in the pediatric patient. A child's epiglottis is at a 45° angle to the anterior pharyngeal wall, whereas an adult's lies parallel to the base of the tongue. The epiglottis is proportionally larger, floppier, and U-shaped compared with an adult **Figure 6-5** and may necessitate use of a straight blade to lift the epiglottis to improve visualization during laryngoscopy. Larger adenoidal tissue, particularly in infants, may result in significant hemorrhaging when traumatized. The cricothyroid membrane is small in children younger than 4 years, which may make needle cricothyrotomy difficult and surgical cricothyrotomy impossible (and contraindicated). There is one additional difference in the pediatric population—children have significantly higher oxygen consumption rates when compared with adults. Oxygen consumption in the pediatric patient is approximately double that of an adult. This increased consumption, coupled with a decreased functional residual capacity, can result in significant hypoxemia despite adequate preoxygenation. With pediatric patients, make sure that you are not overzealous in using the bag-mask device. Excessive volumes exacerbate gastric distention, causing vomiting, and increase the risk of pneumothorax.

Special Populations

Needle and surgical cricothyrotomy may be difficult or impossible in children younger than 4 years due to the small size of the cricothyroid membrane.

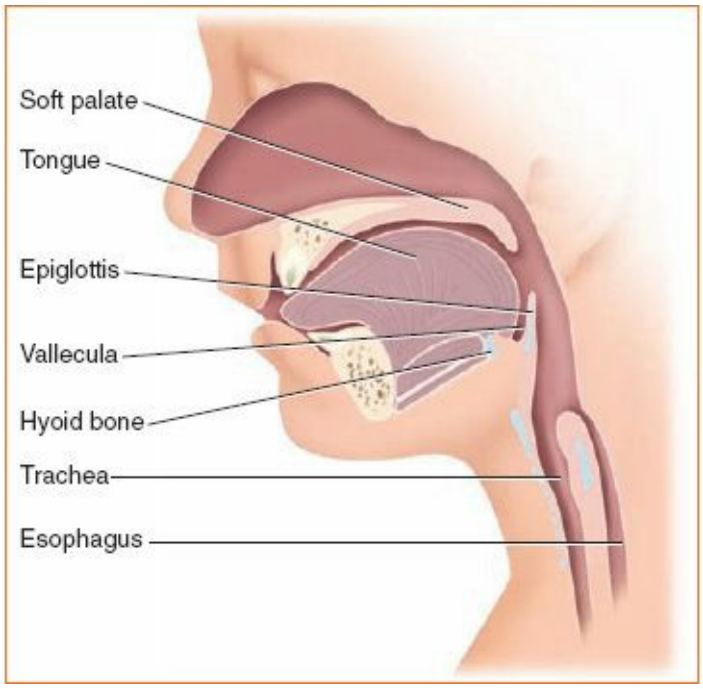


Figure 6-5 The child's epiglottis and surrounding structures.

Special Populations

Children have significantly higher oxygen consumption rates when compared to adults.

Physiology of the Respiratory System

Gas exchange occurs as a result of pressure gradient changes across the AC membrane **Figure 6-6**. In the capillary, the partial pressure of oxygen (PO2) is 40 mm Hg, whereas the partial pressure of carbon dioxide (PCO2) is 45 mm Hg. In the alveolus, the P_{CO_2} is 40 mm Hg and the P_{O_2} is 100 mm Hg. As the gases diffuse they move from an area of higher concentration to an area of lower concentration, seeking equilibrium. The end result is that the blood returning to the left side of the heart via the pulmonary vein contains 100 mm Hg of oxygen and 40 mm Hg of CO_2. (These are normal **arterial blood gas (ABG)** values.) Keep in mind that CO_2 diffuses 20 times faster than oxygen.

Factors that can affect the diffusion of gases across the membrane are known as **ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch**. $\dot{V}/\dot{Q}$ mismatch can be due to inadequate ventilation, perfusion, or both. There are three types of $\dot{V}/\dot{Q}$ mismatch: low, high, and silent.

- Low $\dot{V}/\dot{Q}$ ratio: characterized by perfusion exceeding ventilation. The low ratio results in blood being shunted past the alveoli without adequate gas exchange. A 20% shunting results in severe hypoxia. Causes include pneumonia, atelectasis, tumor, and mucous plug.
- High $\dot{V}/\dot{Q}$ ratio: characterized by ventilation exceeding perfusion, resulting in dead space. The alveoli are inadequately perfused, thus preventing adequate gas exchange from occurring. Causes of high $\dot{V}/\dot{Q}$ ratio include pulmonary embolus, pulmonary infarction, and cardiogenic shock.
- Silent unit: characterized by the result of decreased ventilation and perfusion as seen in patients with

a pneumothorax and severe acute respiratory distress syndrome (ARDS).

Although it is common to assess oxygenation in terms of the partial pressure of oxygen (arterial) (Pa_{O_2}), hemoglobin is the iron-containing protein found in erythrocytes that functions as the transporter of oxygen to tissues. The Pa_{O_2} drives oxygen to be bound to hemoglobin. The relationship is expressed in the oxyhemoglobin dissociation curve **Figure 6-7**. A patient can be experiencing tissue hypoxia if anemia or dysfunctional hemoglobin is present even though the Pa_{O_2} and oxygen saturation are normal. Many substances can bind to hemoglobin and cause it to be dysfunctional. Carbon monoxide, for example, has 240 times the affinity for hemoglobin than oxygen. Carbon monoxide binding to hemoglobin results in the formation of carboxyhemoglobin. The formation of carboxyhemoglobin prevents oxygen from being transported and released to the tissues. Nitrite poisoning can result in the formation of abnormal hemoglobin. Nitrite binds to hemoglobin, resulting in methemoglobinemia. This also alters the oxygen-carrying capacity of the hemoglobin molecule and can result in hypoxia.

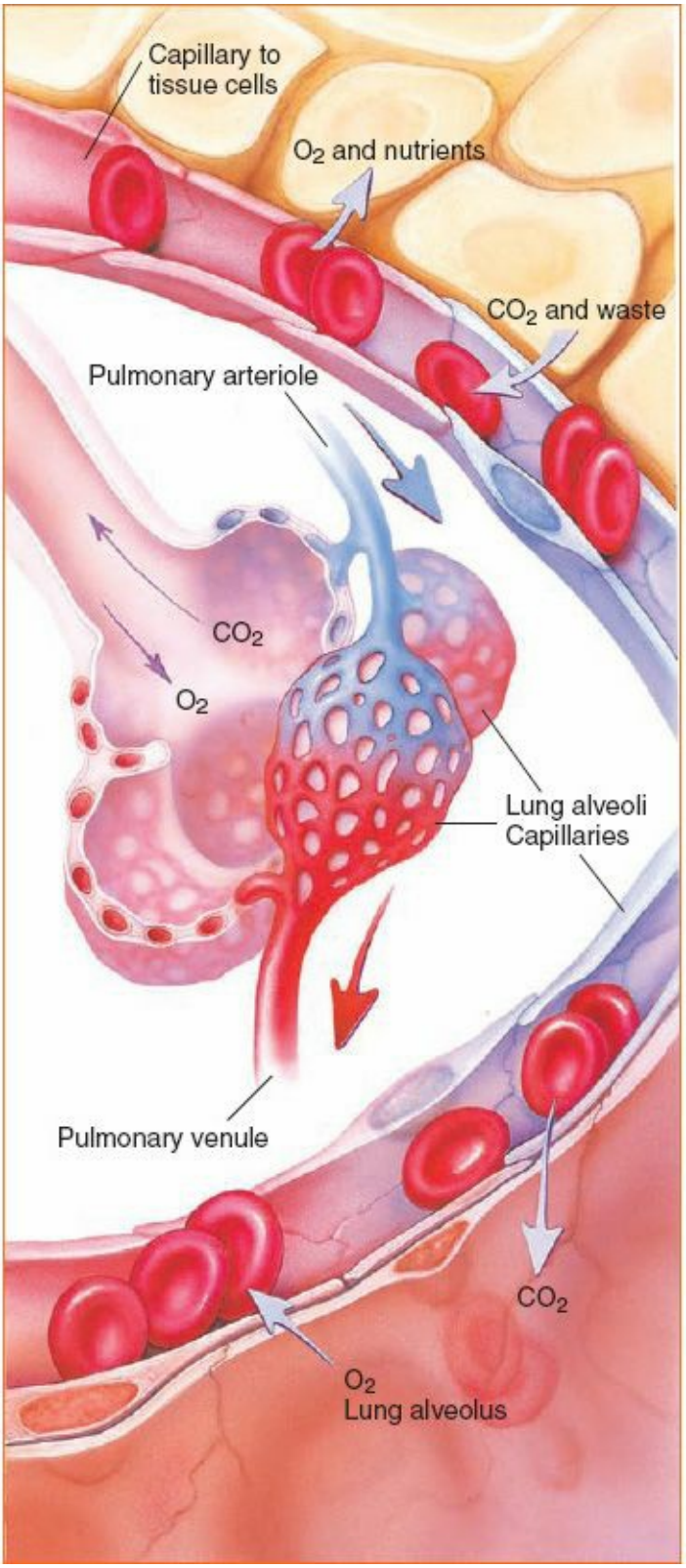


Figure 6-6 With diffusion, molecules of oxygen (O_2) move from the alveoli into the blood because there are fewer O_2 molecules in the blood. Similarly, molecules of carbon dioxide (CO_2) diffuse from the blood into the alveoli because there are fewer CO_2 molecules in the alveoli.

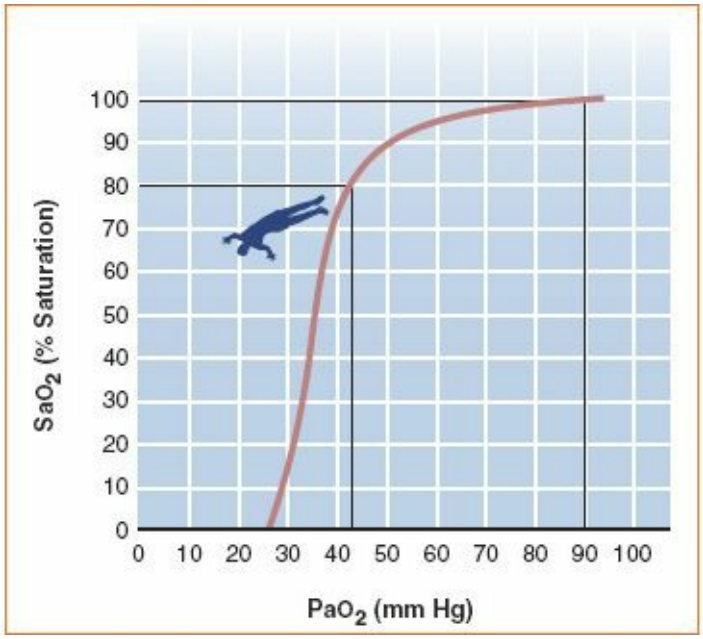


Figure 6-7 Oxyhemoglobin dissociation curve.

The mechanics and dynamics of breathing involve the concepts of elastance, compliance, resistance, and pressure gradients. Lung tissue, in an independent state, would tend to collapse, a condition known as elastance. The construction of the bony thorax and ribs creates an opposing force that prevents the lung tissue from collapsing completely. The ease with which the thorax and lungs expand is referred to as compliance. If compliance is reduced, it is harder for the lungs to expand; therefore, any process that affects the integrity of this relationship results in increased work to expand the lungs. For example, if the chest wall becomes rigid due to pain, injury, or disease, it becomes more difficult to expand and breathing becomes more difficult. If the lung parenchyma becomes diseased and becomes stiffer, more work is needed as well.

We can define compliance as a change in volume per unit of pressure: $\Delta V/\Delta P$. Resistance refers to the amount of force needed to move a gas or fluid through a single capillary tube. Poiseuille's law shows that viscosity, length of the tube, driving pressure, and radius of the tube contribute to the work required to move a liquid or gas through a tube. Of these conditions, the radius of the tube is the most important factor affecting flow. If the radius of the tube is decreased by half, the work required increases 16 times. It is for this reason that you should generally use the largest appropriate size tube possible when intubating. It is also the reason that in broncho-spasm, this decreased radius results in more resistance to flow of gas and that even small amounts of swelling in the pediatric airway can cause significant distress. The decreased flow of gas results in increased work of breathing and respiratory distress. Increased mucus production, basal membrane edema, and artificial airways all reduce airway diameter, increase resistance, and may result in increased work of breathing.

Pressure gradients allow for the bulk movement of gas in and out of the lung. During inspiration, contraction of the muscles of inspiration increases the size of the thorax. The increased size of the thorax results in an increase in the volume contained within the thorax. This increase in volume results in a decrease in the pressure within the thorax relative to the atmosphere, which causes air to move into the lungs. At the end of inspiration, the muscles relax, allowing the thorax to return to its original size. The volume decreases, the pressure increases, and exhalation occurs. Inspiration is an active process that requires work, whereas exhalation is usually a passive event. This concept is applicable during flight; because pressure is required to facilitate the passing of oxygen from blood to the cells, a decrease in pressure can lead to hypoxia.

The thoracic expansion that begins the inspiratory cycle is provided by muscle contraction. The

primary muscles used during inspiration are the diaphragm and external intercostal muscles. In times of increased stress, the accessory muscles such as the pectorals, sternocleidomastoid, and scalenes are utilized. Use of these accessory muscles increases oxygen use. Continued use of these muscles can quickly lead to muscle fatigue, hypercapnia/hypoxemia, and respiratory failure. Some muscles can augment exhalation in times of increased work of breathing, although it is mostly a passive process. When needed, the intercostal and abdominal muscles can assist exhalation **Figure 6-8**.

The stimulus and innervation of breathing is a complex process involving the pulmonary, cardiac, and neurologic systems. Normally, the drive to breathe comes from the need to eliminate CO_2. In the blood, CO_2 combines with water (H_2O) to form carbonic acid, which dissociates into H^+ and HCO_3^-. The end result is that as CO_2 increases, so does the level of H^+. Recall that pH is a function of H^+ concentration. As H^+ increases, pH decreases. This change is transmitted across the blood-brain barrier and stimulates the respiratory centers in the brain stem. As a back-up mechanism, chemoreceptors are located in the aortic arch and carotid arteries. These are sensitive to changes in oxygen levels. When the PaO_2 level drops below 60 mm Hg, these receptors reach maximal stimulation and provide the stimulus to increase breathing.

The respiratory centers are located in the brain stem, which comprises the medulla and pons **Figure 6-9**. In the medulla, the dorsal respiratory group regulates impulses to the diaphragm. The ventral respiratory group controls expiratory impulses, the upper airway muscles, and the intrinsic pattern of breathing. The pontine respiratory group and the pneumotaxic center fine-tune the respiratory pattern. The innervation of the diaphragm is from the phrenic nerve, which branches from the spinal cord at the level of C3 to C5. Spinal cord injury near this level dramatically affects the ability to breathe adequately.

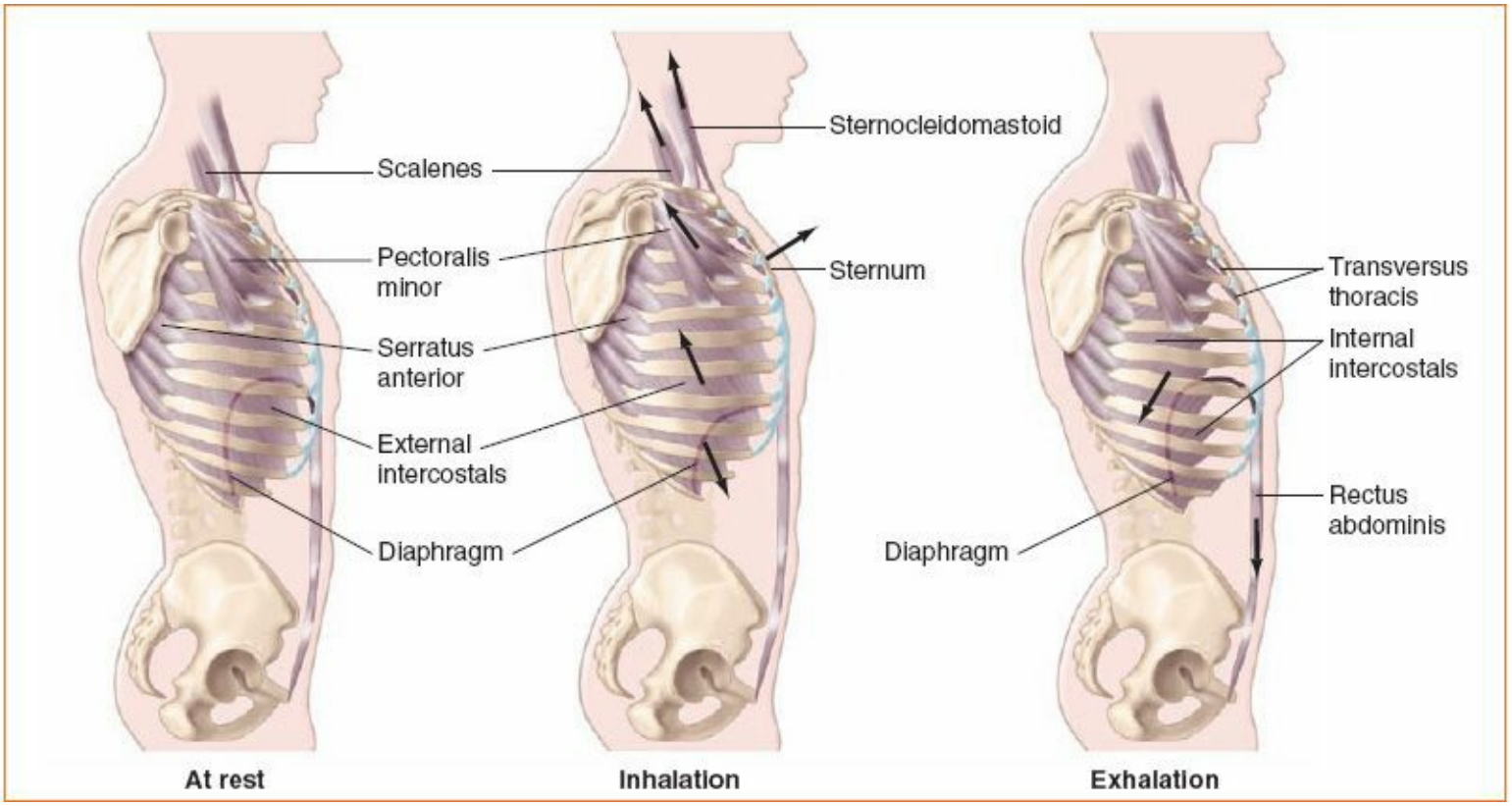


Figure 6-8 The mechanics of breathing.

The proper functioning of the respiratory system can be explained by the following five factors:

1. **Ventilation**: There must be an adequate bulk flow of gas in and out of the lung.

2. **Distribution**: The gas must be delivered into the areas of the lung that are able to engage in gas exchange. Changes in compliance or resistance will send the gas to areas of the lung with an unsuitable $\dot{V}/\dot{Q}$ ratio.
3. **Diffusion**: The AC membrane must be able to engage in gas exchange. Widening of the space (as in pulmonary edema) or disease (such as fibrosis) results in a decreased diffusion capacity.

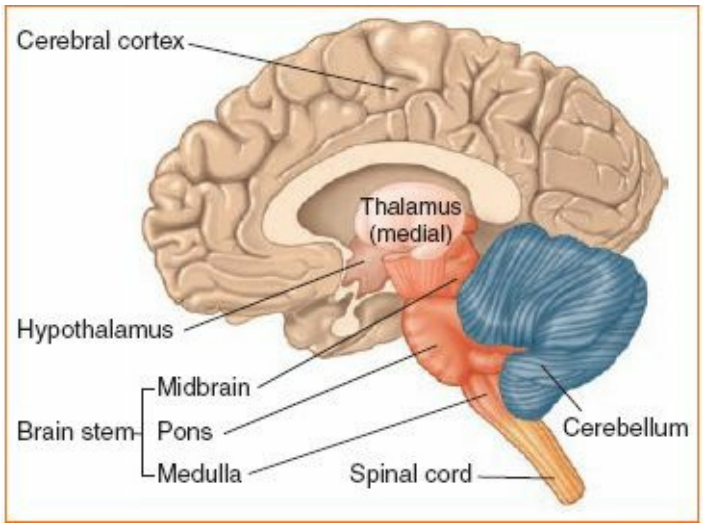


Figure 6-9 The pons and medulla are involved in neurocontrol of respiration.

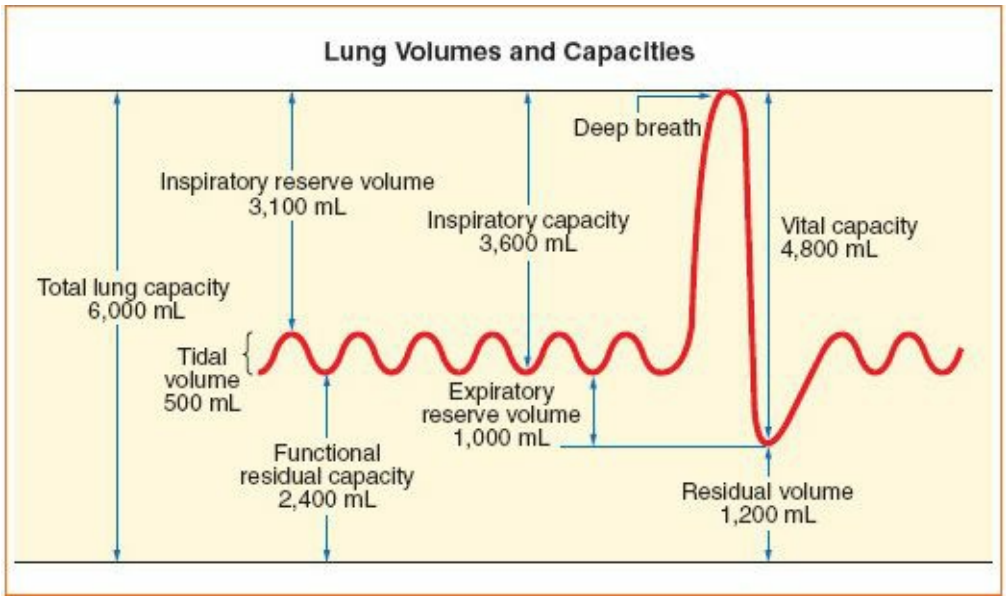


Figure 6-10 Respiratory cycle, capacities, and volume. The numbers are approximations.

4. **Perfusion**: As already discussed, blood flow through the pulmonary vasculature and contact with the alveolus is essential.
5. **Circulation**: The heart must be able to pump blood not only to the lung, but also distribute blood through the systemic circulation. There must also be adequate levels of hemoglobin for delivery of oxygen to the cells.

Respiratory Volumes and Capacities

The lungs of a healthy male adult can hold between 5 and 6 L of gas. This is called the **total lung capacity (TLC)**, which can be divided into different lung volumes and capacities. Several different lung

volumes make up total lung capacity **Figure 6-10**. A spirometer **Figure 6-11** measures respiratory capacities and the volumes of air in liters.

Tidal volume (V_T) is the amount of air inhaled and exhaled during each normal breath. **Minute volume** is the amount of air breathed in 1 minute and is calculated by multiplying the respiratory rate per minute by the average tidal volume. The adequacy and stability of minute ventilation maintain normal oxygen and CO_2 levels. Normal minute ventilation is 5 to 10 L/min. The amount of air that can be inhaled after a V_T is inhaled is the **inspiratory reserve volume (IRV)**, whereas the air that can be expelled from the lungs after a normal exhalation is the **expiratory reserve volume (ERV)**. The **inspiratory capacity (IC)** is the sum of IRV and V_T. **Vital capacity (V_C)** represents the total amount of air that can be exhaled following a maximal inspiration and is referred to as the pulmonary reserve. Normal V_C is 60 to 70 mL/kg of ideal body weight. V_C is dependent on gender, age, and height; decreases with age (approximately 20 mL/kg per year for those > 20 years); and increases with height. Women have a smaller V_C than men. A decrease in vital capacity to less than 10 to 15 mL/kg indicates poor pulmonary reserve and the inability to cough effectively. These persons almost always require some form of mechanical ventilation.

At the end of a maximal forced exhalation, a volume of air remains; this is referred to as the **residual volume (RV)**. RV added to ERV is the **functional residual capacity (FRC)**. The FRC allows for gas exchange between breaths. This is also where the application of **positive end-expiratory pressure (PEEP)** works. Applied PEEP increases the FRC and increases oxygenation. One other volume to be aware of is the **anatomic dead space (V_D)**. This space is found in the upper airway, the areas that do not participate in gas exchange. The volume in this space is normally approximately 2 mL/kg.

Figure 6-11 A spirometer.

Pathophysiology: Obstructive and Restrictive Disease States

Disease states can be categorized in several ways. Disease states resulting in difficulty with moving air out of the lungs can be classified as **obstructive diseases**, which include asthma, COPD, cystic fibrosis, and bronchioectasis, and involve an increase in airway resistance. Diseases that result in difficulty moving air in to the lungs are defined as **restrictive diseases**. These conditions result in the loss of chest or lung compliance, either individually or together. Restrictive diseases typically include occupational lung diseases (asbestosis, mesotheolioma), idiopathic pulmonary fibrosis, pneumonia, atelectasis, chest wall deformities and injuries, and all the neuromuscular diseases that affect breathing (for example, Guillian-Barré syndrome).

Diseases and temporary conditions can alter the $\dot{V}/\dot{Q}$ ratio. Ventilation in excess of blood flow

creates a dead space effect, and gas exchange is impaired (such as in tachypnea, pulmonary embolism). When blood flow is in excess of ventilation, gas exchange is also impaired, but this creates a **shunt** effect. A good example is pneumonia, in which a lobe of the lung is perfused but not ventilated because of infection in the air spaces. Be aware that there are also anatomic shunts that prevent blood from participating in gas exchange. Some anatomic shunts are congenital heart defects; others such as the bronchial and thebesian veins are normal and occur in the lung. The extent of shunt effect is indicated by the abbreviation Qt. The normal Qt range is 3% to 5%; a value greater than 20% indicates that the patient's condition is critical. Because oxygen is vital to the life and metabolism of every cell in the body, the evaluation of the presence of hypoxia and its treatment is crucial. It is important to understand these mechanisms as well as the causes of hypoxemia:

- Hypoxic hypoxia: defined as insufficient oxygen in the blood; tissues are therefore affected. Several causes exist, including hypovolemia, airway obstruction, decreased cardiac output, and coronary artery disease.
  - Low oxygen tension in the alveolus itself, and therefore not enough oxygen can diffuse across the alveolar-capillary membrane; can be caused by hypoventilation, high altitude (low barometric pressure), and suffocation.
  - Diffusion defects such as fibrosis or edema.
  - Shunting can be intrapulmonary, as caused by atelectasis, bronchospasm, and pneumonia, or can be extra-pulmonary, as with a congenital heart defect.
- Anemic hypoxia (also called hypemic hypoxia): caused by reduced or dysfunctional hemoglobin. Hypemic hypoxia interferes with the transportation phase of respiration, causing a reduction in oxygen-carrying capacity. Specific causes of hypemic hypoxia include anemias, hemorrhage, hemoglobin abnormalities, use of certain medications (sulfa drugs), and intake of chemicals. Carbon monoxide is present in exhaust fumes—from ambulances and both conventional and jet-engine aircraft. It is also present in cigarette smoke.
- Stagnant hypoxia: reduced cardiac output resulting in tissue hypoxia due to lack of circulation. Specific causes include heart failure, shock, continuous positive-pressure breathing, acceleration (*g* forces), and pulmonary embolism. A reduction in regional or local blood flow may be caused by extremes of environmental temperatures, postural changes, tourniquets, hyperventilation, embolism by clots or gas bubbles, and cerebral vascular accidents.
- Histotoxic hypoxia: occurs when cells are unable to use oxygen due to inactivation or destruction of key enzymes, such as in cyanide and strychnine poisoning as well as in later stages of carbon monoxide poisoning.

Determining the mechanisms can dramatically affect the course of treatment. Simply providing supplemental oxygen may not be sufficient in alleviating the hypoxia.

Patient Assessment

Chapter 5 covers the techniques of inspection, auscultation, and palpation. This section covers respiratory-specific considerations during the assessment process.

Breath Sound Assessment Techniques

After an adequate airway is in place and secured, assessment of breath sounds is vital. Breath sounds are created as air moves through the tracheobronchial tree and the alveoli, and can be auscultated. The size of the airway determines the type of sound that will be produced. (Recall that there are significant

differences between adult, child, and pediatric airways.) The trachea and bronchi have large diameters, so the sound produced is higher in pitch and is heard during inspiration and expiration. **Tracheal breath sounds**, also called bronchial breath sounds, are heard by placing the stethoscope diaphragm over the trachea or over the sternum. Assess breath sounds for duration, pitch, and intensity. **Vesicular breath sounds** are softer, muffled sounds and have often been described as wind blowing through the trees. The expiratory phase is barely audible. Breath sounds are heard over the majority of the chest, representing airflow in the alveoli. **Bronchovesicular sounds** are a combination of the two and are heard in places where airways and alveoli are found, the upper part of the sternum and between the scapulae. **Figure 6-12** describes the normal breath sounds. Locations for these sounds are shown in **Figure 6-13**. Bronchovesicular sounds should be assessed for duration, pitch, and intensity. Duration refers to the length of time for the inspiratory or expiratory phase of the breath. Normally, expiration is at least twice as long as inspiration. This relationship is expressed by the **I:E ratio**; a normal I:E ratio is 1:2. When a patient's airway is obstructed and the patient has difficulty getting air out, the expiratory component is prolonged and may be four to five times as long as inspiration; in this case the I:E ratio would be 1:5. In patients who are tachypneic, the expiratory cycle is short and approaches that of inspiration, and the I:E ratio may be 1:1. In mechanical ventilation, manipulation of the I:E ratio (expiration being longer than inspiration or inverse I:E) may be used to improve ventilation and hypoxemia in patients with severe lung injury.

Pitch is described as either higher or lower than normal, as in patients with stridor or wheezing. The intensity of sound depends on airflow rate, constancy of flow throughout inspiration, patient position, and the site selected for auscultation. Thickness of the chest wall may affect the intensity. Sounds that are less intense are said to be diminished. A common error in assessing the intensity of breath sounds occurs when auscultation is done over the patient's clothing. Sounds that might be classified as normal, but present in an unexpected area, can indicate an abnormal condition. For example, tracheal sounds in areas that should produce vesicular sounds may indicate consolidation or pneumonia.

The terminology regarding abnormal or **adventitious breath sounds** varies from text to text. The sounds are usually classified as continuous or discontinuous sounds. **Wheezes** and **rhonchi** are continuous sound as air flows through a constricted airway. Wheezes are usually high-pitched, whereas rhonchi are low-pitched, indicating that the airway is not as obstructed and is associated with mucus in the airways. **Crackles** (formerly known as *rales*) occur when airflow causes mucus or fluid in the airways to move. These tend to clear with coughing. Crackles may also be heard when collapsed airways or alveoli pop open. Crackles are classified as discontinuous sounds and may occur early or late in the inspiratory cycle. Early inspiratory crackles usually occur when larger, proximal bronchi open. These sounds are common in patients with COPD and tend not to clear with coughing. Late inspiratory crackles occur when peripheral alveoli and airways pop open and are more common in the dependent lung regions. These sounds are common in patients with reduced lung volumes. **Stridor** results from foreign body aspiration, infection, swelling, disease, or trauma within or immediately above the glottic opening. Stridor produces a loud, high-pitched sound ("seal bark"), particularly in pediatric patients.

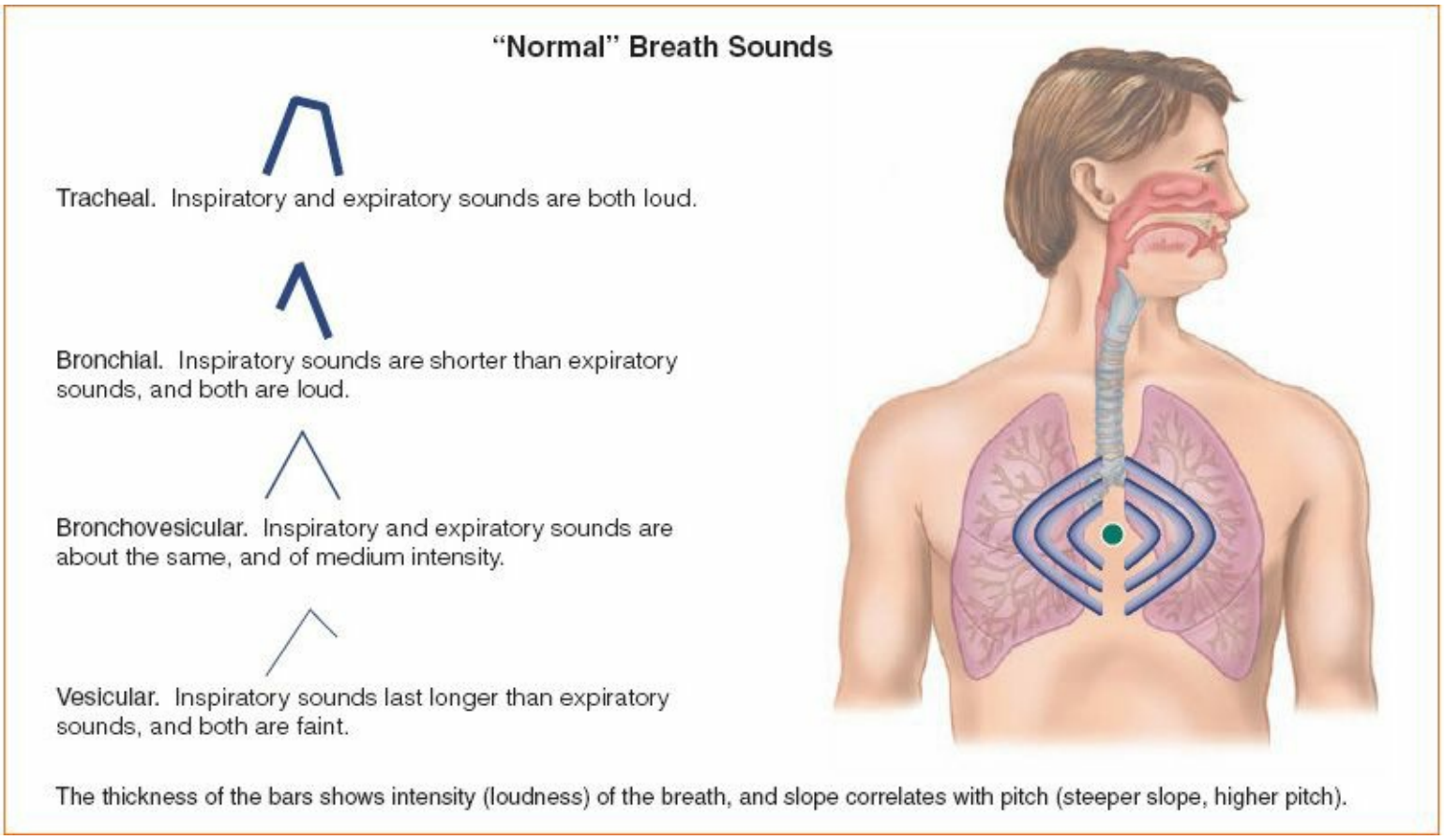


Figure 6-12 Normal breath sounds are heard over different parts of the chest. As you move away from the largest airways, breath sounds become softer. The character of inspiration vs exhalation also changes.

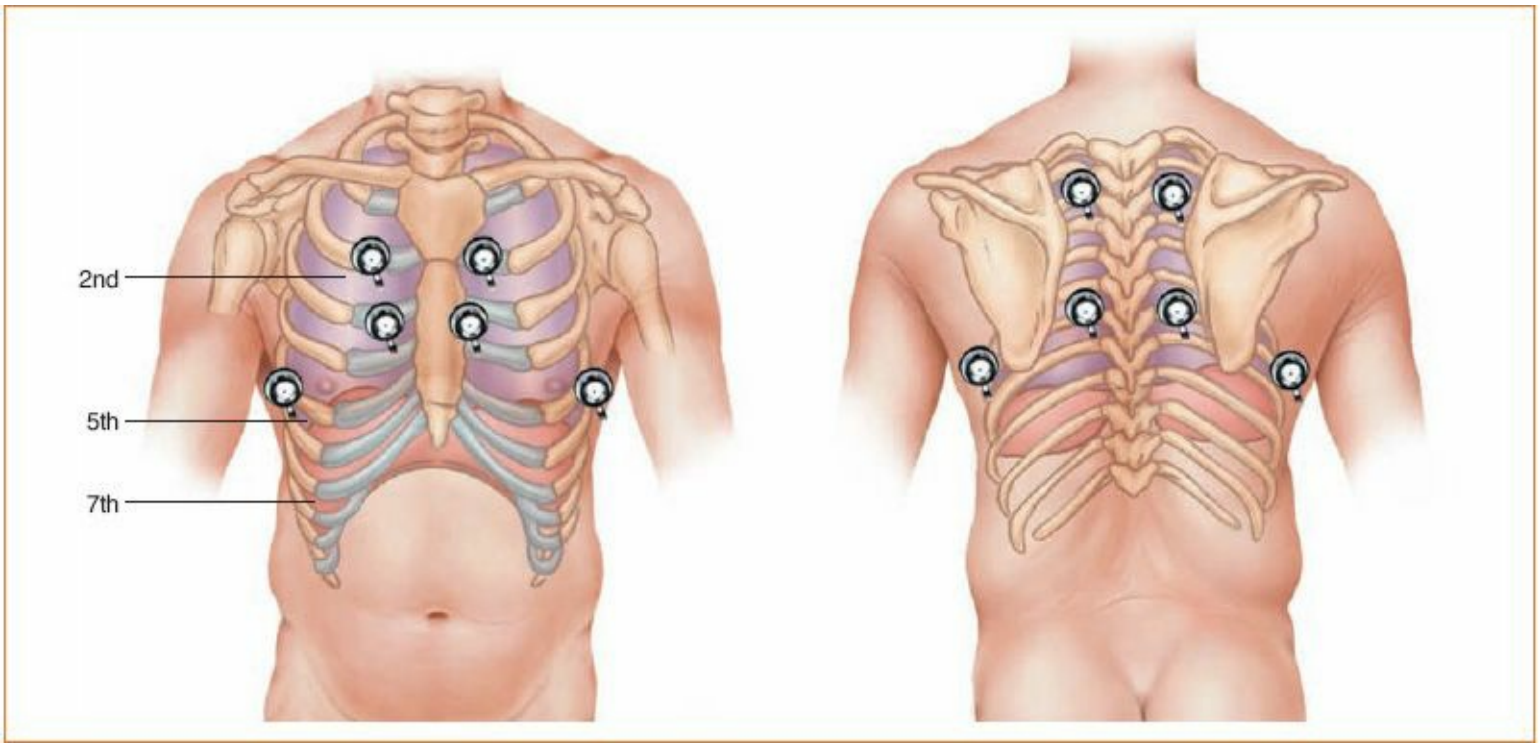


Figure 6-13 Common auscultation sites.

A **pleural friction rub** results from inflammation that causes the pleura to thicken. The pleural space can decrease as a result, allowing the surfaces of the pleura to rub together. This decrease often creates stabbing pain with breathing or any movement of the thorax.

Auscultation

Baseline breath sound assessment is discussed in Chapter 5. For the patient with an invasive airway device such as an endotracheal (ET) tube, Combitube, or laryngeal mask, listen over the epigastrium and sternal notch **Figure 6-14**, as well as the six recommended sites on the anterior chest wall. Assess this area for proper device placement and airflow. The baseline respiratory assessment establishes the standard by which subsequent assessments will be compared.

Breath sounds are best evaluated while the patient is taking slow, deep breaths through an open mouth and seated in a semi-Fowler's or high Fowler's position. Because of clinical circumstances, this often cannot be done. If the patient cannot be elevated, auscultate as many of the six recommended sites as possible. The diaphragm of the stethoscope should be placed firmly on bare skin. Remember, to avoid misinterpretation of lung sounds, auscultation should not be performed through clothing. Be aware of your patient's comfort while assessing breath sounds. Rushing the patient through the process may cause hyperventilation or exacerbate existing dyspnea. Document the sites chosen for auscultation in your patient care report and the status (patency, appearance, integrity) of any artificial airway. In addition to periodic monitoring of breath sounds during transport (which may be difficult to hear), evaluate breath sounds at the following times: immediately upon arrival at the patient's bedside, after any gross patient movement, and immediately before transferring the patient's care to other providers.

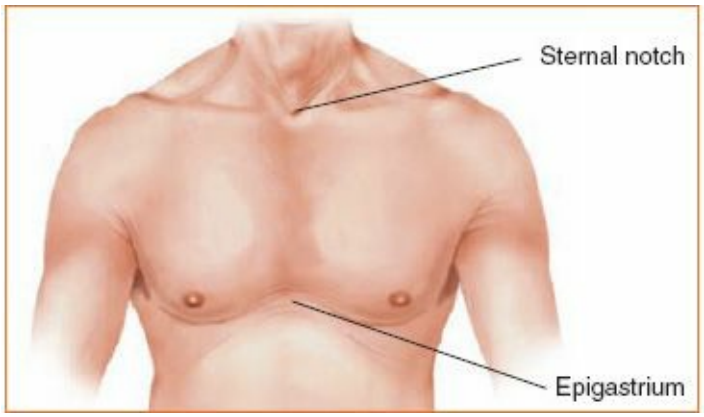


Figure 6-14 In patients with an invasive airway, listen for breath sounds over the epigastrium and sternal notch.

Normal and Abnormal Respiratory Patterns

When you are assessing respiratory patterns, consider the following three characteristics: rate, depth, and rhythm.

Rate

Eupnea is normal breathing at a rate of approximately 12 to 20 breaths/min in the adult patient. Pediatric norms range from 30 to 50 breaths/min in the newborn to 12 to 20 breaths/min in the adolescent. A number of factors can cause **tachypnea**, an abnormally fast respiratory rate—fever, pneumonia, metabolic acidosis, hypoxemia, some poisonings and drugs (aspirin overdose, stimulant use), and lesions of the respiratory center in the brain. Stress, anxiety, and pain can also result in tachypnea. Although tachypnea is a compensatory mechanism, it also has negative effects. Because rapid respiratory rates usually result in shallower breaths, the amount of **alveolar ventilation (V_A)** (the volume of air that comes into contact with the alveolar capillary membrane surfaces and participates in the exchange of gases between the lung and the blood) decreases in the tachypneic patient. More muscle work is used to generate "wasted" ventilation. This increased "work" also results in increased oxygen consumption.

Numerous factors can also lead to **bradypnea**, a slower than normal respiratory rate: narcotic or

sedative drugs, alcohol, metabolic disorders, respiratory system decompensation or fatigue (particularly in pediatric patients), and traumatic and nontraumatic central nervous system (CNS) lesions. Mild bradypnea is normal in certain stages of sleep. **Apnea**, the absence of respiration, may be episodic or periodic. Periods of apnea longer than 15 seconds require immediate intervention. The patient may need ventilatory support or other resuscitative efforts.

Depth

In addition to respiratory rate, always assess the depth of respiration. Assessment of depth can be achieved by direct observation or by palpation. **Hyperpnea** describes a deeper than normal breath, whereas **hypopnea** refers to a shallow breath. Hyperpnea can lead to low levels of CO_2 (thus increasing pH, resulting in respiratory alkalosis), whereas hypopnea can result in increased CO_2 levels (thus decreasing pH, resulting in respiratory acidosis) and decreased oxygen values.

Pattern

Several abnormal respiratory patterns present with characteristic alterations of the respiratory rate, depth, or regularity **Figure 6-15**.

Cheyne-Stokes respiration features a cyclic pattern of increased respiratory rate and depth with periods of apnea. Following the apnea, the patient begins breathing with slow, shallow breaths that increase in rate and depth until apnea returns. The condition can be caused by increased intracranial pressure, renal failure, meningitis, drug overdose, or hypoxia secondary to congestive heart failure. Otherwise, healthy individuals may breathe in a Cheyne-Stokes pattern following exposure to altitude changes or with hyperventilation syndrome. Acidosis, particularly when caused by CO_2 levels, can also trigger Cheyne-Stokes respirations.

Cluster breathing is another abnormal respiratory pattern, in which a cluster of irregular respirations that vary in depth are followed by a period of apnea at irregular intervals.

Biot's (ataxic) breathing is similar to Cheyne-Stokes respirations, but the pattern is irregular. While Cheyne-Stokes has repeating patterns of gradual increases concluding with apnea, three patterns occur in Biot's breathing: (1) slow and deep; (2) rapid and shallow; and (3) apnea without any predictability of which pattern will follow. Causes of Biot's breathing include meningitis, increased intracranial pressure, and central nervous system dysfunction. The condition often indicates lesions higher in the respiratory center in the brain stem than those that produce Cheyne-Stokes respirations.

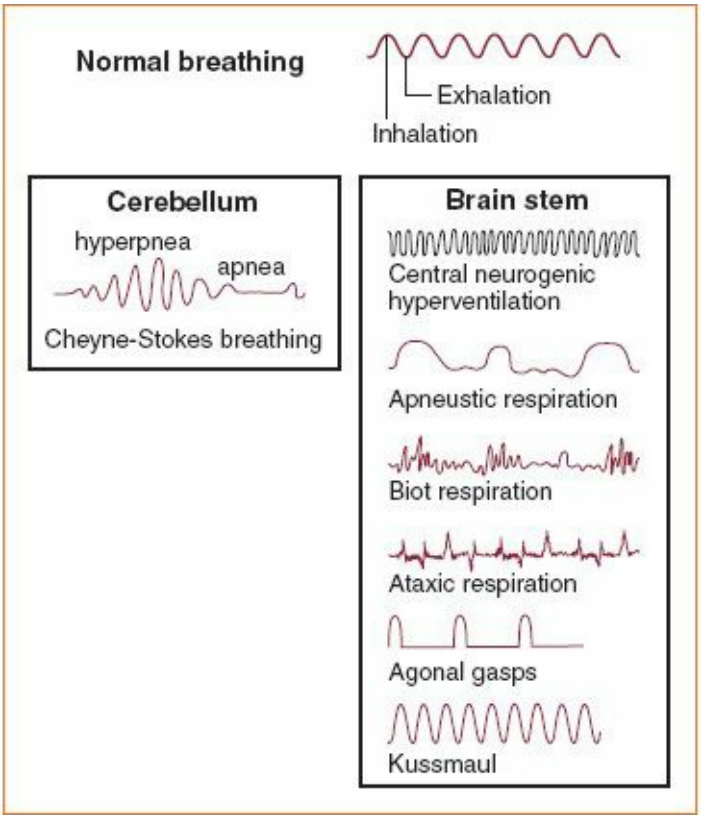


Figure 6-15 Abnormal breathing patterns.

Kussmaul's respiration is fast and deep without periods of apnea. The rate and depth are greater than the normal rate expected for the patient's age group. Breathing is labored, with periods of deep breaths punctuated by sighs. Kussmaul's pattern may indicate metabolic acidosis, including diabetic ketoacidosis, or renal failure. It generally appears in conditions that cause severe acidemia.

Apneustic breathing indicates lesions in the respiratory center of the brain stem. The patient exhibits a prolonged, gasping inspiration followed by an extremely short, ineffective expiration at a rate between 1 and 2 breaths/min. Over a period of several minutes of ineffective exhalation, the patient's lungs may hyperinflate. This pattern results in severe hypoxemia and, if uncorrected, rapid death.

Central neurogenic hyperventilation is a pattern of deep, rapid respirations at rates of 40 to 60 breaths/min. A midbrain lesion or dysfunction is the cause. Patients exhibiting central neurogenic hyperventilation are generally comatose with Glasgow Coma Scale scores of less than 8. These patients require intubation and ventilatory assistance in order to stabilize arterial blood gas parameters. Hyperventilation syndrome presents with increases in rate and depth of respiration. Hyperventilation can result from exertion, fear, anxiety, fever, hepatic coma, acid-base imbalance, or midbrain lesions. A respiratory rate between 20 and 30 breaths/min in the adult is classified as moderate hyperventilation. A prolonged respiratory rate greater than 30 breaths/min is severe, and ventilatory support is often necessary to prevent respiratory muscle fatigue. Use caution when assessing the underlying cause of hyperventilation before initiating any corrective treatment. Never withhold oxygen from a patient showing signs or symptoms of hypoxia.

A state of hyperventilation may develop in a patient who is intubated or being ventilated. Closely monitor vital signs, pulse oximetry, capnometry readings, end-tidal CO_2 ($ETCO_2$) levels, mental status, and skin color in any patient receiving ventilatory support.

Agonal respirations, also abnormal, are a pattern of deep, slow, shallow, irregular, "all-or-none" breaths or occasional gasping breaths, accompanied by a slow respiratory rate.

Another abnormal respiratory pattern is seen in patients with obstructive diseases such as COPD and

asthma. The pathology of these diseases leads to airway collapse and hyperinflation of the lungs. These individuals have difficulty exhaling completely, which leads to air trapping in the lungs and shallow compensatory respirations.

In order to assist with exhalation, these individuals exhale for longer periods of time and with the lips pursed. Pursed lip breathing applies a back pressure to the airways to try to prevent airway collapse. This pursed lip breathing is an attempt to maintain PEEP (the amount of pressure above ambient pressure present in the airway at the end of the respiratory cycle). The I:E ratio in patients with obstructive disease is usually abnormal.

For patients who are on ventilatory support, the ventilator can provide supplemental PEEP and **continuous positive airway pressure (CPAP)**. CPAP is similar to PEEP in that they both can maintain pressure in the airway and alveoli at the end of the exhalation phase.

The appropriate level of intervention in these breathing abnormalities is directly related to the work of breathing and the ability of the patient to maintain that work. Remember that all muscle activity uses oxygen to metabolize glucose and manufacture adenosine triphosphate. CO_2 is produced as a waste product. This increases the need for further ventilation that in turn may exacerbate muscle fatigue. Remember also that the diaphragm is the most efficient of the respiratory muscles. The accessory muscles, the scalene, sternocleidomastoid, and external intercostals, use more oxygen. The ability of the patient to maintain this increased workload depends on age, physical conditioning, degree of cardiopulmonary reserve, and other combined medical conditions. Failure to adequately manage the work of breathing can lead to cardiopulmonary arrest. Do not confuse the work of breathing with shortness of breath. Shortness of breath is a subjective complaint, whereas assessment of the work of breathing is a clinical evaluation.

Palpation

Chapter 5 reviews palpation—placement of the hands directly on the chest wall to evaluate the status of the lungs, skin, and subcutaneous tissues and chest expansion. A "snap, crackle, pop" sensation indicates the presence of subcutaneous emphysema, in which air is escaping from the pleural space and dissecting through the subcutaneous tissues. Although subcutaneous emphysema is benign, the underlying cause may be life threatening. If subcutaneous emphysema is detected, it is important to inquire about recent trauma or invasive procedures (ie, intubation, central line placement) and whether these are new findings.

When a person is speaking, the vibrations of the vocal cords are transmitted to the chest wall. This is called vocal fremitus. Vocal fremitus is assessed when you are performing tactile fremitus, which is done by placing the hands bilaterally on the upper thorax and asking the patient to say "99." This procedure is repeated in a methodical way down the chest. An increase in the fremitus, or vibration, indicates that the underlying lung tissue is more solid (consolidated) and contains less air than normal, which may indicate pneumonia or atelectasis. In addition, if secretions are present in the airways, the vibrations will increase. This finding may indicate that the patient needs to cough or be suctioned. The procedures of vocal fremitus and tactile fremitus are useful in patient assessment, but are usually not practical in the transport setting.

Assessment in Preparation for Transport

Prior to transport, review the patient care report, noting any medications, disease processes, or trauma that might compromise ventilation. Review the patient care record noting lab studies, and review the patient's vital signs. Take special note of when the last arterial blood gas (ABG) measurement was performed and whether the data are valid. If an artificial airway is in place, note the size and type, and confirm proper placement before transport. If the patient requires mechanical ventilation, note the ventilator settings and patient response. If there is any doubt about the placement of tracheal tubes,

nasogastric (NG) tubes, or central lines, communicate your concerns to hospital personnel. If the transport is to be by air, remember that altitude-induced pressure changes can occur during travel by helicopter or fixed-wing aircraft. Boyle's law (at a fixed temperature, the volume of a gas varies inversely with the pressure surrounding it) may cause air or gases within the body to expand as altitude increases (and barometric pressure decreases). These pressure changes are particularly important for air within the pleural space (pneumothorax). Equipment with air-filled spaces, such as the balloon of an ET tube, may need to be replaced with fluid to prevent problems with expansion during ascent during transport. During these transports, monitor the patient closely for evidence of dyspnea or the development of an air embolus.

When assessing the respiratory system, begin with the basics.

1. Assess the ABCs.
2. Determine the stability of the airway.
3. Observe the rate, rhythm, depth, and character of respiration.
4. Inspect the chest looking for symmetric rise and fall with each respiratory cycle. Note any accessory muscle use. Note the presence of any central lines or Hickman catheters, nitroglycerin or other medication patches, or in-dwelling devices such as pacemakers, implanted defibrillators, chest tubes, or medication delivery devices.
5. Note any wounds, abrasion, or bruising. Note the presence and location of bony crepitus or subcutaneous emphysema.
6. Examine any dressings on the chest (wounds, central venous access, chest tubes) and determine if they are dry and intact.
7. Observe any drainage being collected and note its color and consistency.
8. Auscultate the chest noting the presence or absence, quality, and type of breath sounds heard. Compare breath sounds from one side of the chest to the other.
9. If the patient requires oxygen, assess that the oxygen delivery device and the delivered **fraction of inspired oxygen (FIO_2)** meets the patient's needs and adequately maintains the desired oxygen saturation as measured by pulse oximetry (**SpO_2**).

Because cardiac and respiratory functions are intimately linked, assess the patient's circulation to obtain a complete clinical picture. Check the patient's blood pressure, pulse rate, capillary refill time, skin color, and temperature. Mental status is also an important consideration, because hypoxia directly affects mental status and level of consciousness.

ABG Monitoring

The gold standard for assessing the functioning of the respiratory system is measuring the ABG level. In this procedure, blood is obtained from a superficial artery, such as a radial or brachial artery, with a heparinized syringe. The blood is then analyzed for pH, $PaCO_2$, PaO_2, HCO_3^- (concentration of bicarbonate ion), base excess (BE, indicating whether the patient is acidotic or alkalotic), and SpO_2. Normal blood gas values are summarized in **Table 6-1**. The technique for obtaining an ABG value is covered in Chapter 8.

The values for pH, FIO_2, and HCO_3^- are used to evaluate the acid/base status of the patient. The $PaCO_2$ is an indicator of the effectiveness of ventilation. The values for PaO_2 and SpO_2 are indicators of oxygenation. A more complete discussion of blood gas interpretation and abnormalities is in Chapter 8. In

order to maintain normal blood gas values, a relationship between alveolar ventilation and perfusion of the alveolar capillaries must be maintained. This is expressed as the $\dot{V}/\dot{Q}$ ratio. Recall that $\dot{V}/\dot{Q}$ imbalances are the most common cause of hypoxemia.

Although advanced technology has made portable blood gas analysis possible, most CCTPs will rely on noninvasive methods of assessing ventilation and oxygenation. Pulse oximetry provides information about arterial oxygen saturation and the pulse **Figure 6-16**. All pulse oximetry models work on a similar principle. The machine's diode directs two wavelengths of light, red and infrared, through body tissues toward a photoreceptor. Bound hemoglobin alters light absorption; the degree of change in light transmission indicates the level of arterial oxygen saturation. Most detectors also supply the monitor with a pulsatile waveform that shows beat-to-beat changes in pulse amplitude and regularity. The finger, pinnae of the ear, toe, and sometimes the bridge of the nose are used as monitoring sites.

The significance of this reading is based on the relationship between oxygen and hemoglobin. A number of factors can alter this relationship. Abnormal hemoglobin levels, such as those seen in conditions that produce carboxyhemoglobin, methemoglobin, and sulfhemoglobin, significantly reduce the reliability of pulse oximetry. Conventional pulse oximetry is unable to differentiate between normal oxyhemoglobin and the dyshemoglobinemias. Because the dysfunctional hemoglobin is still bound, the pulse oximetry reading will be normal. Newer, pulse carbon monoxide-oximetry uses additional wavelengths of both infrared and visible spectrum light to measure and differentiate oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, methemoglobin, and hemoglobin itself. A pulse carbon monoxide-oximeter can be useful in situations where dyshemoglobins might be present. Decreased tissue perfusion (hypotension, cardiogenic shock) to the periphery may result in impaired light absorption and inaccurate pulse oximetry readings. Hypothermia may also alter pulse oximetry readings because of peripheral vasoconstriction. Peripheral vascular disease (PVD) may also alter blood flow to the periphery and pulse oximetry readings. Normal pulse oximetry readings should be 95% to 100% but can vary with underlying diseases such as COPD.

Capnography (graphic representation of exhaled CO_2) and **capnometry** (numeric value) are both important adjuncts to determining airway patency and appropriateness of ventilation **Figure 6-17**. These devices monitor the exhaled CO_2 concentration by analyzing air samples obtained directly from the airway (endotracheal or tracheostomy), through nasal prongs in the spontaneously breathing patient with a natural airway, or by insertion of a sampling port into the ventilator circuit. Because CO_2 readily diffuses across the alveolar capillary membrane and quickly equilibrates in the alveolar gases, exhaled gases, particularly those present near the end of exhalation, closely approximate arterial CO_2 levels (35 to 45 mm Hg).

TABLE 6-1 Normal ABG Values

Parameter	Normal Value	Range
pH	7.40	7.35–7.45
$PaCO_2$	40 mm Hg	35–45 mm Hg
PaO_2	100 mm Hg	80–100 mm Hg
HCO_3^-	24 mEq/L	22–26 mEq/L
Base excess	± 2 mEq/L	–2–3 mEq/L
SpO_2	97%	> 95%

Abbreviations: $PaCO_2$, partial pressure of carbon dioxide (arterial); PaO_2, partial pressure of oxygen (arterial); Hco_3–, concentration of bicarbonate ion; SpO_2, oxygen saturation as measured by pulse oximetry.

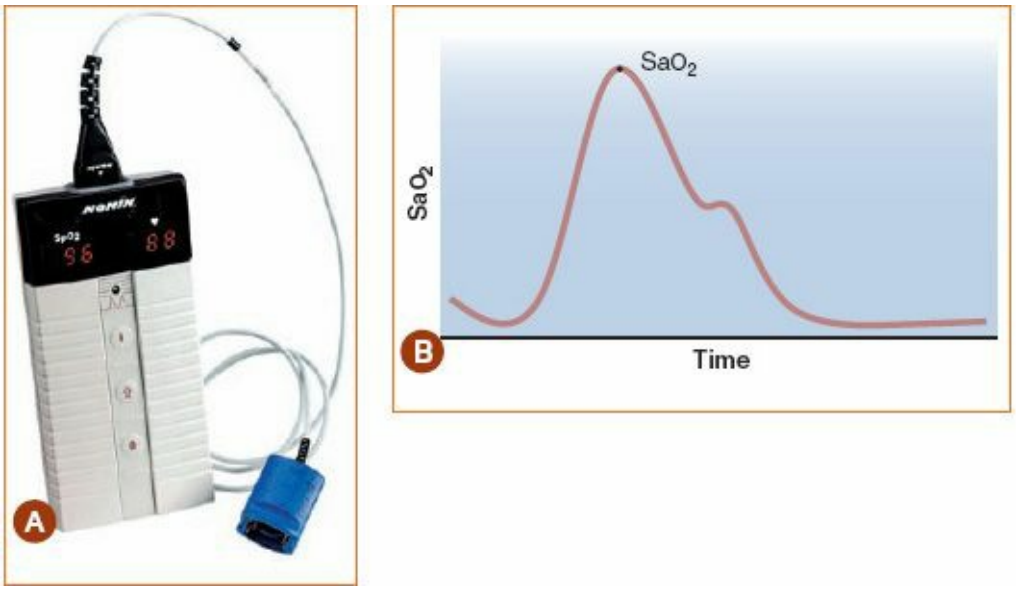


Figure 6-16 **A.** A pulse oximeter. **B.** The characteristic shape of the waveform when the pulse oximeter is properly sensing. Sao_2 indicates percentage of available hemoglobin that is saturated with oxygen.

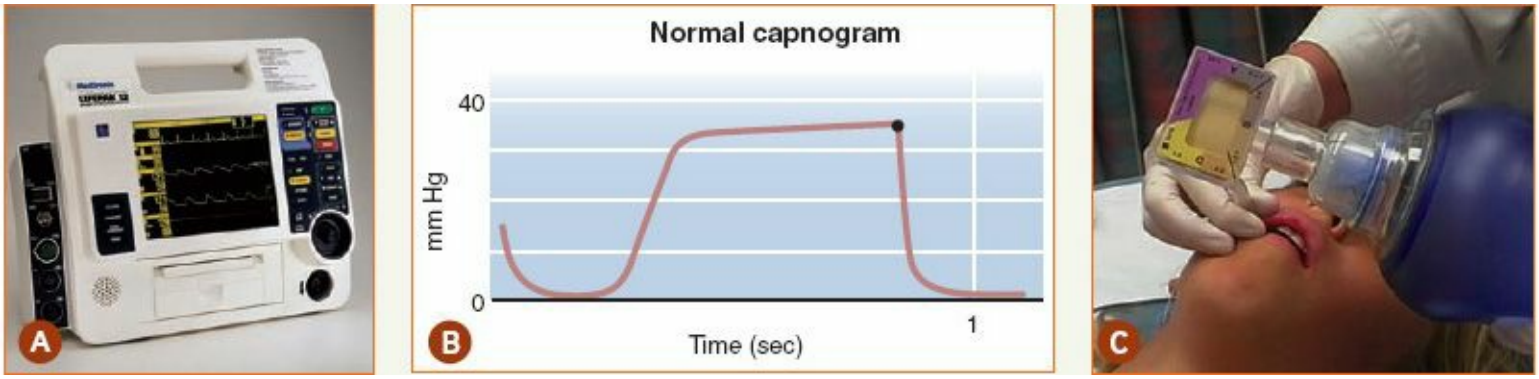


Figure 6-17 Carbon dioxide monitoring equipment. **A.** Capnometer. **B.** A normal capnogram waveform. **C.** An end-tidal carbon dioxide detector.

Capnography and capnometry provide data on the patency of the airway and the adequacy of ventilation. Use of $ETCO_2$ monitoring is limited in the face of cardiac arrest. In a patient who had just arrested, $ETCO_2$ may be detected despite a lack of perfusion. Patients in prolonged cardiac arrest will have no $ETCO_2$, and the return of measurable $ETCO_2$ is an indicator of effective CPR and the possible return of spontaneous circulation.

Controversies

Research suggesting that patients are ventilated too fast when in cardiac arrest has resulted in a push for compression-only CPR. Studies have shown that with patients in cardiac arrest, chest compressions

without ventilations have the same effect as CPR with ventilations. Providers may choose between standard CPR and compression-only CPR. Neither method has been proven as superior to the other; currently, data are inadequate.

Many prehospital systems are now suggesting a ventilation rate of 6 to 8 breaths/min during cardiac arrest and are using metronomic devices to time ventilation rates.

Respiratory and Ventilation Abnormalities

Respiratory insufficiency is the inability of the respiratory system to keep up with the metabolic demands of the body. Careful respiratory assessment helps the CCTP to determine whether the insufficiency is from ventilation or oxygenation. Treatment depends on the cause of the insufficiency. For example, thoracic, head, or spinal cord injury, central nervous system depression, drugs, and conditions in which fatigue challenges the patient's ability to breathe normally may cause **respiratory depression**. Symptoms of respiratory depression consist of a low respiratory rate (<12 breaths/min in adults) for a prolonged period of time or hypoventilation. In this case, increasing ventilation will resolve the insufficiency. Oxygen therapy is indicated in patients who cannot maintain normal oxygen saturation.

Respiratory failure occurs when the respiratory system fails to meet the body's metabolic needs. Respiratory failure can present with decreasing respiratory effort and depth. The patient may be anxious, confused, or obtunded. If not reversed, respiratory failure leads to respiratory or cardiopulmonary arrest. There are two basic types of respiratory failure: oxygenation failure and ventilatory failure. Tachypnea is the hallmark of oxygenation failure. The ability to sustain a high respiratory rate is related to the amount of accessory muscle use that the patient can endure before muscle fatigue occurs. The astute CCTP must intervene before the patient suffers an arrest. Ventilatory failure develops with increased arterial tension of CO_2.

Basic Airway Management

Although intubation is considered the gold standard for airway management, basic airway skills are the starting point in the initial patient assessment and treatment and what we fall back on in times of difficulty. These basic skills may be as simple as positioning the nontrauma victim in the recovery position or using the head tilt–chin lift or jaw-thrust maneuver to maintain airway patency. Other basic skills may use other adjuncts such as the oropharyngeal airway (OPA) or nasopharyngeal airway (NPA).

Positioning

Patients are usually able to assume a position that makes breathing easiest. This position is usually sitting upright in the "sniffing" position. Under most circumstances, you should allow the patient to do this or be prepared to assist the patient if he or she is immobile **Figure 6-18**. Patients with spinal precautions or those with a diminished level of consciousness are unable to assume this position or adequately protect their airway. As the level of consciousness decreases, control of the muscles located within the oropharynx is lost, causing the tongue to contact the posterior pharyngeal wall or soft palate, which results in airway obstruction. Muscular control of the epiglottis may also be lost, causing partial or total airway obstruction.

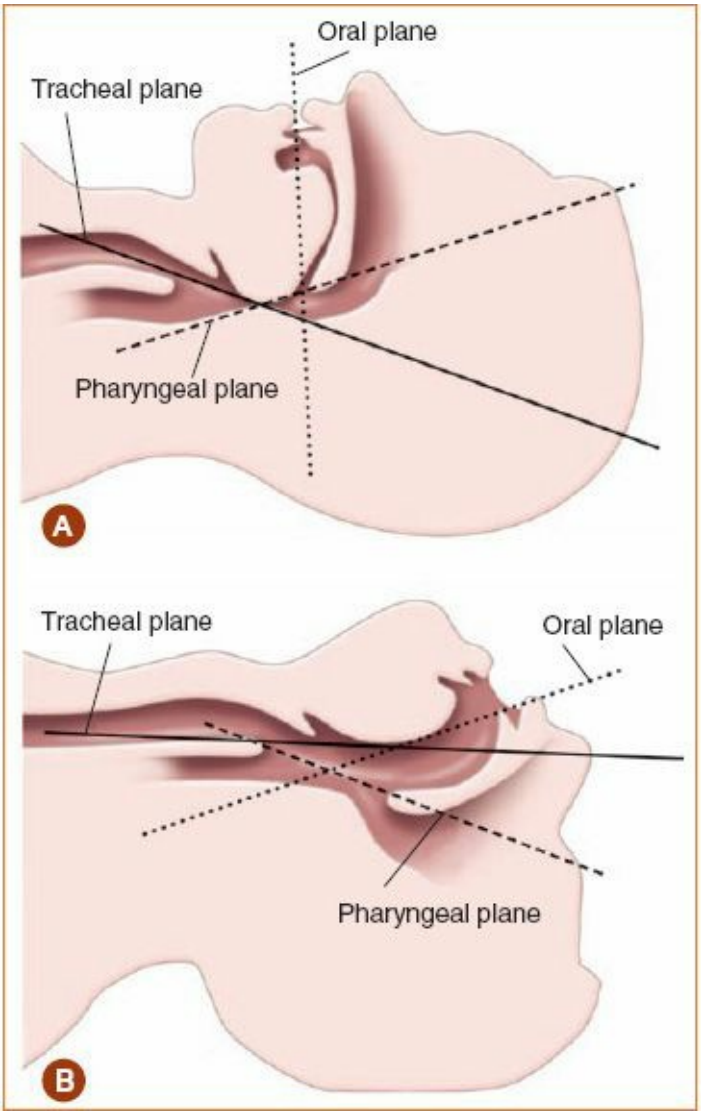


Figure 6-18 Sniffing position. **A.** The three axes of the airway (oral, pharyngeal, and tracheal) in the neutral position. **B.** The three axes in the sniffing position.

As a patient's level of consciousness diminishes, the ability to control secretions also becomes diminished. This diminished control may result in significant aspiration, especially when patients are found unconscious and supine. Patients are also resuscitated in the supine position. Placing unconscious patients (nontrauma) who are breathing in the recovery position helps to minimize airway obstruction and aspiration.

In patients undergoing resuscitation that requires supine positioning, manual airway maneuvers may be used to open the airway:

- Head tilt–chin lift: This is the most commonly known and used manual method of opening the airway of the unconscious, nontrauma victim **Figure 6-19**.
- Tongue-jaw lift: This lift is most commonly used to open and assess a patient's airway for foreign body obstruction. The patient cannot be ventilated using this maneuver **Figure 6-20**.

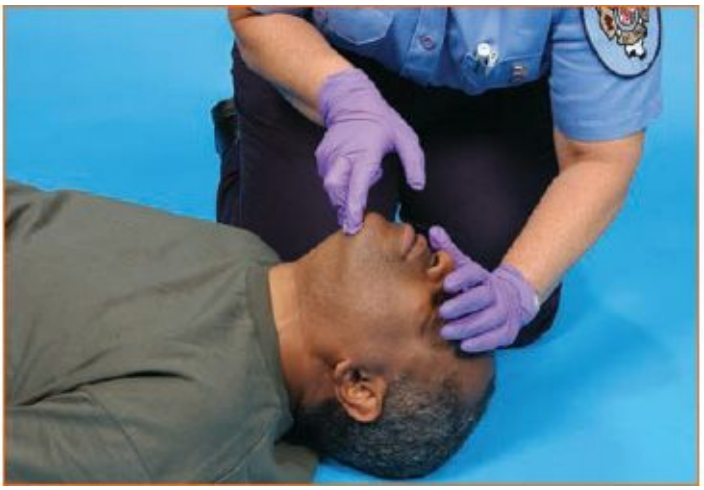


Figure 6-19 Head tilt-chin lift.

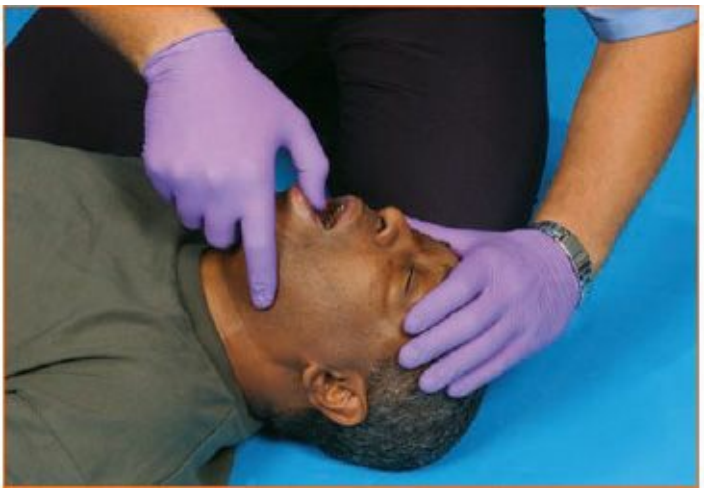


Figure 6-20 Tongue-jaw lift.

Figure 6-21 Jaw thrust maneuver.

- Jaw thrust: This thrust is frequently used to provide a patent airway and cervical spine protection in the unconscious trauma patient. In the nontrauma patient, this thrust may be combined with a slight head tilt to provide better airway opening **Figure 6-21**.

Airway Adjuncts

Both the OPA and the NPA are used along with manual airway maneuvers to provide a patent airway. In

the unconscious patient, neither can be used independently, as both adjuncts require maintaining manual airway maneuvers.

The OPA is a rigid device made of plastic or similar material that is placed in an unconscious patient's mouth **Figure 6-22**. The patient's gag reflex must not be present or the airway must be removed immediately. The size of the airway can be appropriately determined by measuring from the central incisors to the angle of the jaw. An airway that is too small or too large will not provide a patent airway and may worsen an already compromised airway. Placement of the device does not replace the need for manual methods but supplements them.

The NPA is a flexible rubber or silicone device that is placed in the nares and extends to the oropharynx. It is better tolerated than the OPA in semi-conscious patients. Its length can be appropriately sized from the tip of the nose to the tragus of the ear. Its diameter should be the largest possible, but not large enough to cause blanching of the naris. Placement should not be forced as the mucosa of the nose lacerates easily and may bleed significantly. Head trauma with evidence of basilar skull fracture or facial fracture is a contraindication for insertion. As with the OPA, the NPA does not take the place of manual methods but supplements them **Figure 6-23**.

Suction

One of the first rules in airway management is that your patient has a full stomach and is waiting to vomit on you. The ability of the prehospital provider to clear debris (vomit, blood) from a patient's airway, while a basic skill, may be lifesaving. It can restore airway patency and minimize the potential for aspiration. In the context of upper airway suctioning, a large-bore suction apparatus, of which there are various types, both fixed and portable, can be used. These suctioning devices use rigidtype suction catheters (Yankauer, tonsil tip) to remove debris. In the patient with large volumes of debris or vomit, turning the patient to his or her side while using a large-bore suction device works best **Figure 6-24**.

Flexible or soft-type suction catheters are used for suctioning the nasopharynx, the oropharynx, and the lower airways in patients with an artificial airway in place. These suction catheters are not designed to remove large volumes or large particulate matter and come in a variety of sizes referred to as French. Remember that suctioning of the nasopharynx, the oropharynx, and the lower airway may cause hypoxemia and hemodynamic instability. Patients should be preoxygenated, and suction attempts should be limited to 10 seconds or less while withdrawing the catheter. Patients should be oxygenated and ventilated if necessary between suction attempts.

In the critical care transport setting, you will use suctioning while an ET tube is in place. Indications for suctioning in this scenario include:

- Dyspnea
- Obstruction
- Excessive secretions

Complications are as follows:

- Hypoxemia
- Cardiac arrhythmias
- Mechanical trauma
- Infection
- Increased intracranial pressure
- Inability to remove material due to a mucous plug or dried crusting

Skill Drill 6-1 shows the steps for suctioning a patient with an ET tube in place, which are also described here:

1. Choose an appropriate suction adjunct: Yankauer for larger objects, French ("whistle tip") catheter for fluid or ET obstructions.
2. Check, prepare, and assemble your equipment **Step 1**.
3. Maintain universal precautions.
4. Use a sterile technique.
5. Lubricate the suction catheter **Step 2**.

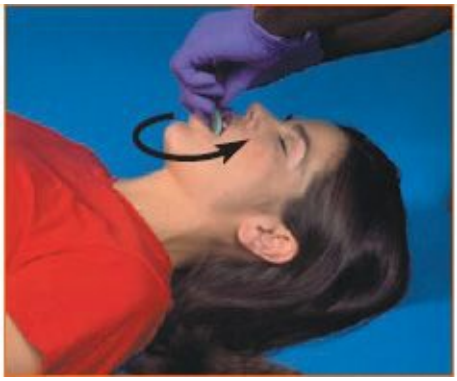


Figure 6-22 Use of an oropharyngeal airway.

Figure 6-23 Use of a nasopharyngeal airway.

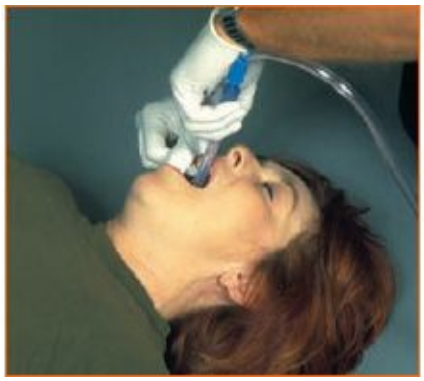


Figure 6-24 Suctioning.

6. Preoxygenate the patient for 1 to 2 minutes with 100% oxygen **Step 3**.
7. Measure the suction catheter tip from the corner of the mouth to the angle of the jaw or insert through

ET tube until patient coughs **Step 4**.

8. Suction for no longer than 5 to 10 seconds as you remove the suction catheter with a twisting motion **Step 5**.
9. Continuously observe patient response to suctioning (vital signs).
10. Rinse the catheter with sterile water.
11. Assess the airway, ET, and lungs for effectiveness. Reattach the bag-mask device and resume ventilation and oxygenation **Step 6**.

Oxygen Administration

Oxygen is the most commonly administered drug in the hospital and prehospital environments. Oxygen administration has few side effects and potentially significant benefit when administered appropriately. As a general rule, all ALS patients should receive some form of supplemental oxygen based on provider assessment, regardless of documented Spo_2. Supplemental oxygen should not be withheld from a patient based upon a normal Spo_2 reading. Administering supplemental oxygen to a patient with a normal Spo_2 will do no harm and will potentially improve regional or global tissue hypoxia. Remember, it is possible for a patient to have an Spo_2 of greater than 95% and still be hypoxic at the cellular level.

Low-flow oxygen devices such as a nasal cannula can safely and comfortably deliver an FIO_2 up to approximately 40% **Table 6-2**. At flows greater than 5 L/min, a nasal cannula-type device can become ineffective and uncomfortable for the patient. Patients in significant respiratory distress or with significant hemodynamic instability require higher flow oxygen, usually through a nonrebreathing mask, which can deliver an FIO_2 closer to 1.0% (100%). Remember, with a critical patient, too much oxygen is better than too little.

The appropriate oxygen delivery device should be chosen based on the physical assessment of the patient. Patients in significant respiratory distress require high-flow oxygen via a nonrebreathing mask or bag-mask type device. Patients with agonal respirations or those who are apneic require bag-mask ventilations with supplemental high-flow oxygen. The device chosen must fit the patient's overall assessment.

Skill Drill 6-1

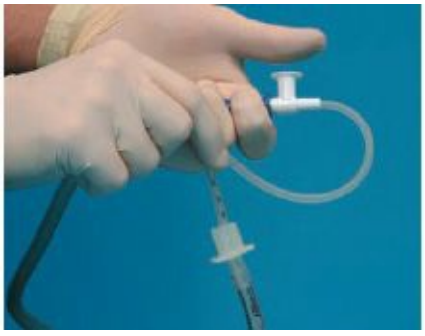
Suctioning a Patient With an Endotracheal Tube in Place

1 Check, prepare, and assemble your equipment.

2 Lubricate the suction catheter.

3 Preoxygenate the patient.

4 Gently insert the catheter into the ET tube until the patient coughs.



5 Suction in a rotating motion while withdrawing the catheter. Monitor the patient's cardiac rhythm and oxygen saturation during this procedure.

6 Reattach the bag-mask device and resume ventilation and oxygenation.

TABLE 6-2 Oxygen vs Approximate FIO_2

Oxygen, L/min	Approximate FIO_2, %
Nasal cannula 1	24
2	27
3	30
4	33
5	36
6	39
Simple face mask, 10	40–60
Nonrebreathing mask, 12–15	100

Supplemental Oxygen Delivery

Gas flow is usually from an area of greater concentration to an area of lesser concentration. Normal inspiration creates negative pressure by contraction of the intercostal muscles and the diaphragm. This expansion creates negative pressure with the thorax causing air to rush in, attempting to equalize intrathoracic pressure. During normal respiration, the amount of air that enters the thorax is approximately 5 to 7 mL/kg.

When gas flow is inadequate, positive-pressure ventilation is required. Delivery of positive-pressure ventilation when spontaneous respiration is inadequate may be lifesaving. Oxygen delivery may take many forms.

- Mouth-to-mouth ventilation: This is one of the oldest forms of artificial ventilation. Its main advantage is that it requires no equipment to deliver. Studies have shown that delivered tidal volumes can exceed those of other types of ventilation. The main drawback is mouth-to-mouth contact and the risk of communicable disease transmission. In actual practice, prehospital providers do not use mouth-to-mouth ventilation while working; alternative ventilation adjuncts should be readily available.
- Barrier device/resuscitation mask: These devices use the same principles of mouth-to-mouth ventilation. The addition of the barrier device reduces the psychological reluctance and minimizes

the transmission of disease **Figure 6-25**.

- Bag-mask ventilation: This is the most commonly used device to deliver supplemental oxygen and can be used with a mask or other invasive airway device **Figure 6-26**.

Advanced Airway Management

Although basic airway maneuvers are a starting point in our assessment and patient care, they are just that—basic skills. They are where you begin. However, these skills are not definitive airway management. Definitive airway management is considered to be the placement of an ET tube or tracheostomy tube within the trachea. This placement facilitates adequate oxygenation and ventilation of the patient. Patients primarily require intubation for two reasons:

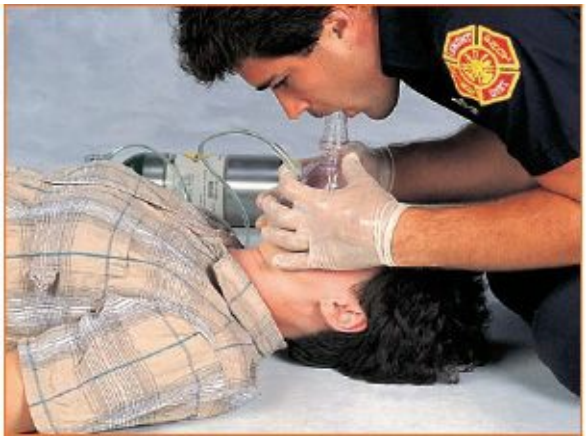


Figure 6-25 Use of a barrier device/resuscitation mask.

Figure 6-26 Use of a bag-mask device.

1. Failure to maintain a patent airway
2. Failure to adequately oxygenate or ventilate

By placing an ET tube within the trachea, the upper airway structures such as the tongue and epiglottis do not have an impact on airway patency. With a cuffed ET tube, aspiration of upper airway secretions and vomitus are minimized. A properly placed and maintained ET tube virtually ensures airway patency. An ET tube also facilitates delivery of oxygen and adequate ventilation. Some indications for ET intubation are as follows:

- Diminished level of consciousness with loss of airway control
  - Absent or diminished gag reflex
  - Glasgow Coma Scale score of 8 or less

- Potential for aspiration (secretions, blood, vomitus)

- Respiratory failure (hypoxemia, hypercarbia)
- Cardiac arrest, after adequate CPR or bag-mask ventilations have been provided

Predicting the Difficult Airway

In the prehospital environment, it is estimated that 20% of all emergency intubations are classified as "difficult." The prehospital provider has to decide how to accomplish airway management. Can I manage this airway at the BLS level? Can I intubate this patient's trachea? There are several factors to consider when treating a difficult airway.

History is one factor. Anatomic findings suggestive of a difficult airway may include congenital abnormalities, recent surgery, trauma, infection, or neoplastic disease (cancer).

A commonly used mnemonic to guide assessment of the difficult airway is LEMON, which stands for:

L	Look externally
E	Evaluate 3-3-2
M	Mallampati
O	Obstruction
N	Neck mobility

As indicated by the "L" in the mnemonic, simply looking at the patient may indicate the relative difficulty that may be encountered in airway management. Patients with short, thick necks may be difficult to intubate. Morbid obesity significantly complicates intubation. Dental conditions such as overbite or "buck" teeth may make intubation difficult.

The "E" in LEMON stands for Evaluate 3-3-2. Three different anatomic measurements are assessed using the **3-3-2 rule** **Figure 6-27**. The first "3" refers to mouth opening. Ideally, a patient's mouth opens at least three fingerwidths (approximately 5 cm). A width of less than three fingers indicates a possibly difficult airway. The second "3" refers to the length of the mandible. At least 3 fingerwidths is optimal. This length is measured from the tip of the chin to the hyoid bone. Smaller mandibles have less room for displacement of the tongue and epiglottis and can make airway management more difficult. The "2" part of this rule refers to the distance (two fingers wide) from the hyoid bone to the thyroid notch.

The "M" is for Mallampati. An anesthesiologist, Mallampati, developed the **Mallampati classification** to predict the relative difficulty of intubation **Table 6-3**. This classification notes the oropharyngeal structures visible in the upright, seated patient who is fully able to open his or her mouth for you. Although this is an accurate predictor of difficult intubation, it is of limited utility in unconscious patients or in patients who cannot follow commands. If a patient is cooperative and able to comply with this evaluation, emergent prehospital intubation is probably not necessary, but the evaluation is important in that it can provide useful information should intubation be needed.

TABLE 6-3 Mallampati Classification

Mallampati Class	Description
I	Entire posterior pharynx fully exposed **Figure 6-28A**

II	Posterior pharynx partially exposed **Figure 6-28B**
III	Posterior pharynx cannot be seen, base of uvula exposed **Figure 6-28C**
IV	No posterior structures can be seen **Figure 6-28D**

The "O" represents obstruction. Anything that might interfere with visualization or tracheal tube placement should be noted. Foreign body, obesity, hematoma, and masses are all examples of situations that can create a difficult airway.

The "N" stands for neck mobility. The ideal position for visualization and intubation is the "sniffing position" with the adult head slightly elevated and extended. The two most common patients with neck mobility problems are trauma patients (due to C-collars or injury) and the elderly (due to osteoporosis or arthritis). The inability to place the patient in the sniffing position can significantly impact the ability to visualize the airway.

Intubation

Once the decision to intubate a patient has been made, the CCTP needs to determine how to most effectively accomplish the procedure. Should the patient be intubated orally or nasally? What size blade should be used? What size ET tube should be used? What should be done if the intubation is unsuccessful? These are all important questions that must be answered.

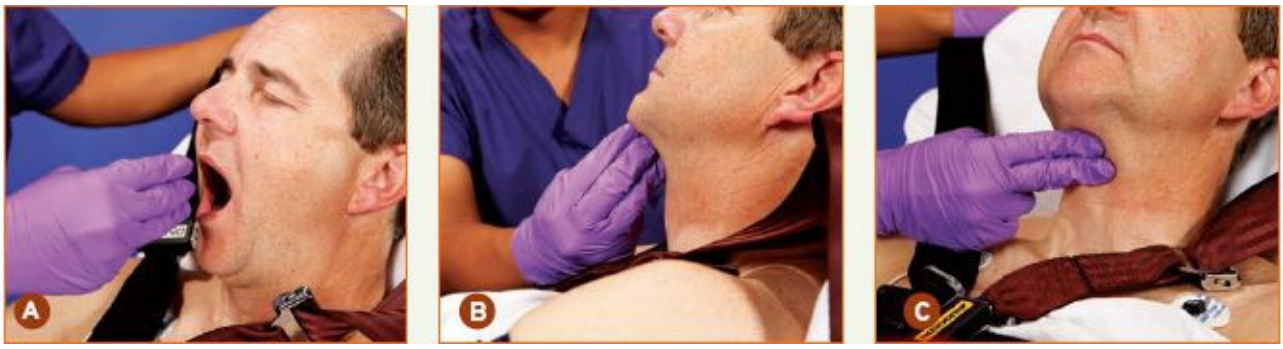


Figure 6-27 The 3-3-2 rule. **A.** The mouth should be at least three fingers wide when open. **B.** The space from the chin to the hyoid bone should be at least three fingers wide. **C.** The distance from the hyoid bone to the thyroid notch should be at least two fingers wide.

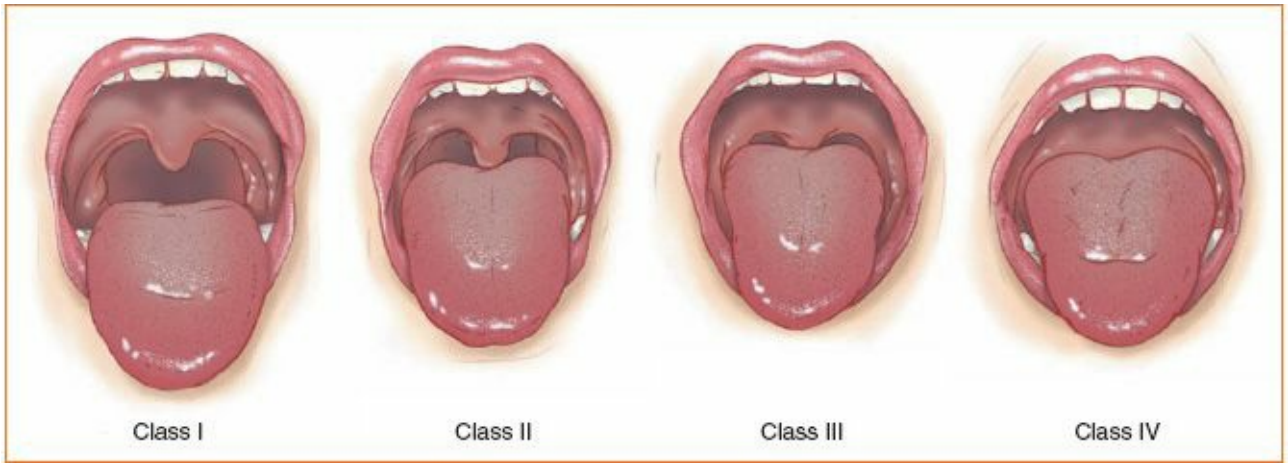


Figure 6-28 Mallampati classification.

The following pages discuss multiple techniques, devices, and procedures for airway management. It is important to abide by the protocols of your state and your service when using airway devices.

Equipment

The following equipment is needed to provide noninvasive advanced airway management.

- Gloves, mask, and goggles
- ET tubes of various sizes
  - Usual size range of 2.0 to 9.0
  - Most adults accept a size 7.0 to 8.0
- Appropriately sized stylet (adult or pediatric)
- Appropriately sized laryngoscope handle and blades (Miller or Macintosh)
- Suctioning equipment
- 10-mL syringe
- Water-soluble lubricant
- Commercial tube holding device
- Age- and size-appropriate bag-mask device with reservoir
- Supplemental oxygen
- Stethoscope
- $ETCO_2$ detector **esophageal detection device (EDD)**, or similar device for confirmation
- Magill forceps
- Topical anesthetic spray (for nasal intubation)
- Rescue airway device (Combitube, King LT, laryngeal mask airway [LMA])
- 20-mL syringe
- 40-mL syringe

Special Populations

In infants, children, and young adults, there are many methods to estimate the appropriate size ET tube to be used.

- For children older than 1 year, calculate the appropriate size by adding 16 plus the child's age in years, then dividing this number by 4.
- Look at the size of the child's small finger nail to estimate the diameter of the tube.
- Use a weight-based chart.

The laryngoscope handles and blades must also be appropriately sized.

- Straight blades: sizes 00-0-1-2-3-4
  - Miller
  - Wisconsin
- Curved blades: sizes 1-2-3-4
  - Macintosh

Many believe the type of blade used can have a significant impact on the success of intubation. For many

older children and adults, the choice of the blade is not as important as the comfort and experience of the CCTP with a particular blade type and the technique used in the intubation attempt. For smaller children, because of age-related anatomic differences, the choice of a straight blade to actually "pick up" the epiglottis may facilitate successful intubation. Remember, the curved blade is placed in the vallecula, which is located at the base of the tongue and displaces the epiglottis indirectly via pressure on the underlying hyoepiglottic ligament. After choosing the appropriately sized handle, blade, and ET tube, the CCTP must visualize the glottic opening and pass the tube. The following considerations need to be made during laryngoscopy.

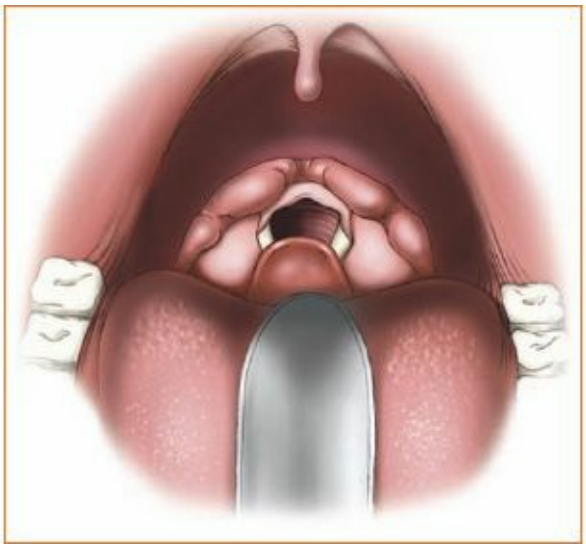


Figure 6-29 Laryngoscopic view of the vocal cords (white fibrous bands).

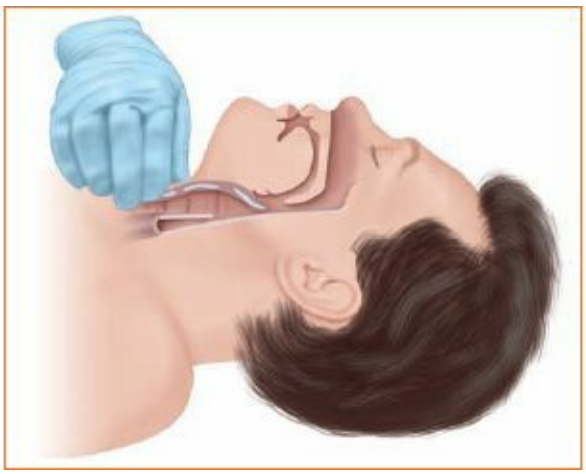


Figure 6-30 Cricoid pressure (Sellick maneuver).

The view obtained at laryngoscopy can be graded. The Cormack-Lehane grading system grades the view of the glottic opening. There are four grades as follows:

- Grade 1: The entire glottic opening is visible.
- Grade 2: The arytenoid cartilages or the posterior portion of the glottic opening is visible.
- Grade 3: Epiglottis only is visible.
- Grade 4: Tongue and/or soft palate only is visible.

Grades 3 and 4 views on laryngoscopy can result in significant difficulty intubating the trachea due to inability to visualize any part of the glottic opening **Figure 6-29**.

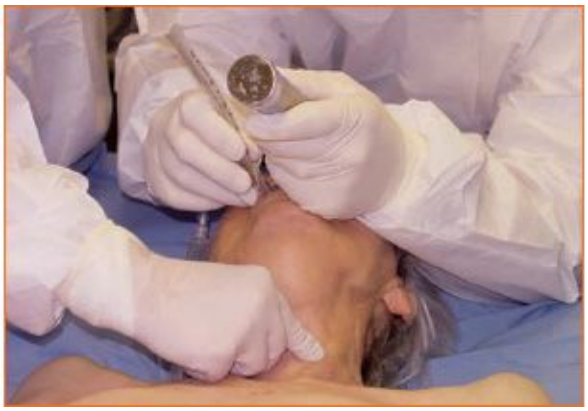


Figure 6-31 BURP maneuver.

Attempts at laryngoscopy should be limited to approximately 30 seconds or less. Lengthy attempts at intubation can result in significant hypoxemia, hypercarbia, and hemodynamic instability.

Cricoid pressure (Sellick maneuver) should be used to minimize passive regurgitation, although this has not been proven, and possibly improve laryngeal view, although this point is debated **Figure 6-30**.

If you are attempting intubation and not visualizing what you need, stop, ventilate, and try something different.

1. Reposition the head, if possible, to elevate the ear to the level of the sternal notch. In some patients, achieving this position may require that multiple blankets be placed under the head and shoulders and/or elevation of the head of the stretcher.
2. If spinal precautions are present, remove the front of the collar while maintaining manual spinal precautions to allow for increased mandibular displacement.
3. Consider performing the BURP maneuver: backward/upward/rightward pressure applied by an assistant to improve laryngeal view **Figure 6-31**. This is also called external laryngeal manipulation. In this technique, the prehospital care provider uses the right hand to manipulate the larynx to improve laryngeal view and then has an assistant maintain this view while passing the ET tube under direct visualization **Figure 6-32**.
4. Consider using a gum elastic bougie or intubating stylet. The gum elastic bougie is a 60-cm long, 15F intubating stylet with a 1-cm/30° bend at the distal tip. Reusable or disposable versions are available in adult and pediatric sizes. The stylet is utilized in epiglottis-only views to facilitate intubation **Figure 6-33**. Rather than attempting to use a standard stylet to "hockey stick" the ET tube and pass it beneath the epiglottis and into the trachea, the bougie is placed blindly, with its shape maximizing the chance of correct placement within the glottis. You may feel a "click" as the tip passes over the tracheal rings, usually stopping at the level of the carina. The ET tube is subsequently passed over this stylet and into the trachea.

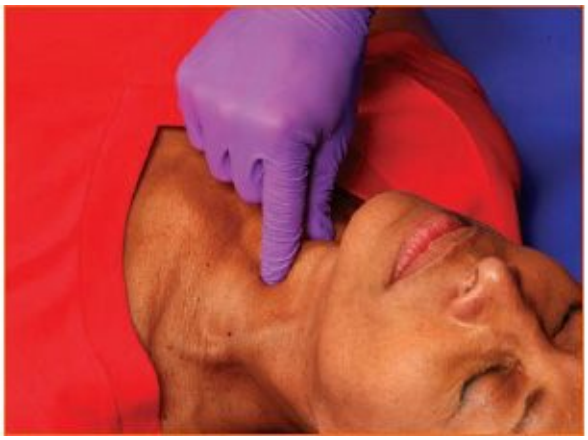


Figure 6-32 External laryngeal manipulation.

Figure 6-33 The gum elastic bougie.

Special Populations

Pregnant patients have a four times higher *failed* intubation rate when compared with nonpregnant patients. Keep the following facts in mind when you are treating a pregnant patient:

1. The airway in pregnant patients is usually more anterior.
2. The airway tissues of a pregnant patient are often friable, and will bleed easily when manipulated.
3. Pregnant patients are at a high risk of aspiration secondary to the hormone relaxin.
4. Pregnant patients have more oral secretions and require more suctioning, even after being intubated.

The following sections discuss the indications, contraindications, and steps for the various types of intubation and other advanced airway maneuvers.

Orotracheal Intubation

Indications for orotracheal intubation include the following:

- Airway control as a result of coma, respiratory arrest, and/or cardiac arrest
- Ventilatory support prior to impending respiratory failure
- Prolonged artificial ventilatory support is required
- Patients without a gag reflex
- Medication administration (lidocaine, atropine, epinephrine, naloxone)
- Impending airway compromise (burns, trauma)

Relative contraindications for orotracheal intubation include:

- Inability to open the mouth because of trauma, dislocation, or pathologic condition
- Epiglottitis

- Inability to see the glottic opening
- Copious secretions, vomit, or blood in the airway

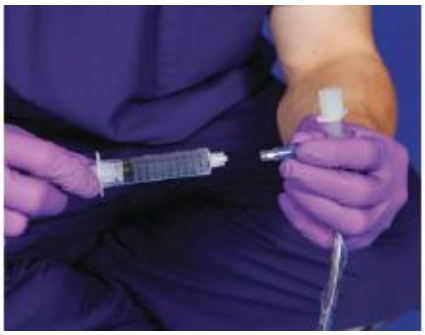
Skill Drill 6-2 shows the steps for orotracheal intubation, which are described as follows:

1. Use BSI precautions.
2. Preoxygenate the spontaneously breathing patient for as long as possible with high-flow oxygen. Attempt to avoid bag-mask ventilations if possible. Avoiding bag-mask ventilation minimizes air insufflation of the stomach and reduces the potential for regurgitation and aspiration **Step 1**.
3. Check, prepare, and assemble your equipment **Step 2**.
4. Choose an appropriately sized tube, with the tube still in the package to maintain sterility. Insert the stylet, lubricate the tube, and bend to the desired position **Step 3**. It is also a good idea to have a tube of a slightly smaller diameter prepared if needed.
5. Auscultate breath sounds prior to intubation.
6. Have Magill forceps and a commercial tube securing device ready.
7. Have a large-bore suction unit at the patient's side and turned on.
8. Place the patient in sniffing position if no trauma is involved **Step 4**. If the patient is a trauma patient, maintain cervical spine immobilization while intubating **Step 5**.
9. Apply cricoid pressure (Sellick maneuver) **Step 6**.
10. Insert the laryngoscope blade into the right side of mouth, sweep the tongue to the left, and visualize the cords **Step 7**.
11. If you are unable to visualize the glottic opening, change cricoid pressure to the BURP maneuver or external laryngeal manipulation.
12. Insert the ET tube to the appropriate depth until the cuff is just below the vocal cords.
13. Inflate the cuff with 10 mL of air and remove the syringe **Step 8**. Check placement with an EDD.
14. Attach the bag-mask device and ETCO2 detector and begin to ventilate **Step 9**.
15. Verify tube placement by auscultating the epigastrium and bilateral chest **Step 10**.
16. Observe for bilateral chest expansion, compliance of the bag-mask device, condensation in the tube, and the proper colorimetric change on the $ETCO_2$ detector.
17. Secure the ET tube with a commercial device or tape **Step 11**.
18. Frequently verify placement of the ET tube, ideally with waveform capnography. If continuous waveform capnography is not available, the tube position should be re-verified, at a minimum, each time the patient is moved.

Skill Drill 6-2

Performing Orotracheal Intubation

1 Use BSI precautions. Preoxygenate the patient.

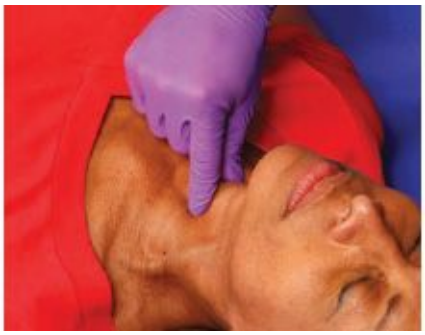


2 Check, prepare, and assemble your equipment.

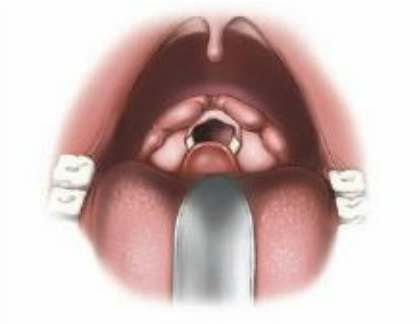
3 Insert the stylet, lubricate the tube, and bend to the desired position. Auscultate breath sounds.

4 Place the patient in a sniffing position if no trauma is involved.

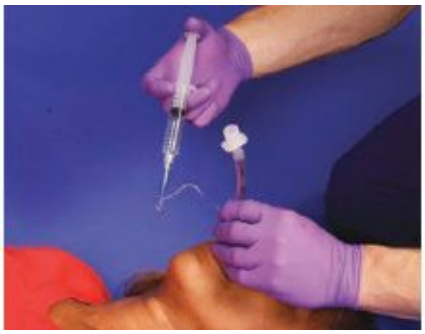
5 If the patient is a trauma patient, maintain cervical spine immobilization while intubating.



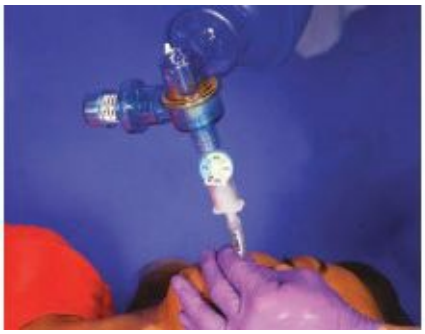
6 Apply cricoid pressure (Sellick maneuver).



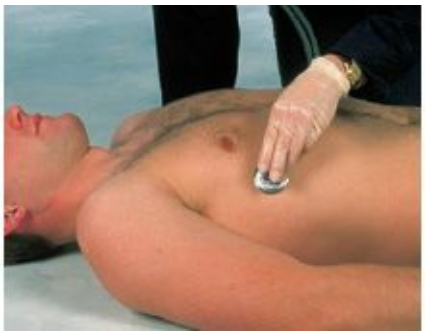
7 Insert the laryngoscope blade into the right side of the mouth, sweep the tongue to the left, and visualize the cords. Insert the ET tube to the appropriate depth until the cuff is just below the vocal cords.



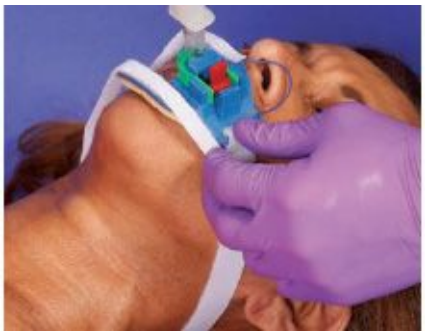
8 Inflate the cuff with 10 mL of air and remove the syringe. Check placement with an EDD.



9 Attach the bag-mask device and ETCO2 detector and begin to ventilate.



10 Auscultate the epigastrium and bilateral chest.



11 Secure the ET tube with a commercial device or tape.

Flight Considerations

When you are performing orotracheal intubation in a pressurized air medical environment, also consider filling the cuff with water to occupy the equivalent volume of 10 mL of air. This decreases the risk of tracheal injury due to overexpansion of the balloon with increased altitudes.

Nasotracheal Intubation

Nasotracheal (nasal) intubation was once the preferred airway management technique in patients suspected of having a spinal cord injury. With the advent of rapid sequence intubation (RSI), nasal intubation is, at best, used infrequently. Most patients who undergo nasotracheal intubation are in the prehospital environment where RSI may not be permitted or are spontaneously breathing but the potential for a difficult airway has been identified, making RSI inadvisable. Nasotracheal intubation also requires a spontaneously breathing patient. Success with nasotracheal intubation may be enhanced by using endotracheal tubes specifically designed for nasotracheal placement (Endotrol tube).

Nasotracheal intubation, however, carries a fairly high failure rate and also takes longer to perform than direct laryngoscopy. Also, prolonged attempts may result in significant hypoxemia and glottic edema

secondary to trauma. The procedure may also cause significant bleeding and vomiting. The risks of hypoxemia and hypercarbia in a patient with inadequate respirations must be weighed against the risks of performing nasal intubation. Combativeness, facial trauma with suspected basilar skull fracture, coagulopathy, and upper airway infection are a few of the relative contraindications to nasotracheal intubation. It also frequently requires placement of an ET tube of a smaller diameter compared with those placed during direct laryngoscopy. Indications for nasotracheal intubation include:

- Patients who are awake and breathing but are in danger of respiratory failure
- Patients with a gag reflex
- Patients who are breathing but cannot open their mouth

Contraindications to nasotracheal intubation include:

- Apneic or near-apneic patients
- Inability to pass the tube through the nostril
- Blood clotting or anticoagulation therapy
- Severe nasal, facial, or basilar skull fractures

Skill Drill 6-3 shows the steps for nasotracheal intubation, which are described as follows:

1. Use body substance isolation (BSI) precautions (gloves and face shield).
2. Preoxygenate the patient whenever possible with 100% oxygen by appropriate delivery device **Step 1**.
3. Check, prepare, and assemble your equipment **Step 2**.
4. Place the patient's head in a neutral position **Step 3**.
5. Select the proper size ET tube and form it into a circle **Step 4**.
6. Apply topical anesthetic spray to nostrils and pharynx **Step 5**.
7. Lubricate the tip of the tube with a water-soluble gel **Step 6**.
8. Release the circle from the ET tube and gently insert into either nostril with the bevel of the tube toward the septum **Step 7**.
9. Advance the tube until the tip passes through the nasopharynx **Step 8**. Listen for breath sounds and look for condensation in the tube.
10. As the tube approaches the larynx, breath sounds will be amplified. Gently and evenly push the tube into the larynx during inspiration.
11. The 15-mm adapter should rest close to the nostril. Passing the tube through the trachea may stimulate the gag reflex. This could result in the patient coughing and bucking. Watch and monitor for vomiting.
12. Inflate the distal cuff with 5 to 10 mL of air and detach the syringe **Step 9**. Check placement with an EDD.
13. Attach an $ETCO_2$ detector to the ET tube **Step 10**.
14. Attach the bag-mask device and ventilate. Verify tube placement by auscultating the chest bilaterally and over the epigastrium **Step 11**.
15. Secure the ET tube.

Skill Drill 6-3

Performing Nasotracheal Intubation

1 Use BSI precautions (gloves and face shield). Preoxygenate the patient.

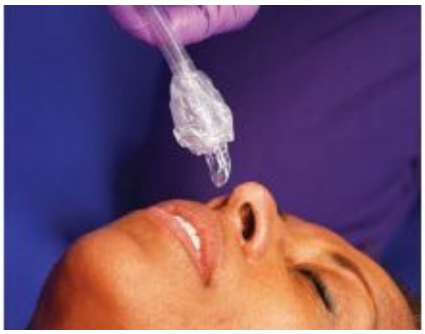
2 Check, prepare, and assemble your equipment.

3 Place the patient's head in a neutral position.

4 Select the proper size ET tube and form it into a circle.

5 Apply topical anesthetic spray to nostrils and pharynx.

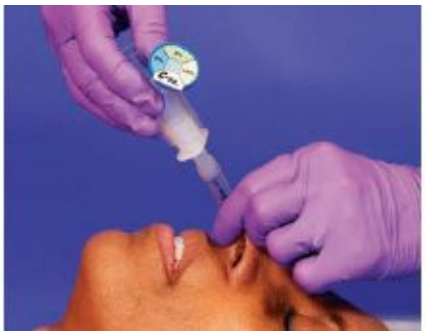
6 Lubricate the tip of the tube with a water-soluble gel.



7 Release the circle from the ET tube and gently insert into either nostril with the bevel of the tube toward the septum.

8 Advance the tube until the tip passes through the nasopharynx. Listen for breath sounds and look for condensation in the tube.

9 Inflate the distal cuff with 5 to 10 mL of air and detach the syringe. Check placement with an EDD.



10 Attach an $ETCO_2$ detector to the ET tube.

11 Attach the bag-mask device and ventilate. Auscultate the chest bilaterally and over the epigastrium. Secure the ET tube.

Digital Intubation

Digital intubation was one of the first techniques developed to facilitate the placement of an ET tube. With this technique, the epiglottis is manually elevated using the middle finger and the ET tube is guided into place by feel. Advantages are that other than an ET tube, no additional equipment is needed. Also, in confined space incidents when laryngoscopy may not be possible or a laryngoscope may not be available, such as during a building collapse or entrapment, digital intubation may be life saving. The major limitation of digital intubation is safety of the provider. Any patient who undergoes digital intubation must be profoundly unconscious without airway reflexes, or some type of bite block must be used to prevent injury.

Indications for digital intubation include:

- Deeply unconscious patients who are apneic and without a gag reflex When other techniques have failed, the patient is obese, or the patient has a short neck
- A laryngoscope is not available or the patient is in a confined space

- Oral secretions are obscuring the view, and the head cannot be moved due to trauma, or immobilization equipment is complicating other techniques
- Massive trauma has made identification of intubation landmarks impossible

Contraindications to digital intubation include:

- Patients with a gag reflex
- Inability to open mouth due to trauma, dislocation, fracture, or pathologic condition

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Skill Drill 6-4 shows the steps for digital intubation, which are described as follows. Only attempt this maneuver if the patient is deeply comatose and oral and nasal intubation have failed or are not possible.

1. Use BSI precautions (gloves and face shield) **Step 1**.
2. Kneel next to the patient. Preoxygenate the patient for as long as possible with 100% oxygen via appropriate delivery device **Step 2**.
3. Check, prepare, and assemble your equipment **Step 3**.
4. Bend the ET tube by placing a slight curve at its distal end (like a hockey stick) **Step 4**.
5. Place the patient's head in a neutral position **Step 5**.
6. Place a bite block in between the patient's molars to prevent the patient from biting your fingers **Step 6**.
7. Insert your left index and middle fingers into the patient's mouth and shift the patient's tongue forward as you advance your fingers toward the larynx **Step 7**.
8. Palpate and lift the epiglottis with your left middle finger **Step 8**.
9. Advance the tube between your fingers with your right hand and into the trachea **Step 9**. Guide it in between the vocal cords with your left index finger.
10. Remove the stylet from the ET tube **Step 10**.
11. Inflate the distal cuff of the ET tube with 5 to 10 mL of air and detach the syringe **Step 11**.
12. Attach the $ETCO_2$ detector to the ET tube **Step 12**.
13. Attach the bag-mask device and ventilate. Verify tube placement by auscultating the chest bilaterally and over the epigastrium **Step 13**.
14. Secure the ET tube **Step 14**.

Retrograde Intubation

When intubation is unsuccessful by standard means, the technique of **retrograde intubation** may be utilized. In retrograde intubation, a needle is placed percutaneously within the trachea via the cricoid membrane. A wire is placed cephalad through the needle upward through the trachea and into the mouth. The wire is then visualized and secured, and the ET tube is placed over the wire and guided into the trachea. The wire is subsequently removed and the ET tube is advanced and secured.

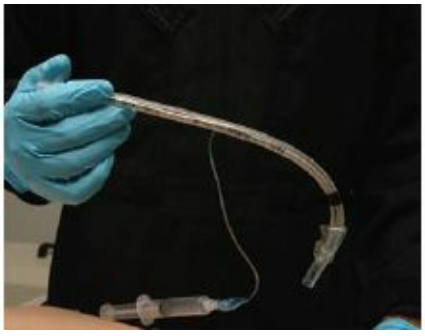
Skill Drill 6-4

Performing Digital Intubation

1 Use BSI precautions (gloves and face shield).

2 Kneel next to the patient and preoxygenate.

3 Check, prepare, and assemble your equipment.

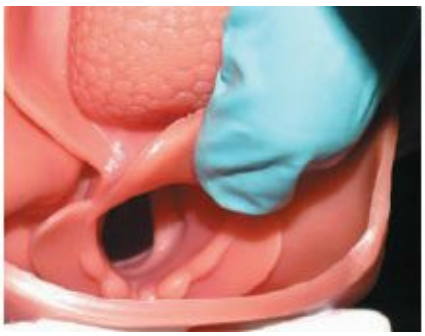


4 Bend the ET tube by placing a slight curve at its distal end (like a hockey stick).

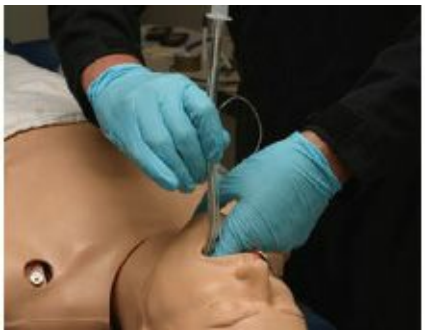
5 Place the patient's head in a neutral position.

6 Place a bite block in between the patient's molars to prevent the patient from biting your fingers.

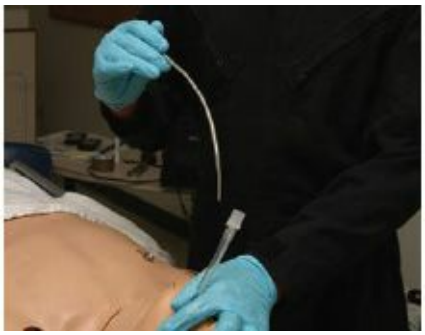
7 Insert your left index and middle fingers into the patient's mouth and shift the patient's tongue forward as you advance your fingers toward the larynx.



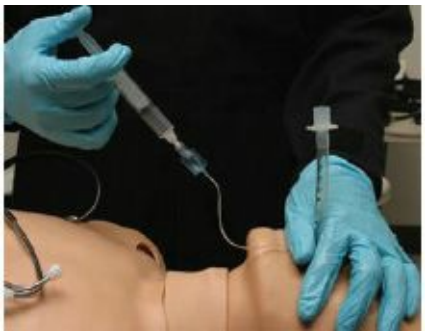
8 Palpate and lift the epiglottis with your left middle finger.



9 Advance the tube between your fingers with your right hand and into the trachea.



10 Remove the stylet from the ET tube.



11 Inflate the distal cuff of the ET tube with 5 to 10 mL of air and detach the syringe.

12 Attach the $ETCO_2$ detector to the ET tube.

13 Attach the bag-mask device and ventilate. Auscultate the chest bilaterally and over the epigastrium.

14 Secure the ET tube.

Indications for retrograde intubation include:

- Dyspnea
- Obstruction
- Secretions
- Failure to intubate the trachea by other less invasive methods Contraindications to retrograde intubation include:
- Lack of familiarity with procedure
- Laryngeal trauma
- Unrecognizable or distorted landmarks
- Coagulopathy (relative)
- Severe hypoxia (due to inability to ventilate during procedure and time to perform procedure)

Complications of retrograde intubation include:

- Hypoxemia
- Cardiac arrhythmias
- Mechanical trauma
- Infection
- Increased intracranial pressure

Because retrograde intubation is essentially a different technique for placing the ET tube, assessment findings and transport complications with retrograde intubation are the same as with standard ET intubation.

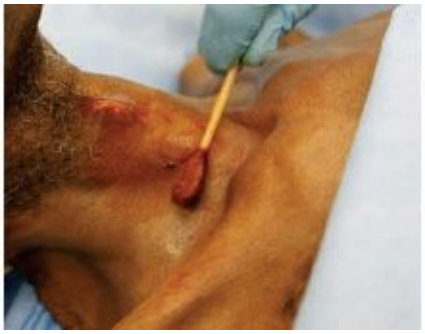
Skill Drill 6-5 shows the steps for retrograde intubation, which are described as follows:

1. Place the patient supine.
2. Ventilate the patient with high-flow oxygen via the appropriate device while preparing the equipment and the patient.
3. Cleanse the anterior part of the neck from the laryngeal prominence to just below the cricoid ring and position a fenestrated drape **Step 1**.

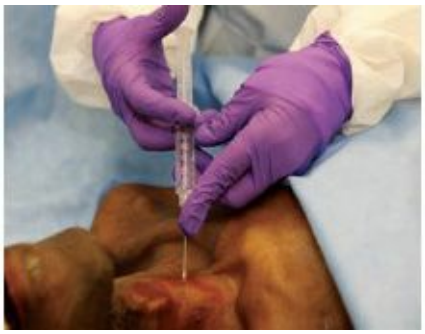
4. If the patient is conscious, consider numbing the area over the cricothyroid membrane using a local anesthetic **Step 2**.
5. Puncture the cricothyroid membrane using a large needle aligned with the airway and pointed approximately 30° cephalad from the perpendicular at the level of the cricothyroid membrane **Step 3**.
6. Identify the tracheal lumen by aspiration of air into the syringe attached to the needle **Step 4**.
7. Pass the 70-cm guide wire through the catheter until it appears in the oropharynx, mouth, or one of the nares **Step 5**.
8. If guide wire is in the oropharynx, grasp it with a clamp or Magill forceps and pull the wire partially out of the mouth, ensuring that the distal end is still emerging from the neck and the wire is pulled taut **Step 6**.
9. Insert the guide wire emerging from the mouth, through Murphy's Eye of the ET tube, and pass through the lumen of the ET tube **Step 7**.
10. Advance the ET tube into the trachea **Step 8**.
11. Verify tube placement by auscultating the chest bilaterally and over the epigastrium **Step 9**.
12. Secure the ET tube in place and ventilate **Step 10**.
13. Once tube placement is confirmed, remove the guide wire by pulling on the distal end emerging from the neck, then advance the tube 2 to 3 cm futher **Step 11**.
14. If tube placement is incorrect, remove and attempt to ventilate. If ventilating adequately, continue to ventilate with high-flow oxygen and reassess. Determine if additional attempts at retrograde intubation are warranted or if another means of securing the airway is necessary (cricothyrotomy) **Step 12**.

Skill Drill 6-5

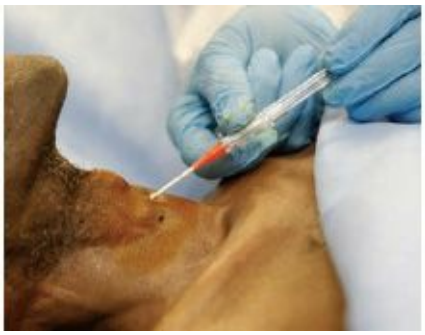
Performing Retrograde Intubation



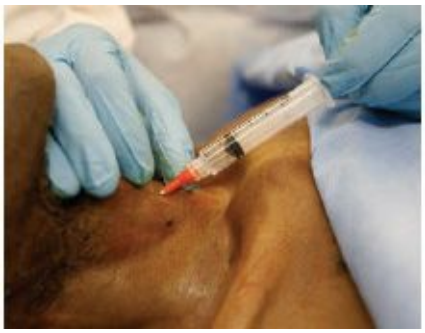
1 Place the patient supine. Ventilate the patient while preparing the equipment and the patient. Cleanse the anterior part of the neck from the laryngeal prominence to just below the cricoid ring and position a fenestrated drape.



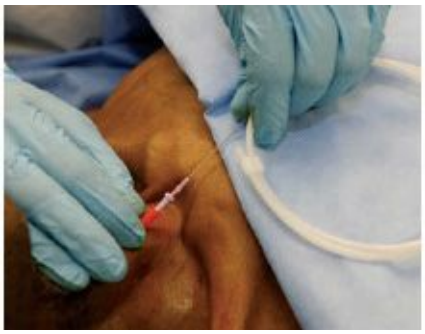
2 Numb the area over the cricothyroid membrane using a local anesthetic.



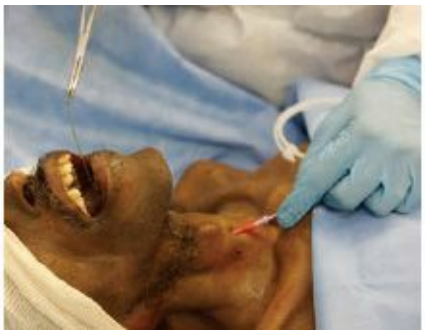
3 Puncture the cricothyroid membrane using a large needle aligned with the airway and pointed approximately 30° cephalad from the perpendicular at the level of the cricothyroid membrane.



4 Identify the tracheal lumen by aspirating the syringe attached to the needle.



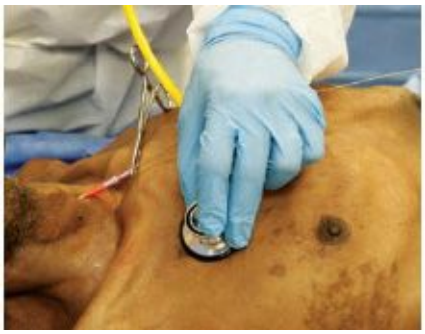
5 Pass the 70-cm guide wire through the catheter until it appears in the oropharynx, mouth, or one of the nares.



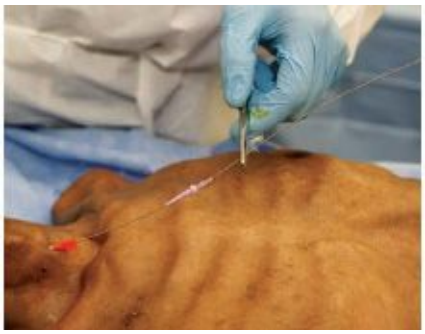
6 If guide wire is in the oropharynx, grasp it with a clamp and pull the wire partially out of the mouth, ensuring that the distal end is still emerging from the neck and the wire is pulled taut.

7 Insert the guide wire emerging from the mouth, through Murphy's Eye, and pass through the lumen of the ET tube.

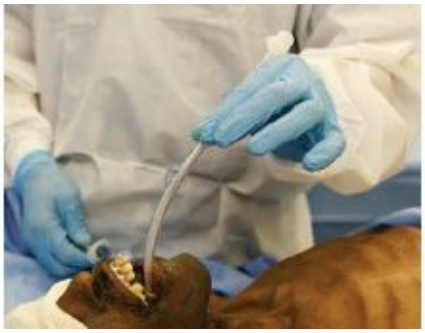
8 Advance the ET tube into the trachea.



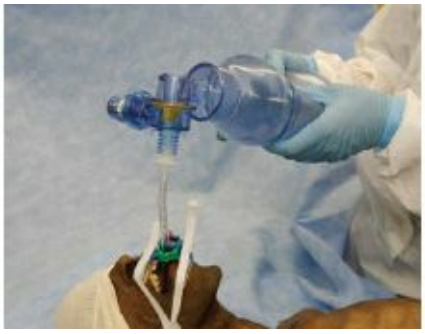
9 Auscultate the chest bilaterally and over the epigastrium.



10 Secure the ET tube in place and ventilate.



11 Once tube placement is confirmed, remove the guide wire by pulling on the distal end emerging from the neck.



12 If tube placement is incorrect, remove the tube and start over or switch to a different technique.

Face-to-Face Intubation

Intubation may be performed with the provider's face at the same level as the patient's face when other positions are not possible—for example in a motor vehicle collision where the patient is in a seated position or in a tight space where the space above the head cannot be accessed. This is called **face-to-face intubation**, or the "Tomahawk" method. The procedure is the same as orotracheal intubation except for the following:

- The patient's head cannot be placed in the sniffing position. It is manually stabilized by a second provider during the entire procedure.
- Hold the laryngoscope (with a Macintosh blade) in your right hand with the blade facing downwards like a hatchet, while holding the ET tube in your left hand. Insert the laryngoscope blade into the right side of patient's mouth, sweep the tongue to the patient's left, and visualize the cords.
- Once the laryngoscope blade has been placed, the provider who is intubating may slightly adjust the patient's head for better visualization by pulling the mandible forward while pressing down.

Transillumination

With **transillumination**, a light source placed within the trachea is able to be visualized through the thin tissue that covers the trachea **Figure 6-34**. Direct visualization of the glottis is not required with this procedure. However, a specific piece of equipment is required, and it may be difficult to perform this procedure in morbidly obese patients and in patients with short necks because of increased amounts of soft tissue overlying the trachea, or in brightly lit conditions where the transillumination is difficult to see.

Figure 6-34 In transillumination intubation, a lighted stylet is inserted into the ET tube.

Complications of Intubation

Misplaced ET Tube

In the event of a misplaced ET tube, do the following:

- If breath sounds are not heard bilaterally but are heard over the epigastrium, deflate the cuff and extubate. Suction the airway and repeat the steps for oral intubation.
- If breath sounds are heard on the right side only, deflate the cuff and retract the ET tube until you can auscultate breath sounds bilaterally. Reinflate the cuff, resecure the tube, and auscultate breath sounds again.

Failed Intubation

It is estimated that, overall, less than 2.5% of attempted intubations result in failure, although several dedicated EMS studies seem to indicate that the failure rate in the prehospital environment is higher. It is further estimated that less than 1% of attempted intubations result in the critical "can't intubate/can't ventilate" scenario. A failed airway attempt is defined as the failure to maintain acceptable oxygen saturations during or after one or more failed intubation attempts or a total of three failed intubation attempts by an experienced intubator, even when the oxygen saturations can be maintained. Ways to minimize complications of airway management have been discussed. However, as stated earlier, you frequently do not have a choice in the prehospital environment to manage the airway. So, what do you do? There are many rescue airway techniques available.

- Perform simple BLS airway maneuvers with an OPA and/or an NPA and a bag-mask device. With good technique, adequate oxygenation and ventilation can be provided.
- Consider using a laryngeal mask airway (LMA), discussed next, or another blind insertion airway device.

The LMA

The LMA was designed by a British anesthesiologist, Archie Brain, for use as an alternative to mask ventilation and intubation in the operating room. The LMA has been advocated for use as a rescue airway

in the setting of failed intubation in both the emergency and EMS environments. LMAs have various designs, some of which allow an ET tube to be passed through it or a tube to decompress the stomach. The basic design is similar to an ET tube at the proximal end in that a standard adapter is present to allow ventilation. The distal end is equipped with an elliptical cuff, which, when inflated, covers the supraglottic area and allows ventilation.

An advantage of the LMA is ease of insertion because placement of the device does not require laryngoscopy. It also provides superior oxygenation and ventilation compared with bag-mask ventilation. Some disadvantages associated with LMA use are the risk of aspiration and difficulty with obtaining an adequate seal, allowing a loss of tidal volume and gastric insufflation.

Indications for LMA use include:

- Deep coma, cardiac arrest, and/or respiratory arrest
- When ET intubation is not possible or available Contraindications to LMA use include:
- Patients with a gag reflex
- Facial and/or esophageal trauma
- Suspected foreign body airway obstruction

Skill Drill 6-6 shows the steps for LMA placement, which is described as follows:

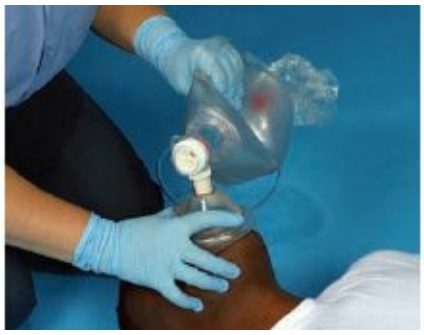
1. Check the cuff of the LMA by inflating it with 50% more air than is required for that size airway. Then deflate the cuff completely **Step 1**.
2. Apply a water-soluble lubricant to the base of the device **Step 2**.
3. Preoxygenate the patient with a bag-mask device and 100% oxygen. Ventilation should not be interrupted for more than 30 seconds to accomplish LMA placement **Step 3**.
4. Assemble and check equipment.
5. Place the head in the sniffing position. Insert your finger between the cuff and the tube. Place the index finger of your dominant hand in the notch between the tube and the cuff. Open the patient's mouth **Step 4**.
6. Insert the LMA along the roof of the mouth with the aperture of the mask facing the tongue and the back of the mask against the roof of the mouth **Step 5**. Use your finger to push the airway against the hard palate.
7. Blindly push until resistance is felt.
8. The black line on the tube shaft should be opposite the upper lip.
9. Inflate the cuff rim of the mask with the amount of air indicated for that size airway. Remove the syringe **Step 6**.
10. Attach the bag-mask device and begin to ventilate the patient. Check for chest rise and lung or epigastrium sounds **Step 7**. Continuously monitor the patient.

Skill Drill 6-6

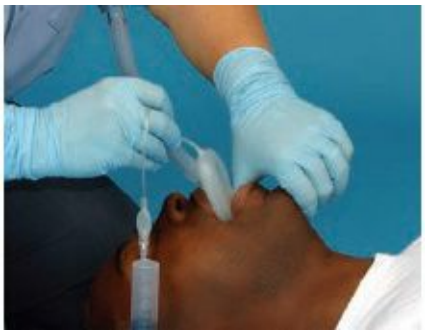
LMA Insertion

1 Check the cuff of the LMA by inflating it with 50% more air than is required for that size airway. Then deflate the cuff completely.

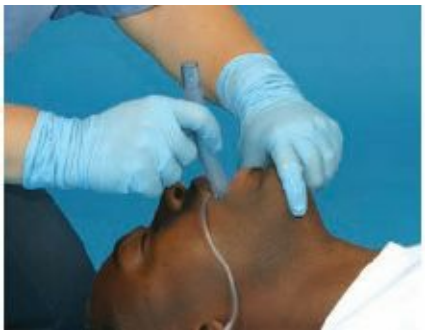
2 Apply a water-soluble lubricant to the base of the device.



3 Preoxygenate the patient. Ventilation should not be interrupted for more than 30 seconds to accomplish LMA placement.

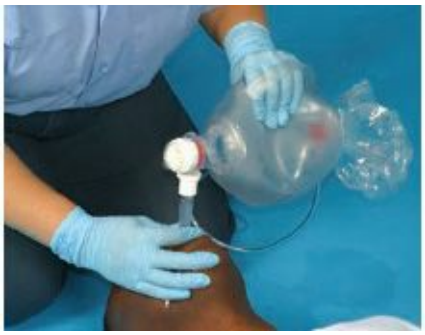


4 Place the head in the sniffing position. Insert your finger between the cuff and the tube. Place the index finger of your dominant hand in the notch between the tube and the cuff. Open the patient's mouth.



5 Insert the LMA along the roof of the mouth with the aperture of the mask facing the tongue and the back of the mask against the roof of the mouth.

6 Inflate the cuff with the amount of air indicated for that size airway.



7 Attach the bag-mask device and begin to ventilate the patient. Check for chest rise and lung or epigastrium sounds.

Esophageal Tracheal Combitube

Several supraglottic airways are available that can be used as rescue airways. The Combitube is a dual-lumen, dual-cuffed supraglottic airway that may be placed blindly. Placement occurs mostly within the esophagus. However, its dual-lumen design results in successful ventilation regardless of whether the placement is esophageal or, less likely, tracheal.

Indications for Combitube use are:

- When ET intubation is not possible, not available, or not successful
- As a rescue airway in failed intubation, especially in the setting of failed RSI. If the Combitube is used as a rescue airway after RSI, remember, the patient must be kept sedated and paralyzed once proper placement has been verified.
- Deep coma, cardiac arrest, and/or respiratory arrest
- To reduce the risk of gastric distention

Contraindications for the use of the Combitube are:

- Patients with a gag reflex
- Upper airway obstruction or suspected foreign body airway obstruction
- Facial and/or esophageal trauma
- Known esophageal disease
- Possibly caustic ingestions
- Children younger than 16 years
- Anyone shorter than 4′ (use the small adult [SA] Combitube for adults between 4′ and 6′ tall, and use the regular Combitube for adults taller than 6′)

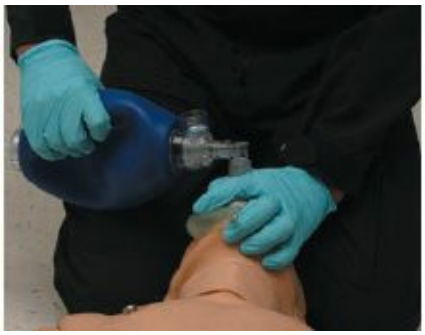
Skill Drill 6-7 shows the steps for Combitube placement, which are described as follows:

1. Use BSI precautions (gloves and face shield) **Step 1**.
2. Preoxygenate the patient with a bag-mask device and 100% oxygen **Step 2**.

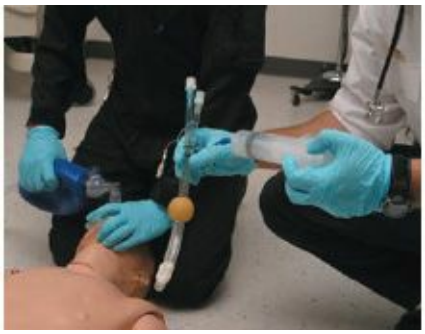
Skill Drill 6-7

Insertion of the Combitube

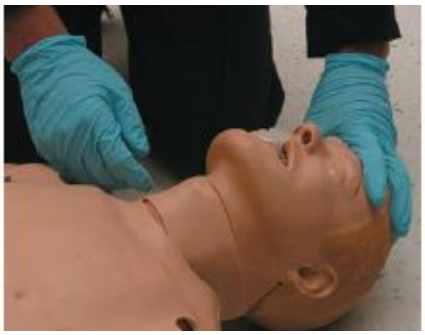
1 Use BSI precautions (gloves and face shield).



2 Preoxygenate the patient.

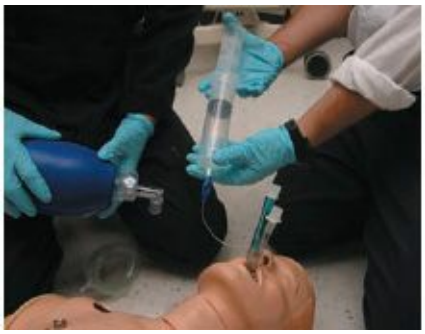


3 Assemble and check equipment.



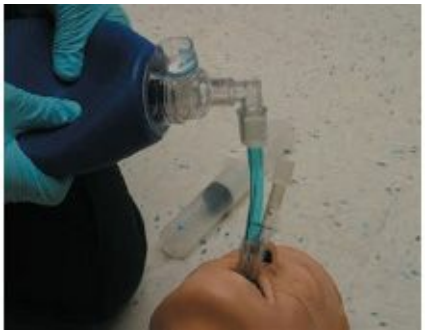
4 Apply water-soluble lubricant to the tube. Place the patient's head in the sniffing position.

5 Open the patient's mouth with the tongue-jaw lift maneuver and insert the Combitube in the midline of the patient's mouth. Gently insert the tube until the incisors lie between the two reference marks.

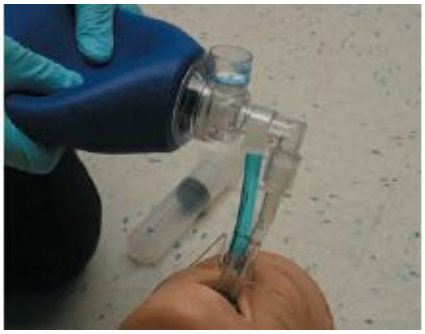


6 Inflate line 1 (blue pilot balloon) leading to the pharyngeal cuff with 100 mL of air and remove syringe.

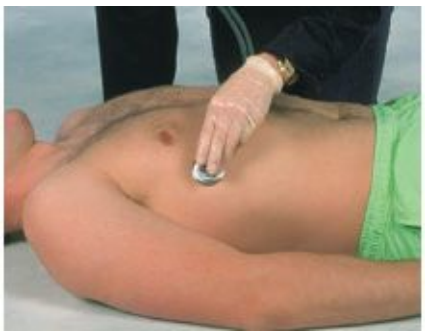
7 Inflate line 2 (white pilot balloon) leading to the distal cuff with approximately 15 mL of air and remove syringe.



8 Ventilate the patient through the pharyngeal (blue) tube first. Look for chest rise, which indicates esophageal placement of the distal tip, and listen for breath sounds and epigastric sounds.



9 If the chest does not rise and epigastric sounds are present, this indicates tracheal placement. Attempt ventilation through the shorter, clear tube. If it has entered the trachea, the chest should rise.



10 Confirm placement by auscultating for breath sounds over the lungs and gastric sounds over the abdomen.

3. Assemble and check equipment **Step 3**.
4. Familiarize yourself with the various tubings in the airway. Identify the long and the short tube prior to insertion.
5. Apply water-soluble lubricant to the tube.
6. Place the patient's head in the sniffing position **Step 4**.
7. Open the patient's mouth with the tongue-jaw lift maneuver and insert the Combitube in the midline of the patient's mouth. Gently insert the tube until the incisors lie between the two reference marks **Step 5**.
8. Inflate line 1 (blue pilot balloon) leading to the pharyngeal cuff with 100 mL of air and remove syringe **Step 6**.
9. Inflate line 2 (white pilot balloon) leading to the distal cuff with approximately 15 mL of air and remove syringe **Step 7**.
10. Ventilate the patient through the pharyngeal (blue) tube first. Look for chest rise, which indicates esophageal placement of the distal tip, and listen for breath sounds and epigastric sounds **Step 8**.
11. *If the chest rises, breath sounds are present, and epigastric sounds are not present* , continue to ventilate through the longer, blue tube.
12. *If the chest does not rise, no breath sounds are present, and epigastric sounds are present* , this indicates tracheal placement of the Combitube. Attempt ventilation through the shorter, clear tube. If it has entered the trachea, the chest should rise. Continue to ventilate through the shorter clear tube **Step 9**.

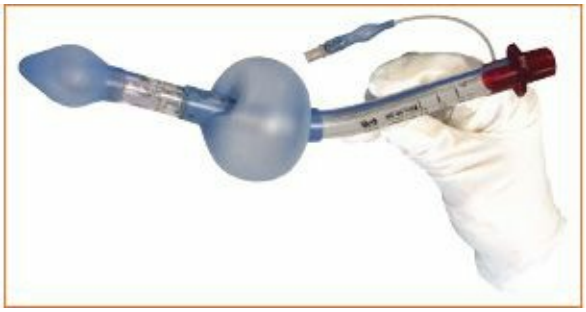


Figure 6-35 King LT airway.

13. Confirm placement by auscultating for breath sounds over the lungs and gastric sounds over the abdomen **Step 10**.
14. After determining which tube is to be used for ventilating, cover the end of the tube not being used with a glove or the enclosed adapter to ensure ventilation through the proper tube.

Another type of supraglottic airway includes the King LT airway, which is a single-lumen, dual-cuff supraglottic airway that is inserted blindly and functions similar to the Combitube **Figure 6-35**. Also, the cuffed oropharyngeal airway is an OPA with the addition of a high-volume, low-pressure cuff and proximal airway adapter that permits positive-pressure ventilation.

Surgical Airways

If you encounter a patient who cannot be intubated or ventilated, the patient requires placement of an airway by other means. This frequently means some type of surgical airway. First, an adequate understanding of the anatomy of the anterior part of the neck is needed.

The most prominent structure in the anterior part of the neck is the thyroid cartilage (Adam's apple). It is more easily palpable in males than in females. Directly inferior to the thyroid cartilage is the cricoid cartilage. The cricothyroid membrane lies between these two structures. A thin membrane covered only by skin, the cricothyroid membrane is an important landmark to identify in the setting of a surgical airway.

Equipment

The following equipment is needed to provide invasive advanced airway management.

- ET tube or tracheostomy tube (various sizes)
- Scalpel
- Curved hemostats
- Suction equipment
- 14-gauge or larger over-the-needle catheter
- ¼′ tape
- 10-mL syringe
- Three-way stop cock
- Two pieces of standard oxygen tubing, 4′ to 5′ each
- Y-connector
- Oxygen cylinder (coupled with a 50-psi step-down regulator and a needle flow meter)
- Povidone-iodine swabs
- Body surface isolation, including sterile gloves
- Sterile fenestrated drape (hole in center)
- 4″ × 4″ gauze pads
- Bag-mask device
- Guide wire (70 cm)
- Cotton tie or commercial trach holder

Needle Cricothyrotomy

A surgical airway is used to provide temporary oxygenation when intubation by other means is not possible. A surgical airway may be needed in the event of the unusual situation in which the patient cannot be intubated or ventilated. In the pediatric population younger than 12 years, in whom a formal cricothyrotomy is contraindicated, needle cricothyrotomy is the surgical airway of choice in the emergent setting. In a needle cricothyrotomy, a large-bore angiocath (14 to 16 gauge) is placed through the cricothyroid membrane into the trachea. With a transtracheal jet ventilator, it is possible to provide temporary oxygenation. Exhalation is passive and requires supraglottic airway patency. Needle cricothyrotomy does not allow optimal ventilation, only oxygenation, so hypercarbia is an expected complication. It also does not protect the airway or allow suctioning. It is a temporary airway until a more definitive airway can be placed.

Indications for needle cricothyrotomy include:

- Intubation is not feasible
- Intubation does not relieve obstruction
- Field procedure is to establish a temporary airway

Contraindications to needle cricothyrotomy include:

- Patients with severe airway obstruction below the site of the catheter insertion

Complications of needle cricothyrotomy are:

- Hemorrhage
- Subcutaneous emphysema
- Infection
- Misplacement of cannula
- Accidental removal
- Subglottic stenosis
- Mediastinal emphysema
- Tracheal and esophageal laceration
- **Barotrauma**

Skill Drill 6-8 shows the steps for needle cricothyrotomy, which are described below:

1. Use BSI precautions (gloves and face shield) **Step 1**.
2. Attach a 14- to 16-gauge IV catheter to a 10-mL syringe containing approximately 3 mL of sterile saline or water **Step 2**.
3. With the patient's head in a neutral position, palpate for and locate the thyroid cartilage, cricothyroid membrane, and suprasternal notch **Step 3**.
4. Cleanse the area with an iodine-containing solution **Step 4**.
5. Attach the syringe (if preferred) to the needle.
6. Stabilize the larynx and insert the needle into the cricothyroid membrane at a 45° angle toward the feet **Step 5**.
7. Push the needle until it "pops" into trachea. Aspirate with the syringe to determine correct catheter placement **Step 6**.
8. Confirm tracheal placement by noting movement of air.
9. Advance the catheter over the needle until the hub rests against the skin **Step 7**.
10. Place the syringe and needle in a puncture-proof container **Step 8**.
11. Connect one end of the oxygen tubing to the catheter and the other end to the jet ventilator **Step 9**.
12. Open the release valve on the jet ventilator and adjust the pressure accordingly to provide adequate chest rise **Step 10**. If a jet ventilator is unavailable, temporary ventilation can be provided with a bag-valve-mask device by attaching a 15-mm BVM adapter from a 3-0 endotracheal tube to the catheter, or by attaching a 3-mL syringe with the plunger removed and an adapter from a 7-0 tube.
13. Auscultate the apices and bases of both lungs and over the epigastrium to confirm correct catheter placement **Step 11**.
14. Secure the catheter with a 4″ × 4″ gauze pad and tape. Continue ventilations while frequently reassessing for adequate ventilations and any potential complications **Step 12**.

Surgical Cricothyrotomy

There are many techniques for performing a surgical cricothyrotomy. Techniques range from a fully open technique using a scalpel and an ET tube or tracheostomy tube to modified techniques using various premanufactured kits. Availability of kits depends on the state of practice and medical command

physician. The advantages of an open cricothyrotomy are speed of insertion compared with formal tracheostomy and improved oxygenation and ventilation compared with needle cricothyrotomy.

Indications for surgical cricothyrotomy include:

- Intubation is not feasible
- Intubation does not relieve obstruction
- Field procedure to establish a temporary airway Contraindications to surgical cricothyrotomy are:
- Inability to identify anatomic landmarks, usually secondary to trauma
- Pediatric patients younger than 8 years. Children younger than 8 have a smaller cricothyroid membrane that generally cannot accept a larger bore tube, making needle cricothyrotomy the preferred procedure.

Skill Drill 6-8

Performing Needle Cricothyrotomy

1 Use BSI precautions (gloves and face shield).

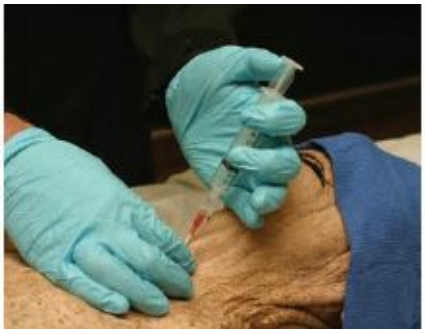
2 Attach a 14- to 16-gauge IV catheter to a 10-mL syringe containing approximately 3 mL of sterile saline or water.

3 With the patient's head in a neutral position, palpate for and locate the thyroid cartilage,

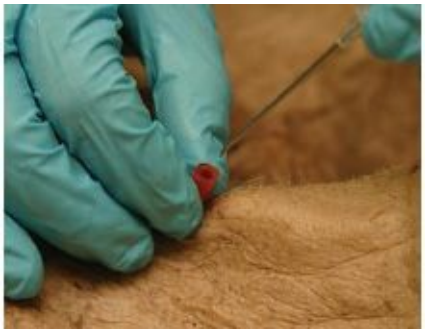
cricothyroid membrane, and suprasternal notch.

4 Cleanse the area with an iodine-containing solution.

5 Attach the syringe (if preferred) to the needle. Stabilize the larynx and insert the needle into the cricothyroid membrane at a 45° angle toward the feet.

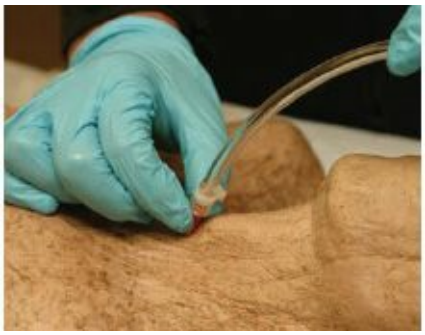


6 Push the needle until it "pops" into trachea. Aspirate with the syringe to determine correct catheter placement.



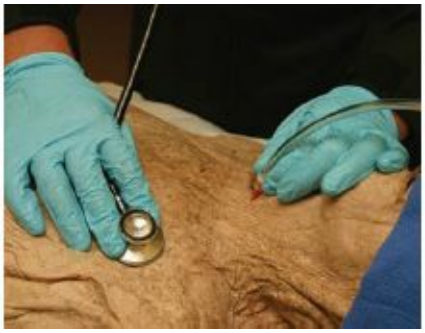
7 While holding the catheter, slide the catheter off of the needle until the hub of the catheter is flush with the patient's skin.

8 Place the syringe and needle in a puncture-proof container.



9 Connect one end of the oxygen tubing to the catheter and the other end to the jet ventilator.

10 Open the release valve on the jet ventilator and adjust the pressure accordingly to provide adequate chest rise.



11 Auscultate the apices and bases of both lungs and over the epigastrium to confirm correct catheter placement.

12 Secure the catheter with a 4″ × 4″ gauze pad and tape. Continue ventilations while frequently reassessing for adequate ventilations and any potential complications.

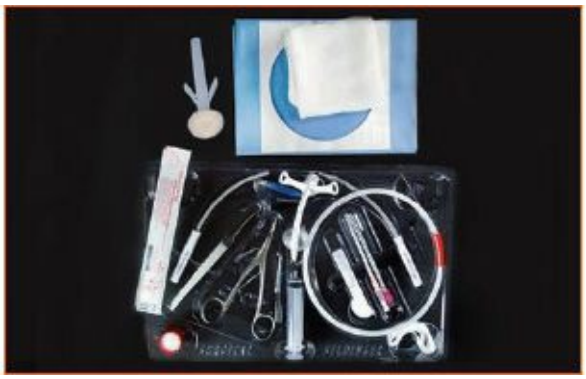


Figure 6-36 The Cook critical care Melker cricothyrotomy catheter kit.

Complications to surgical cricothyrotomy include: Hemorrhage

- Infection
- Misplacement of cannula/tube
- Accidental removal
- Subglottic stenosis
- Mediastinal emphysema
- Tracheal and esophageal laceration

There are several types of surgical cricothyrotomies. Open cricothyrotomy involves the incising of the patient's skin and cricothyroid membrane with a scalpel and placing an ET tube or tracheostomy tube. A modified cricothyrotomy is another type of cricothyrotomy. Several commercial modified cricothyrotomy kits are available, many of which use a modification of the **Seldinger technique** to enable placement of the airway. The Seldinger technique uses a needle and guide wire/guide catheter or tube placement in blood vessels or other hollow organs **Figure 6-36**. Other devices for performing cricothyrotomy, such as the Nu-Trake and Pertrach, are commercially manufactured airway placement devices that may be used in the prehospital environment. These devices do not use the Seldinger technique; they use a device that functions both as an introducer and an airway **Figure 6-37**.

Skill Drill 6-9 shows the steps for surgical cricothyrotomy, which are described as follows:

1. Use BSI precautions (gloves and face shield) **Step 1**.

2. Place the patient supine. Ventilate the patient.
3. Check, assemble, and prepare the equipment **Step 2**.
4. Approach the patient from the left side of the neck. With the patient's head in a neutral position, palpate for and locate the cricothyroid membrane **Step 3**.

Figure 6-37 **A.** Nu-Trake kit. **B.** Pertrach kit.

5. Cleanse the anterior part of the neck from the laryngeal prominence to just below the cricoid ring and position a fenestrated drape **Step 4**.
6. Palpate the cricothyroid membrane.
7. Stabilize the thyroid cartilage with your nondominant hand.
8. With a scalpel in your dominant hand, make a 1-to 2-cm vertical skin incision over the cricothyroid membrane **Step 5**.
9. Puncture the cricothyroid membrane and make a horizontal incision 1 cm in each direction from the midline (a shallow incision, so that the posterior tracheal wall is not injured) **Step 6**.
10. Place a curved hemostat through the incision *before* removing the scalpel. Open the hemostats, ensuring that the tips are well within the trachea **Step 7**.
11. Remove the scalpel.
12. With your free hand, insert the ET tube between the tips of the open hemostats, advancing the balloon about 1.0 to 1.5 cm below the lower margin of the incision **Step 8**.
13. Remove the hemostats.
14. Inflate the cuff on the tube with 8 to 10 mL of air and remove the syringe **Step 9**.
15. Attach an ETCO2 detector in between the tube and the bag-mask device **Step 10**.
16. Ventilate the patient and verify tube position by auscultating the chest bilaterally and over the epigastrium **Step 11**.
17. Secure the tube to the neck and reconfirm correct tube placement. Continue to ventilate **Step 12**.

Rapid Sequence Intubation

Rapid sequence intubation (RSI) involves the coadministration of both anesthetic agents and neuromuscular blocking agents to produce a state of unconsciousness and paralysis to allow tracheal intubation. Originally developed in 1946 to facilitate airway management in obstetric patients requiring intubation with a presumed full stomach, it was further refined in the 1960s with the development of the Sellick maneuver (cricoid pressure) to further reduce the risk of aspiration in patients requiring emergent intubation (although this has never been proven). The use of RSI, compared with blind nasotracheal intubation, facilitated intubation using sedation and topical anesthesia or just forcing the tube into place, has proven to have increased success rates and decreased associated complications. It has become the procedure of choice in the emergency department and trauma bay. Its use in the prehospital setting is relatively new. Initially limited to air medical providers, RSI is now being used by ground providers and is not without controversy.

The indications for performing RSI include many of the same indications for ET intubation with the exception of the patient in cardiac arrest who should not require the administration of medications to facilitate intubation. Remember, by administering sedatives and paralytics, you are taking away a patient's ability to protect his or her airway and breathe. You must be able to provide an airway and adequately ventilate and oxygenate your patient. RSI also assumes that patients have eaten prior to needing emergent intubation. By administering medications to sedate and paralyze a patient, the risk of regurgitation and aspiration is minimized, but not eliminated.

Skill Drill 6-9

Performing Surgical Cricothyrotomy

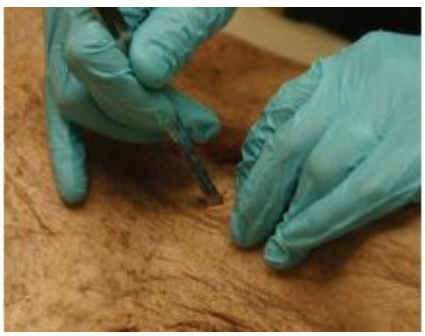
1 Use BSI precautions (gloves and face shield).

2 Check, assemble, and prepare the equipment.

3 Approach the patient from the left side of the neck. With the patient's head in a neutral position, palpate for and locate the cricothyroid membrane.

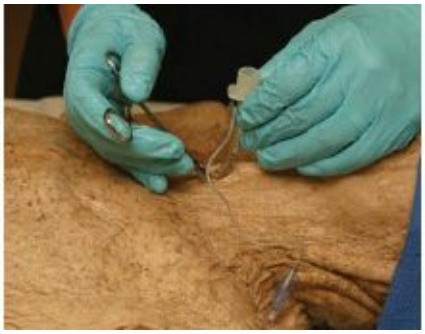
4 Cleanse the anterior part of the neck from the laryngeal prominence to just below the cricoid ring and position a fenestrated drape.

5 With a scalpel in your dominant hand, make a 1- to 2-cm vertical skin incision over the cricothyroid membrane.

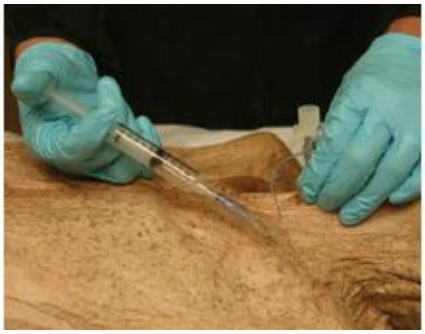


6 Puncture the cricothyroid membrane and make a horizontal incision 1 cm in each direction from the midline (a shallow incision, so that the posterior tracheal wall is not injured).

7 Place a curved hemostat through the incision before removing the scalpel. Open the hemostats, ensuring that the tips are well within the trachea.

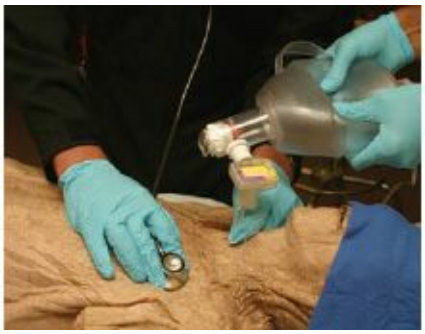


8 With your free hand, insert the ET tube between the tips of the open hemostats, advancing the balloon about 1.0 to 1.5 cm below the lower margin of the incision.



9 Inflate the cuff on the tube with 8 to 10 mL of air and remove the syringe.

10 Attach an $ETCO_2$ detector in between the tube and the bag-mask device.



11 Ventilate the patient and auscultate the chest bilaterally and over the epigastrium.

12 Secure the tube to the neck and reconfirm correct tube placement. Continue to ventilate.

The normal contraindications and complications for standard intubation apply for RSI. The most important contraindication is fear of the inability to intubate, and the most devastating complication is not being able to ventilate. This scenario will almost invariably result in a catastrophic patient outcome. The use of RSI requires careful patient selection (which can sometimes be difficult in the emergency and pre-hospital environment) and preparation.

Transport considerations for the patient undergoing RSI as well as any intubated patient are that capnography, preferably continous waveform, should be available, considered mandatory, and used throughout all phases of transportation of the patient.

Equipment for performing RSI includes:

- Gloves, gowns, mask, goggles
- Neuromuscular blocking agents
- Sedative agents
- Cardiac agents
- Syringes
- Needles
- Intubation equipment
- Suction
- Oxygen

RSI is a series of steps to facilitate ET intubation. It begins with preparation. All equipment necessary for intubation, rescue ventilation, and surgical airway must be available, checked, and working prior to use. The patient also requires at least one working IV access (preferably two because one may infiltrate, fall out, or be pulled out inadvertently) for the administration of the appropriate medications.

Special Populations

Children, obese patients, and pregnant patients desaturate much more rapidly.

The next step is preoxygenation. All patients undergoing RSI should be preoxygenated prior to beginning the procedure. In the patient who is spontaneously breathing with adequate tidal volumes, the application of high-flow oxygen via a nonrebreathing mask is sufficient. In the patient with apnea or hypoventilation, assistance with high-flow oxygen via a bag-mask device may be necessary. Bagging with RSI should be used only when indicated in order to avoid gastric insufflation and possible regurgitation.

Preoxygenation with high-flow oxygen saturates the air remaining in the lungs after normal expiration (functional residual capacity, approximately 30 mL/kg) with 100% oxygen. Many RSI protocols recommend administering high-flow oxygen for at least 5 minutes, if possible. This will allow for a prolonged period of apnea without significant desaturation in most patients. Children, obese patients, and pregnant patients desaturate much more rapidly.

The medications used in RSI fall under two broad categories: sedative or induction agents to induce unconsciousness and neuromuscular blocking agents to induce paralysis. The combination of both types of medications is essential to successful RSI and intubation. For both types of medications, the ideal agent would have a rapid onset, a short duration of action, a stable hemodynamic profile, and few if any adverse side effects. Unfortunately, agents that meet all of these requirements do not exist. Also, the use of these agents individually may result in adverse outcomes. The use of sedative/induction agents alone frequently does not result in successful intubation secondary to intact airway reflexes. The use of neuromuscular blocking agents alone may result in a paralyzed yet awake patient. The combination of both sedation/induction and neuromuscular blockade results in significantly greater intubation success with fewer complications.

Skill Drill 6-10 shows the steps for performing RSI, which are described as follows:

1. Prepare and assemble the equipment.
2. Preoxygenate with 100% oxygen **Step 1**.
3. Administer medications to premedicate, sedate, and paralyze the patient. When premedicating:
   - **a.** If your medical director or protocol recommends it, consider a defasciculating dose of a nondepolarizing neuromuscular blocking agent.
   - **b.** Consider lidocaine or opiates to decrease cardiovascular effects and elevations of intracranial pressure associated with upper airway stimulation.
   - **c.** Consider atropine to decrease bradycardia associated with the administration of succinylcholine.
4. When sedating the patient, use an agent that induces sedation and amnesia provided the patient is hemodynamically stable (systolic blood pressure >90 mm Hg).
5. Finally, paralyze the patient using an appropriate agent.
6. Apply posterior cricoid pressure **Step 2**.
7. Intubate the patient **Step 3**.
8. Confirm ET tube placement **Step 4**.
9. Release cricoid pressure.

10. Maintain paralysis and sedation.

Pharmacologic Agents Used in RSI

Sedative/Induction Agents

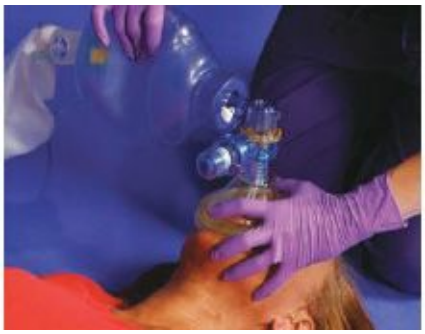
Table 6-4 lists sedative/induction agents that may be used in RSI, and their doses, onsets, durations, advantages, and disadvantages.

The induction agent chosen for use in RSI must be appropriate for the individual patient. The ultra-short acting barbiturates sodium thiopental and methohexital have a rapid onset of action and a short duration of action, which makes them ideal for use as induction agents, but requires a hemodynamically stable patient because use of these agents may precipitate myocardial depression and hypotension. The adverse hemodynamic profiles of these agents and the emergence of other, more hemodyamically stable agents has led to less use of sodium thiopental and methohexital in areas other than anesthesia. The benzodiazepines midazolam, lorazepam, and diazepam have a slightly longer onset of action and a significantly longer duration of action, which makes them less attractive as induction agents. They also have the ability to cause hypotension secondary to vasodilation. However, they are frequently used for sedation after intubation in the hemodynamically stable patient. Ketamine is an agent that has a stable hemodynamic profile and bronchodilatory properties that make it an attractive induction agent. The use of this agent, however, can increase intracranial pressure (ICP) and has been associated with adverse reactions. Propofol is an agent widely used by anesthesiologists. Its use as an induction agent in situations other than during surgery has been limited by associated vasodilation and hypotension. Many prehospital care providers have begun to use etomidate as the induction agent of choice because of its rapid onset of action, short duration of action, and stable hemodynamic profile.

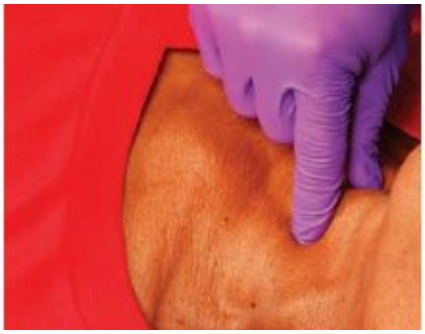
Other potentially beneficial agents used in RSI are lidocaine and atropine. Lidocaine can be administered at 1.5 mg/kg 2 to 3 minutes prior to intubation. Lidocaine has been shown to have mixed results regarding its ability to blunt the sympathetic response to intubation but it has been shown to blunt the rise in ICP associated with suctioning and laryngeal stimulation; no studies have been performed regarding intubation. Atropine should be administered in select patients receiving succinylcholine. Heightened vagal stimulation in pediatric patients can result in significant bradycardia. Many professionals recommend administering atropine to all children younger than 1 year who will be receiving succinylcholine and to consider adminstration for children between 1 and 10 years old. Also, adult patients receiving a second dose of succinylcholine should receive a dose of atropine prior to receiving additional succinylcholine. Adult patients with a heart rate of less than 60 beats/min should receive atropine prior to receiving succinylcholine and performing laryngoscopy.

Skill Drill 6-10

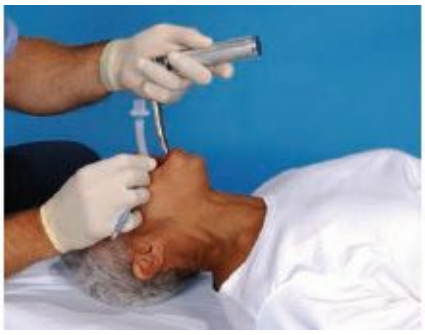
Performing Rapid Sequence Intubation



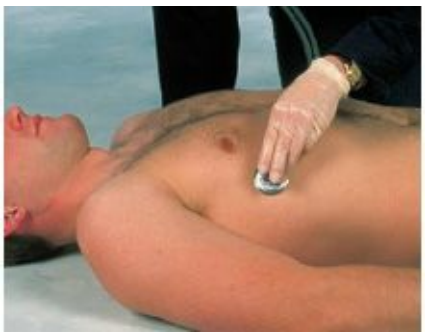
1 Prepare and assemble the equipment. Preoxygenate the patient. Administer medications to premedicate, sedate, and paralyze the patient.



2 Apply posterior cricoid pressure.



3 Intubate the patient.



4 Confirm ET tube placement. Release cricoid pressure.

Special Populations

Heightened vagal stimulation in pediatric patients can result in significant bradycardia. It is a good idea to administer atropine to children younger than 1 year who will be receiving succinylcholine and to

have it available for children between 1 and 10 years old.

The dose of atropine for pediatric patients is 0.02 mg/kg IV. The minimum dose is 0.1 mg. The maximum dose is 0.5 to 1.0 mg total. The adult dose of atropine is 0.5 to 1.0 mg IV. Atropine should be administered approximately 1 to 2 minutes before administering succinylcholine and attempting intubation.

Neuromuscular Blocking Agents

There are two types of neuromuscular blocking agents, depolarizing and nondepolarizing. The depolarizing agents act by rapidly depolarizing the neuromuscular end plate causing resistance to further stimulation. The nondepolarizing agents block the binding of acetylcholine to its receptors in the neuromuscular end plate. Both result in paralysis. The only depolarizing neuromuscular blocking agent in use in the United States is succinylcholine. Succinylcholine has several important absolute contraindications for its use. It should not be used in patients who have burns older than 48 hours, crush injuries, stroke, or spinal cord injuries less than 6 months previously, or degenerative muscle diseases (ie, ALS, muscular dystrophy).

There are several nondepolarizing neuromuscular blocking agents utilized in RSI. They are vecuronium, pancuronium, rocuronium, and cisatracurium. These agents are primarily used in three ways as a part of the RSI sequence.

- They can be used as pretreatment agents to prevent the fasciculations encountered with succinylcholine. The defasciculating dose range is 10% to 20% of the intubating dose. The use of defasciculating doses is rare in the prehospital setting. For vecuronium and pancuronium, the range is 0.01 to 0.02 mg/kg; and for rocuronium, the dose is 0.06 mg/kg.

TABLE 6-4 Sedative/Induction Agents

Drug Category	Drug Name	Dose	Onset	Duration	Advantages	Disadvantages
Ultra-short acting barbiturates	Sodium thiopental (Penothal)	3-6 mg/kg	<30 s	5-10 min	Significant anticonvulsant properties, cerebro-protective, can reduce ICP.	Histamine release. A potent vasodilator and myocardial depressant.
	Methohexital (Brevital)	1-3 mg/kg	<30 s	5-10 min		
Benzodiazepines	Midazolam (Versed)	2-5 mg on induction, 5 mg PRN for sedation	30-60 s	15-30 min	Provide amnesia, hypnosis, sedation. Anticonvulsant properties.	Vasodilation, hypotension, myocardial depression.
	Lorazepam (Ativan)	0.03 to 0.06 mg/kg	1-2 min	1-2 hr		
	Diazepam (Valium)	0.3 to 0.6 mg/kg	45-60 s	15-30 min		
Anesthetic agents	Etomidate (Amidate)	0.2-0.6 mg/kg	15-45 s	3-12 min	Usual induction dose is 0.3 mg/kg. Preferred induction agent for RSI secondary to its rapid onset, short duration, and stable hemodynamic profile. Cerebroprotective, reducing cerebral blood flow and metabolism in the setting of elevated ICP.	Injection site pain secondary to the diluent (propylene glycol). Myoclonic movements that may mimic seizure activity. Not FDA approved for use in pediatric patients, although many published reports in pediatric patients report no adverse effects. Plasma cortisol suppression has also been reported with etomidate use in the ICU environment, long term. Short-term use in RSI has not shown cortisol suppression.
	Ketamine (Ketalar)	1-2 mg/kg	45-60 s	10-20 min	Phencyclidine derivative with analgesic, anesthetic, and amnestic properties. Also has a stable hemodynamic profile secondary to catecholamine release and stimulation of the sympathetic nervous system. Also a bronchodilator, making it an ideal induction agent in the setting of bronchospasm.	In the setting of elevated ICP, the use of ketamine may increase ICP further. Emergence reactions are also an occasional complication of ketamine administration. Reactions are rarely an issue when ketamine is used in the setting of RSI because the benzodiazepine used for post RSI sedation attenuates this response.
	Propofol (Diprivan)	1-2 mg/kg	15-45 s	5-10 min	Decreases cerebral oxygen demand and ICP.	Vasodilation and myocardial depression, that may result in hypotension and a subsequent reduction in CPP. Pain at injection site.
Opiates	Fentanyl (Sublimaze)	2-3 µg/kg	3-5 min	30-60 min	Attenuation of the sympathetic response to laryngoscopy.	Hypotension and apnea.

Abbreviations: CPP, cerebral perfusion pressure; FDA, Food and Drug Administration; ICP, intracranial pressure; ICU, intensive care unit; PRN, pro re nata (as needed); RSI, rapid sequence intubation.

- They can be used as the primary paralytic in the event of contraindications to succinylcholine (hyperkalemia).
- Most commonly, they are used to maintain postintubation paralysis.

Neuromuscular blocking agents are summarized in **Table 6-5**.

Tracheostomy Management

Patients receiving interfacility transport may already have a tracheostomy in place for a variety of reasons as follows:

- Facial trauma

- Significant tracheal trauma
- Head injury
- Most commonly, failure to wean/long-term ventilator support

Contraindications to a tracheostomy include coagulopathy, neck tumor, and infection; however, these are relative contraindications, because the alternative is death.

Complications of a tracheostomy include:

- Accidental removal, particularly a "fresh" or nonmature tracheostomy
- Infection
- Hemorrhage Aspiration
- Mediastinal emphysema
- Tracheoesophageal fistula
- Tracheal stenosis
- Tracheomalacia
- Tracheoarterial fistula (frequently from a "low-lying" tracheostomy that erodes into the innominate artery)

Tracheostomy placement does not occur in the field and is therefore usually not in the skill set of the pre-hospital provider. It takes longer to perform than other surgical airways and requires equipment not usually found in the prehospital environment. In general, patients with long-term tracheostomy devices can easily remove and replace them. Endotracheal tubes can be placed in a tracheotomy stoma if a device cannot be replaced or is malfunctioning. In a patient who needs positive-pressure ventilation, the cuff on a tracheostomy device should be inflated or replaced with a cuffed endotracheal tube if ventilation is inadequate.

Mechanical Ventilation

Mechanical ventilation refers to the application of a device that provides patients varying degrees of ventilatory support. These devices range from simple to complex, depending on the needs of the patient and the capabilities of the machine. At one time, the classification and description of ventilators were simple; however, with the increase in biotechnology, the classification of these devices has become more complicated. All ventilators, however, have several common characteristics.

1. Power source: All ventilators require an external power source. The source can be electric or pneumatic (requiring a 50-psi gas source).
2. Cycling: This refers to which variable terminates the inspiratory phase of a breath. Pressure, volume, time, and flow can terminate a breath, depending on the ventilator.
3. Breath delivery: The ventilator can deliver a breath using either negative pressure **Figure 6-38** or postive pressure **Figure 6-39.**
4. Parameters: **Mode,** tidal volume or inspiratory pressure, respiratory rate, flow or **I time**, FIO_2, and PEEP are selected by the clinician. Modes and parameters are discussed in detail later in this section.

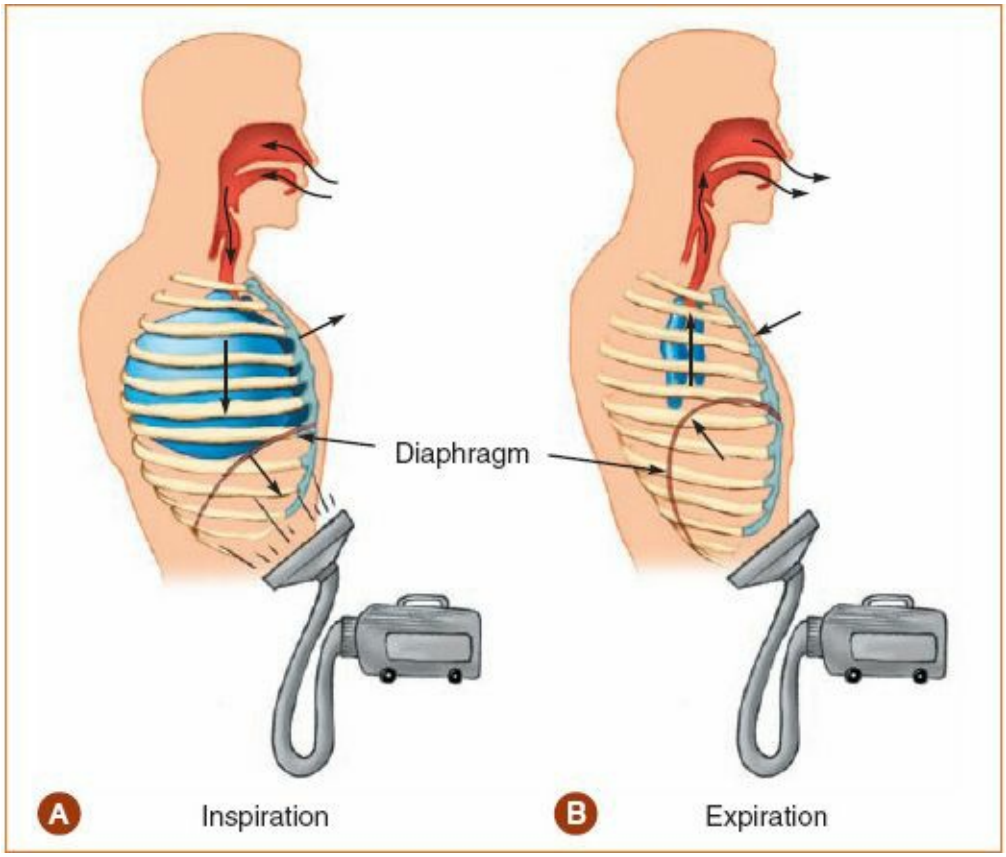


Figure 6-38 Normal ventilation is negative-pressure ventilation, which is similar to a vacuum cleaner. Negative pressure pulls down the diaphragm, causing the lungs to fill **(A).** When the pressure is released, the diaphragm relaxes and the lungs empty **(B).**

TABLE 6-5 Neuromuscular Blocking Agents

Drug Category	Drug Name	Dose	Onset	Duration	Advantages	Disadvantages
Depolarizing neuromuscular blocking agents	Succinylcholine (Anectine, Quelicin)	1-2 mg/kg	< 1 min	5 min	Rapid onset, short duration of action.	Fasciculations. Hyperkalemia. Increased intracrainal pressure, increased intraocular pressure, and increased intragastric pressure. It may cause or worsen bradycardia, particularly in pediatric patients. It has been implicated in malignant hyperthermia and may cause prolonged neuromuscular blockade.
Nondepolarizing neuromuscular blocking agents	Vecuronium (Norcuron)	For intubation: 0.15 mg/kg Postintubation paralysis: 0.01-0.1 mg/kg	90-120 sec	60-75 min	Longer duration means that effects are less likely to wear off during transport.	Possible prolonged paralysis with long-term use, renal or hepatic dysfunction. Possible prolonged paralysis with concurrent use of corticosteroids. No effect on level of consciousness, so they must be administered with adequate anesthesia, analgesia, or sedation. In patients with apnea, establish a patent airway prior to use.
	Rocuronium (Zemuron)	For intubation: 0.6 to 1.2 mg/kg Postintubation paralysis: 0.1-0.2 mg/kg	< 2 min	30-60 min		
	Pancuronium (Pavulon)	For intubation: 0.1 mg/kg Postintubation paralysis: 0.015-0.1 mg/kg	1-2 min	45-60 min		
	Cisatracurium (Nimbex)	For intubation: 0.15-0.2 mg/kg Postintubation paralysis: 0.03 mg/kg	2-3 min	30-40 min		

5. Ventilator circuit: An external circuit, which varies depending on the ventilator, used to connect the ventilator to the patient.
6. Alarms: Varying types of audio and/or visual alarms warn of ventilator malfunction or parameters outside set alarm limits. They should be set for each individual patient and *never* disabled.

Remember that not all ventilators have the same range of characteristics, capabilities, or features. All users need to be familiar with the capabilities of the devices, and no one should use any ventilator without being properly educated on its use and capabilities.

The indications for mechanical ventilation range from apnea, managing the work of breathing, and improving distribution of inhaled gases, to ventilatory and respiratory failure. Use of mechanical ventilation, while lifesaving, can have negative effects on several body systems:

1. Positive-pressure ventilation increases intrathoracic pressure, which can result in barotrauma (pneumothorax).
2. An increase in intrathoracic pressure can result in reduced venous return to the right side of the heart, which may result in poor cardiac output and hypotension.

3. Increased intrathoracic pressure may reduce blood flow to the liver and kidneys.
4. The diminished venous return to the right side of the heart, poor cardiac output, and possible hypotension along with reduced hepato/renal blood flow may fool the body into believing it is volume depleted, which results in various hormonal changes such as increased vasopressin and aldosterone activity and decreased levels of atrial natriuretic hormone. All of these changes result in fluid retention.

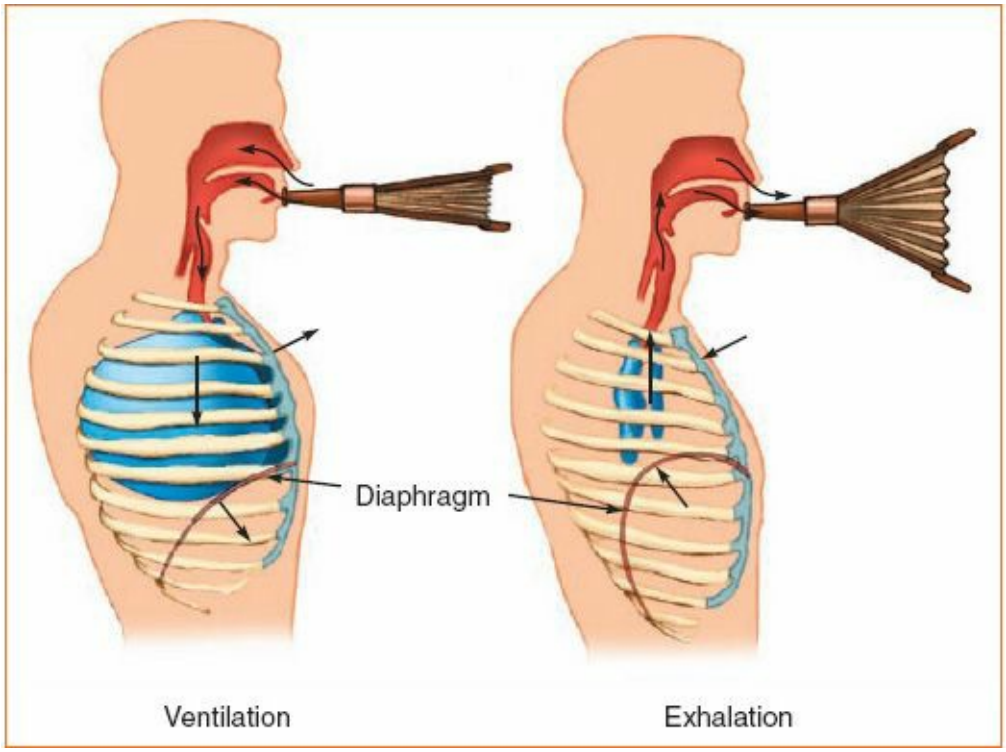


Figure 6-39 With positive-pressure ventilation, air is pushed into the respiratory tract with bag-mask ventilation. This is the opposite of negative-pressure ventilation.

Negative-Pressure Ventilators

During the polio epidemics of the 1950s, iron lungs were common **Figure 6-40**. Recall that in normal spontaneous breathing, a breath is initiated by a drop in the transrespiratory pressure gradient. **Negative-pressure ventilators** operate in the same way. A negative pressure is transmitted by the ventilator to the chest wall, therefore allowing inspiration to occur. The original iron lung is no longer used, but has been replaced by smaller and more convenient devices. The two most common devices are the cuirass, or "turtle shell," and the poncho type. The cuirass resembles a chest plate of armor and is strapped onto the chest. A hose is then connected to the ventilator that sucks on the chest during inspiration. The poncho requires a lightweight frame that is fitted onto the chest with a lightweight airtight jacket placed over the patient's frame. The ventilator hose is then attached to the jacket. Both require that a tight seal be maintained. These devices are not found in acute care facilities, but are used in rehabilitation and long-term care facilities as well as the home. The devices are used chiefly on patients with neuromuscular diseases who still have airway control.

Positive-Pressure Ventilators

Positive-pressure ventilators are more prevalent than negative-pressure ventilators. The tidal volume is delivered at pressures greater than ambient pressure and is therefore paradoxical to spontaneous breathing. There are potential hazards to this type of life support, however, such as pneumothorax, subcutaneous emphysema, and decreased cardiac output. Later on in the chapter, we will discuss

strategies that attempt to minimize these complications. Positive-pressure ventilators come in various sizes, power sources, and capabilities, and can be as small and portable as a laptop computer or as large as a desk. Each institution selects the type of ventilator based on its capabilities and patient needs, as well as cost and operation.

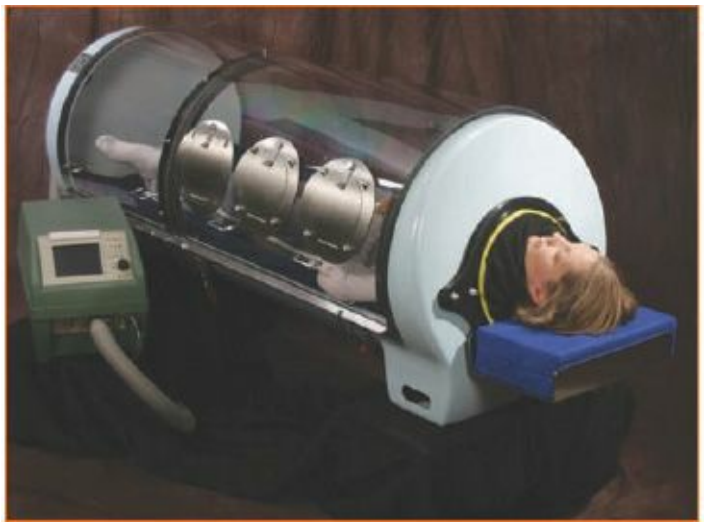


Figure 6-40 An iron lung.

Positive-pressure ventilators are commonly described by which variable terminates the inspiratory phase of the breath. **Pressure ventilators** end the delivery of the tidal volume based on a clinician-selected predetermined pressure. Therefore, the volume is variable. In **volume ventilators**, the breath ends when the predetermined tidal volume is achieved, and therefore the pressure is variable. **Flow-cycled ventilators** end inspiration when a predetermined **flow rate** is achieved, and **time-cycled ventilators** end inspiration after a selected inspiratory time has been achieved. Volume ventilators are the most common ventilator used. When you are using pressure ventilators, remember that the machine terminates inspiration when a preset pressure is achieved. Therefore, if the patient is biting down on the ET tube or is obstructed with mucus, the ventilator still delivers the pressure, but the tidal volume is significantly decreased. Increased airway resistance and decreased thoracic compliance also have the same effect. Careful monitoring of breath sounds, oxygen saturation, $ETCO_2$, visual inspection for adequate chest expansion, and monitoring delivered V_T are essential when pressure ventilation is being used.

Volume ventilators deliver a preset volume using varying pressures. The pressure required by the ventilator depends on the size of the tidal volume, flow rate, airway resistance, and lung–chest wall compliance. Because volume ventilators use varying pressures, it is important that you monitor the **peak airway pressure**. The high-pressure alarm alerts you that excessive pressures are being used to deliver the tidal volume. This alarm is set at approximately 10 cm H_2O greater than the patient's average peak pressure. Secretions, bronchospasm, and a decrease in thoracic/pulmonary compliance will result in increased airway pressures.

All ventilators, regardless of type, rely on an external circuit that connects the ventilator to the patient. Intrinsic to the circuit is some method for exhaled gas to be vented to the environment. On some ventilators, this is accomplished through the exhalation limb of the circuit that is connected to the ventilator. An internal exhalation valve then regulates exhalation as well as PEEP. Other ventilators use an external exhalation valve.

In either case, a closed system must exist between the ventilator and the patient. Any loose connections, cracks, or disconnections will result in ventilator malfunction and can be potentially life threatening. The integrity of patientventilator connection via the circuit should be visually inspected

throughout transport.

Invasive vs Noninvasive Ventilation

For years, conventional ventilation required **invasive ventilation** in the form of placement of an artificial airway, chiefly the ET tube. Because of the hazards and complications of ET tubes, other interfaces began to be used. Mouthpieces and masks of varying sizes and styles were attached to conventional ventilators, giving rise to "noninvasive" ventilation. By definition, **noninvasive ventilation** is any form of mechanical ventilation without an artificial airway. This now includes other devices such as CPAP and **bi-level positive airway pressure (BiPAP)**. The modalities use nasal or full-face masks to deliver a preset pressure to the lungs during spontaneous breathing. In CPAP, the prescribed pressure raises the breathing baseline above ambient pressure. The increased pressure across the entire breathing cycle increases the **mean airway pressure** and stents the airway. The increased mean pressure also increases the functional residual capacity (FRC) and decreases the effort that the patient has to exert to get a full breath, thus improving oxygenation.

BiPAP works similarly, but two separate pressures are used—inspiratory positive airway pressure (IPAP) and PEEP, also called expiratory positive airway pressure (EPAP). IPAP is always set higher than EPAP by at least 4 cm H_2O. BiPAP has the same benefits as CPAP. Additionally, the pressure gradient enhances ventilation. The reduced expiratory pressure makes exhalation easier and increases patient tolerance. Current EMS research indicates that CPAP use in the field is well tolerated and can significantly improve oxygenation and ventilation, potentially avoiding the need for intubation. Both are contra-indicated in patients who cannot protect their airways, have unstable facial fractures, or are uncooperative. Because they augment spontaneous breaths, neither is considered to be "life support" equipment.

Ventilator Modes and Parameters

Ventilator modes describe what types of breaths are delivered to the patient. This chiefly refers to whether the breath is a mandatory or machine breath or a spontaneous breath. The various modes of mechanical ventilation may ensure a particular minute ventilation and control the work of breathing. The result is an alphabet soup of acronyms that can be confusing to the novice ventilator user. Once again, not all ventilators have all the modes available. **Table 6-6** lists the most common modes of ventilation.

Ventilator parameters refer to the other settings that may be adjusted and should be monitored continuously by the clinician. Again, the availability of modes, alarms, and other parameters can vary from machine to machine. The clinician must be familiar with the ventilator being used. Ventilator parameters and alarms are listed in **Table 6-7**. This is not intended to be an all-inclusive list. Some ventilators have many more audio/visual alarms, and the CCTP must understand these prior to transport.

Note that expiratory time is not a set parameter. Expiratory time is a function of set respiratory rate and inspiratory time and is "left over" after inspiration is completed.

PEEP

PEEP, which is not a true mode, occurs at the end of a mandatory machine breath. Instead of exhaling back to ambient pressure, the ventilator is set to stop exhalation at a prescribed pressure. The result is an increase in the amount of air in the alveoli. This increases the FRC and improves oxygenation. PEEP also reduces the shunt fraction and increases lung compliance. Detrimental effects of PEEP include decreased venous return, a resultant decrease in cardiac output, increased ICP, decreased renal and portal blood flow, and increased risk of barotrauma. PEEP is contraindicated in patients with untreated pneumothorax

or bronchopleural fistula. PEEP values are usually determined by the ability to improve the Pa_{O_2} and decrease the F_{IO_2}. An important consideration during transport is to maintain PEEP levels even when using a manual resuscitator (bag-mask device). An external PEEP valve should be attached to the bag. The patient's hemodynamic status, as well as oxygenation, should be carefully monitored when applying and adjusting PEEP.

Special Populations

Pregnant patients at term increase their tidal volume by about 40% and they live in a compensated respiratory alkalosis. Keep this in mind when adjusting ventilator settings.

Nitric Oxide

Nitric oxide (NO, or nitrogen monoxide) has seen and will continue to see increased use during critical care transports. This increase is due in part to the introduction of portable NO administration units and expansion of the patient populations in which NO appears to be effective. Currently in the United States, the only Food and Drug Administration-approved use of NO is for treatment of refractory hypoxemic respiratory failure in term and near term newborns. In neonatal applications when NO is used, the response rate to NO exceeds 60%. Total costs of care are reduced, and the need for extracorporeal membrane oxygenation (ECMO) therapy has been reduced by up to 20%.

TABLE 6-6 Modes of Mechanical Ventilation

Mode	Definition	Indication
Control, or continuous, manual ventilation	Ventilator delivers a specific set respiratory rate and volume, controlled by the ventilator. The patient is not allowed any other breaths.	Used when a specific minute volume is required, such as with a head injury. Can also be used when a patient is fighting or "bucking" the ventilator. The respiratory center is controlled by using neuromuscular blockade and sedation.
Assist/control	The ventilator guarantees a minimum breath rate. The patient is allowed to breathe over the set rate, but each breath is at the preset tidal volume. Necessitates the proper setting of the **sensitivity** (ventilator control that regulates the amount of negative pressure required by the patient to initiate or "trigger" a breath).	Controls the work of breathing, but allows the patient to set his or her own respiratory rate.
Synchronized intermittent mandatory	A set respiratory rate and tidal volume are delivered and synchronized with each patient-initiated breath. Each patient breath is at the patient-initiated volume. The machine will deliver a certain	Allows the patient to assume some or most of the work of breathing, depending upon the mandatory

ventilation	number of mandatory breaths. All other breaths are spontaneous breaths at the patient's own rate and depth.	rate. Can also be used as a weaning technique.
PCV	The machine is set to deliver a preset pressure rather than volume. PIP is set and flow is delivered until the set PIP is reached. Tidal volume may vary significantly with changes in patient pulmonary status.	Used when volume ventilation requires too much pressure, such as in acute respiratory distress syndrome. Commonly used in conjunction with inverse ratio ventilation (see below). One of the lung protective strategies.
Pressure-regulated volume control ventilation	The machine will deliver the desired tidal volume at the least amount of pressure.	Used when both pressure and volume regulation are needed.
Inverse ratio ventilation	The ventilator is set so that the inspiratory phase is longer than the expiratory phase.	Used to increase oxygenation when the F_{IO_2} is too high (> 0.60). Must ensure that all volume is exhaled to prevent breath stacking and hyperinflation. Often used with PCV.
Pressure support	A clinician-selected amount of positive pressure augments a spontaneous breath.	Used to augment a spontaneous breath. The pressure overcomes the superimposed resistance of the airway and ventilator circuit and improves lung compliance.
CPAP	The baseline pressure for spontaneous breathing is raised above ambient, increasing oxygenation and mean airway pressure.	Improves oxygenation during spontaneous breathing.
Bi-level positive airway pressure	CPAP with the ability to sense inspiration and deliver additional positive pressure.	Improves oxygenation and ventilation with spontaneous breathing.
PEEP	The ventilator is set to stop exhalation at a prescribed pressure.	Measured in centimeters of water. Some practitioners use 5 cm of PEEP for every patient, a kind of "physiologic PEEP." PEEP is used to increase the functional residual capacity or the amount of air remaining in the lungs at the end of expiration. Increased PEEP helps keep the alveoli open and improves gas (oxygen) exchange. It is frequently titrated upward in response to hypoxemia in an effort to improve oxygenation and lower high F_{IO_2} levels.

Abbreviations: CPAP, continuous positive airway pressure; FIO_2, fraction of inspired oxygen; PCV, pressure control ventilation; PEEP, positive end-expiratory pressure; PIP, peak inspiratory pressure.

TABLE 6-7 Ventilator Parameters

Parameter	Definition	Setting Range
Tidal volume	Amount of air delivered with each machine breath	Approximately 10 mL/kg of ideal weight; 5–7 mL/kg if poor lung compliance (for example, from acute lung injury or restrictive or obstructive disease) is present. Smaller volumes may be used to prevent alveolar overdistention.
Respiratory rate	Number of breaths delivered in a minute	10–14 based on desired mandatory minute ventilation.
Fraction of inspired oxygen	Percentage of inhaled oxygen expressed as a decimal	0.21–1.0. Titrated to achieve adequate oxygenation (oxygen saturation, 0.90%). For initial management an FIO_2 of 1.0 (100%) is used and then titrated downward in an attempt to maintain adequate arterial saturation at the lowest FIO_2 possible (50% or less) to avoid oxygen toxicity.
Peak flow	Speed at which the tidal volume is delivered	Can vary greatly between 35 and 100 L/min.
Inspiratory time	The time set to deliver the desired flow or tidal volume	Can vary between 0.5 and 1.5 sec.
Peak inspiratory pressure	The peak pressure generated during ventilation. May vary with volume ventilation.	Set parameter with pressure ventilation. Should be maintained at the lowest value possible to minimize barotrauma/volutrauma (trauma from excessive lung inflation volumes).
Inspiratory plateau pressure	The pressure applied in volume ventilation to the small airways and alveoli. This is measured by applying an inspiratory pause.	Plateau pressure should be maintained at less than 30 cm H_2O to minimize volutrauma.
Low-pressure alarm	Alerts clinicians to a leak in the system or a patient disconnect	Set 5–10 cm H_2O lower than the average cycling (peak) pressure.
High-pressure alarm	Alerts clinician that the ventilator is using high pressures to deliver the tidal volume	Set 10 cm H_2O higher than the peak inspiratory pressure.
Power		

failure/low battery/low pressure source alarm	Alerts clinician to ventilator failure due to a loss of power	Varies from ventilator to ventilator.

Research is ongoing, and many institutions have standing agreements with their Institutional Review Boards to allow compassionate use of NO in adults with refractory hypoxemia from a wide range of clinical syndromes. Note that NO is not the same as nitrous oxide.

NO is a gas that increases blood flow by promoting vasodilatation. This mechanism occurs via the endothelial blood vessel linings, which receive the signal from NO for smooth muscle to relax. NO also discourages smooth muscle contraction of the blood vessels. Therefore, when hypoxia results from pulmonary vascular vasoconstriction, NO can significantly improve flow and oxygenation. However, an overabundance of NO can contribute to reperfusion injury, for example if an excess is produced after an ischemic injury.

NO is presently patented and available from a single manufacturer under a business model that charges for gas usage and controls administration devices, gas supply, and disposables used to deliver the gas to ventilator circuits. NO gas is bled into the inspiratory limb of a ventilator circuit at a dilution measured in parts per million. Published studies list effective doses at 0.25 to 80 ppm, although ranges vary by institutional policy and practice. Effects of NO vary and are patient specific.

Parameters for titration of NO need to be defined prior to initiating therapy. Patient assessment modalities vary considerably by institution. Typical measurements that suggest a favorable response to NO include a 20% improvement in oxygenation and/or a 30% decline in pulmonary vascular resistance. In the absence of invasive monitoring capability, improvements in oxygenation, hemodynamic stability, and less frequent desaturations have been used to gauge effectiveness (or lack thereof).

Abrupt discontinuation of NO has been associated with a rebound phenomenon with significant desaturations and hemodynamic instability. CCTPs should be certain to calculate gas volumes and battery lifespan and to ensure that needed equipment will be available to continue NO administration throughout a transport.

NO gas readily reacts with other gases, including oxygen and water vapor found in ventilator circuit tubing. Highly corrosive nitrogen dioxide (NO_2) results from this oxidative process and poses significant risk of acute lung tissue injury with resultant pulmonary edema. NO_2 concentrations are monitored by the commercially available administration device—elevated levels can be remedied by purging the NO gas system or lowering the NO bleed. Health care worker occupational exposure to NO and NO_2 is a theoretical risk of treatment but has yet to be demonstrated in published research.

Methemoglobinemia is another potential complication of NO therapy caused by oxidation of hemoglobin to methemoglobin, a dyshemoglobin that cannot carry oxygen. Monitoring methemoglobin levels at least daily is important to prevent inducing an iatrogenic hypoxia. Pulse carbon monoxide oximeters capable of measuring methemoglobin are helpful for spot assessment or continual monitoring of methemoglobin levels.

NO can be a valuable adjunct for critical care transport of extremely hypoxic patients, to optimize conventional therapies, to continue care started prior to transfer, or as a bridge to more advanced therapies such as ECMO or high-frequency jet ventilation. The greatest dangers during transport are iatrogenic overdoses of NO with resultant acute lung injury and pulmonary edema and/or methemoglobinemia. Abrupt discontinuation of NO therapy due to gas loss or equipment malfunction can result in catastrophic rebound effects. Use of NO during transport requires the CCTP be thoroughly

familiar with NO equipment operation, troubleshooting, and emergency procedures in case of failure.

Ventilator Management

There may be instances when you will use a portable ventilator during patient transport. Indications for using a portable ventilator include:

- Impending or actual respiratory failure
- Inadequate respiratory drive or apnea
- Inadequate gas exchange
- Decrease work of breathing and oxygen cost

Complications of using a portable ventilator include:

- Mechanical failure
- Patient anxiety
- Improper settings
- Increased intrapulmonary pressure
- Cardiovascular compromise
- Gastrointestinal disturbances
- Infection
- Impaired clearance and drying of secretions

Skill Drill 6-11 shows the steps for using a portable ventilator, which are described as follows:

1. Initiate power
2. Attach the ventilator circuit **Step 2**.
3. Adjust continuous flow **Step 3**.
4. Select the ventilator rate, inspiratory rate, flow rate, and oxygen concentrations **Step 4**.
5. Make mode selections: CPAP and PEEP, intermittent mandatory ventilation (IMV)/synchronized IMV (SIMV), assist control **Step 5**.

During transport of the critically ill patient receiving mechanical ventilation, only necessary adjustments to the ventilator should be made. These adjustments usually include maintaining adequate oxygenation and minute ventilation. If mechanical ventilation is instituted, it is recommended that the guidelines listed below be followed after securing a patent airway. In most cases, the CCTP will use the settings already in place. The guidelines should be used by the CCTP who is intubating and establishing the patient on the ventilator. Keep in mind that any occult pneumothorax or pneumome-diastinum may worsen with the initiation of positive-pressure ventilation.

1. Start with an F_{IO_2} of 1.0 and titrate down if possible to maintain an Sp_{O_2} of 92% to 94%.
2. To manage the work of breathing, use the assist/control mode. If the patient is paralyzed and sedated, there is NO difference between assist control (AC) and SIMV.
3. Tidal volumes can be set at approximately 7 to 10 mL/kg of ideal body weight.
4. Set the respiratory rate between 10 and 12 breaths/min.

5. Adjust the peak flow rate or inspiratory time to accommodate the patient's inspiratory flow demand and to allow for sufficient expiratory time and avoidance of **auto-PEEP**.
6. Adjust the sensitivity to –1 cm H_2O.

It is the responsibility of the transport team to ensure that ventilator function and the patient's ventilatory status are maintained during the transport. This can be accomplished by:

- Maintaining a stable and patent airway.
- Verifying and documenting the ventilator settings before, during, and after arrival.
- Ensuring proper power supply and oxygen during the transport.
- Assessing and documenting breath sounds before, during (if possible), and after transport.
- Continuously monitoring pulse oximetry (SpO_2) and $ETCO_2$.
- Having a manual resuscitator (bag-mask device) available if there is any doubt about the functioning of the ventilator.

Skill Drill 6-11

Using a Portable Ventilator

1 Initiate power.

2 Attach ventilator circuit.

3 Adjust continuous flow.

4 Select the ventilator rate, inspiratory rate, flow rate, and oxygen concentrations.

5 Make mode selections: CPAP and PEEP, IMV/SIMV, assist control.

Flight Considerations

When you are using a portable ventilator in the air medical setting, pneumatic-controlled ventilators are not accurate in-flight—electronic-controlled ventilators should be used for air transport. Watch for signs/symptoms of pneumothorax. In interfacility patients with significant respiratory compromise, a trial of 15 to 30 minutes on the transport ventilator prior to departure may be warranted to ensure that the ventilator changes will be tolerated.

Troubleshooting

Troubleshooting ventilator function is not as difficult as it seems. First, it is imperative that a manual resuscitator accompanies every patient on a ventilator. When in doubt of ventilator function, the

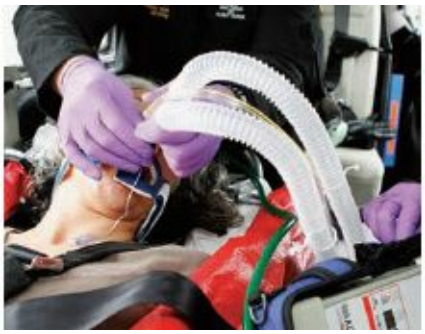
resuscitator must be used to ensure the safety of the patient. A good rule of thumb in troubleshooting equipment is to start at the patient and work your way, methodically, toward the equipment/wall.

Although alarms vary from ventilator to ventilator, the more common alarms are:

1. **Low battery/power source**: This alarm sounds when the electrical supply to the ventilator is inadequate or the gas inlet pressure is low. It is corrected by restoring the proper power source.
2. **Low-pressure alarm**: Usually for a leak or disconnect, this alarm may also sound if the cuff on the ET tube or tracheostomy tube is leaking or deflated, resulting in a significant air leak.
3. **High-pressure alarm**: The high-pressure alarm sounds when the ventilator uses too much pressure to deliver the tidal volume. It can be caused by many factors, such as bronchospasm, secretions, kinks in the external circuit or airway, biting on the ET tube, coughing, gagging, breathing asynchronously or "bucking" the ventilator, alveolar overdistention, and improper flow rates and/or I:E ratio. Migration of the ET tube to the esophagus and pneumothorax is the more serious cause. Assess the patient carefully and implement the appropriate intervention.

Skill Drill 6-12

Troubleshooting a Ventilator Low-Pressure Alarm



1 Quickly inspect the ventilator-patient interface for a disconnection. Tighten all connections.

2 If the alarm is still sounding, disconnect the ventilator and bag the patient.

3 Place a gloved finger over the patient ventilator connector and observe the next ventilator breath cycle.

If the manometer attempts to rise and the high-pressure alarm sounds, the ventilator circuit is intact and the leak is with the patient.

Skill Drill 6-12 If the low-pressure alarm sounds, follow the steps as described here:

1. Quickly inspect the ventilator-patient interface for a disconnection. Tighten all connections **Step 1**.
2. If the alarm is still sounding, disconnect the ventilator and bag the patient **Step 2**.
3. Place a gloved finger over the patient ventilator connector and observe the next ventilator breath cycle **Step 3**.
4. If the manometer attempts to rise and the high-pressure alarm sounds, the ventilator circuit is intact and the leak is with the patient.

Summary

Assessing the respiratory system of a critical care transport patient involves a thorough physical assessment as well as a review of the patient history and patient care chart. A thorough report by transferring personnel will help establish the clinical picture.

Establishing a baseline assessment and the ability to differentiate normal and abnormal findings in the respiratory system are paramount to comprehensive respiratory care during transport. Essential skills include auscultating breath sounds, evaluating oxygenation and ventilatory adequacy, and setting up and troubleshooting the ventilator.

Concepts such as tidal volume, minute volume, and anatomic dead space are crucial to understanding respiratory function. Additional devices and laboratory studies are also helpful in monitoring the patient's condition. ABGs, if available, reveal the acid-base balance and blood levels of oxygen and CO_2. Pulse oximetry, capnometry, and ETCO2 detection offer a continuous, noninvasive means of measuring these gases during transport.

Transport of the critically ill patient who is receiving ventilation must be planned to ensure patient safety. First, be familiar with the capabilities of the ventilator but always have a manual resuscitator on hand. Prepare for the transport by having an adequate power supply. Second, know why the patient is being vented and what the settings are prior to transport. Obtain a baseline assessment parameter for the patient and the ventilator before beginning the transport. Continue to monitor ventilator performance and patient response during the transport. Last, ensure the same upon arrival. Verify and document airway patency, respiratory assessment, and parameters at the time of transfer. By observing these guidelines, the transport should be a safe one for you and your patient.

Case Study

YOUR TEAM IS DISPATCHED TO A RURAL HOSPITAL 75 MILES AWAY for a 23-year-old, 70-kg woman in status asthmaticus. She was brought to the emergency department 2 hours ago by local EMS for difficulty breathing. She presented to the emergency department in acute respiratory distress, with audible

inspiratory and expiratory wheezes, intercostal and supraclavicular retractions, and tripod positioning. Her initial vital signs included the following: respiratory rate, 32 breaths/min and labored; heart rate, 126 beats/min; blood pressure, 126/84 mm Hg; and Spo_2, 92% on an 8-L nebulizer.

Brush fires in the area have placed the county under an air quality warning. The patient's attack occurred after she had taken her dog for a walk. The patient's medical history includes asthma, which is controlled with daily use of an Advair inhaler. She has been hospitalized three times prior to this episode. One admission resulted in an intensive care stay for respiratory failure requiring intubation.

A radiograph of the patient's lungs taken upon arrival at the emergency department demonstrated hyperinflation. The initial ABG reading while the patient was receiving a nebulizer treatment using 8 L of oxygen was pH 7.52, $PaCO_2$ 27 mm Hg, and PaO_2 88 mm Hg. A subsequent ABG reading 45 minutes later with the patient on a non-rebreathing mask at 15 L/min was pH 7.46, $PaCO_2$ 33 mm Hg, and PaO_2 79 mm Hg. All other chemistry and blood values were within normal limits.

You and your team arrive at the emergency department 4 hours after the patient's arrival. While your partner obtains her paperwork and speaks with the nurse caring for her, you assess the patient. You observe a young woman who is minimally responsive to verbal stimuli. She exhibits poor air exchange by shallow respirations accompanied by severe intercostal and supraclavicular retractions and minimal air exchange upon auscultation. Current vital signs include the following: pulse rate, 87 beats/min; respiratory rate, 18 breaths/min, labored and shallow; blood pressure, 112/78 mm Hg; Spo_2, 86% on a nonrebreathing mask with oxygen administered at 15 L/min. As you are completing your assessment of the patient, the nurse brings you the most recent ABG results drawn with the patient on the nonrebreathing mask showing pH 7.25, PCO_2 62 mm Hg, and PaO_2 65 mm Hg.

1. Does this patient meet the criteria for intubation?
2. Which factors should be considered when assessing the patient for a difficult airway?
3. Which medications are used during rapid sequence intubation?

Per protocol, you and your partner prepare for rapid sequence intubation. Using the LEMON rule, you determine that the patient should not be a difficult intubation. While your partner provides oxygen to the patient using a bag-mask device attached to 100% oxygen, you prepare the following medications for administration: lidocaine, 70 mg; etomidate, 21 mg for sedation; vecuronium, 0.7 mg as a defasciculating agent prior to paralyzing; and succinylcholine, 140 mg for paralysis. Your partner administers the lidocaine, etomidate, and succinylcholine while you are ventilating the patient and waiting for muscle relaxation. After 90 seconds you notice that the patient still doesn't feel completely relaxed and presents with an intact gag reflex. You follow your protocol and administer a second dose of succinylcholine through another IV line. You are able to intubate the patient without difficulty and confirm tube placement by auscultation, visualization, and use of capnography. The patient is placed on the transport ventilator and prepared for the trip back to your facility. Prior to leaving the hospital you administer an additional 7 mg of vecuronium and 3.5 mg of lorazepam (Ativan) for continued paralysis and sedation during transport. Additional treatment includes a continuous albuterol nebulizer at 10 mg/h, 125 mg of Solu-Medrol IV, and a normal saline drip at 150 mL/h.

Once you have the patient loaded in the back of your helicopter you notice that the patient's end-tidal CO_2 is rising and her oxygen saturation is dropping. Reassessment of the endotracheal tube indicates that the tube has become dislodged and is now ventilating the stomach. You and partner decide to pull the tube and reattempt intubation. After two unsuccessful attempts, you give your partner a chance; your partner is unable to visualize the vocal cords. Your King Airway is retrieved and inserted without difficulty. After confirming placement of the King Airway, you secure the tube with a commercial tube holder, place your

patient back on the ventilator, and continue your transport. You arrive at the receiving hospital approximately 90 minutes later following an uncomplicated trip. A detailed patient report is given to the awaiting staff upon your arrival, which includes a list of all the medications given along with the reason for the insertion of the King Airway.

4. Was it an acceptable practice to administer a second dose of succinylcholine and what are some possible causes for having to repeat the dose?
5. What is a King Airway and why was it able to be inserted without difficulty when the ET tube was not?

Analysis

This patient is a candidate for ET intubation due to her decreased mental status and respiratory failure, as shown by ineffective respirations, minimal air movement, and ABG results. Initially, the patient arrived at the emergency department awake and alert in respiratory distress. The first ABG results showed respiratory alkalosis, a common finding in early stages of an acute asthma attack. The second blood gas analysis came back with close to normal values with the exception of oxygenation. This finding should be a warning sign and should prompt you to reassess the patient to determine whether the condition is improving or the patient is getting tired and bears close watching. Don't let the numbers trick you!

Patient history and assessment play an important role in the preparation of intubation. Questions that can be answered by a review of patient history include the presence of congenital anomalies, recent surgery, trauma, infection, or cancer. Pertinent assessment findings include patients with short necks or those who are obese and patients with dental abnormalities such as an overbite. There are two assessment tools that can be used to guide your decision: the Mallampati method and the 3-3-2 rule.

The Mallampati method ranks the difficulty of intubation (class I is the easiest and class IV is the most difficult) based on how much of the oropharyngeal structures can be visualized in a patient who is seated in an upright position with a fully open mouth. A class I airway is one in which all oropharyngeal structures can be visualized. With a class II airway, the glottis is only partially visible. With a class III airway, the glottis cannot be exposed, but the corniculate cartilages can be seen. Finally, the class IV airway prevents the visualization of any oropharyngeal structures.

The 3-3-2 rule refers to three easy anatomic methods. For the first "3," have the patient open his or her mouth as wide as possible. This opening should allow for a minimum of three finger widths (approximately 5 cm). An opening of less than three finger widths may be an indicator of a difficult airway. The second "3" is a measurement of the length of the mandible. This is measured from the tip of the chin to the hyoid bone. Again, an optimal measurement is one that is the width of at least three fingers. The final measurement, "2," refers to the distance from the hyoid bone to the thyroid notch. An ideal airway would have a distance of at least two finger widths.

Medications administered during rapid sequence intubation are given to premedicate, sedate, and paralyze the patient. Medications given to premedicate the patient include a smaller dose (also known as a defasciculating dose) of a nondepolarizing neuromuscular blocking agent, such as vecuronium (Norcuron); lidocaine, to minimize cardiovascular complications and prevent a rise in intracranial pressure during intubation; atropine, to decrease the bradycardic effects associated with succinylcholine; and a sedative, or induction agent (for example, etomidate [Amidate]) to provide sedation and amnesia.

Succinycholine can be repeated if needed using the same dosing regimen. The most likely reason that the succinylcholine didn't work is due to an infiltrated intravenous line. However, because succinylcholine is temperature sensitive, that particular vial of medication might have been exposed to extreme temperatures for a prolonged period of time, thus contributing to the ineffectiveness of the medication.

The King Airway is a supraglottic airway device that doesn't require the direct visualization of the vocal cords to be inserted. The device is actually inserted into the esophagus, after which a distal and proximal balloon is inflated and the patient's lungs are ventilated through fenestrations located between the two balloons. Even though this is a supraglottic device, the patient can be mechanically ventilated without difficulty.

Prep Kit

Ready for Review

- The goal of the CCTP is to ensure the adequacy of ventilation and oxygenation of the critically ill patient during transport. The CCTP must have a good understanding of respiratory function and assessment.
- An understanding of airway anatomy and physiology, including the structures of the nose, mouth, pharynx, larynx, and trachea, is essential to successful airway management.
- The airway anatomy of pediatric patients is different from an adult's. The small size of the cricothyroid membrane in children younger than 4 years may make needle and surgical cricothyrotomy difficult or impossible. Children also have significantly higher oxygen consumption rates than adults.
- The respiratory system is divided into the conducting zone and the respiratory zone, with the conducting zone further divided into the upper airway and lower airway. An adult lung contains approximately 300 million alveoli, each one in contact with a pulmonary capillary, and the interface is called the alveolar capillary (AC) membrane.
- The pulmonary circulatory system is a low-pressure system, and for gas exchange to occur, blood flow must be in contact with the alveoli.
- Gas exchange occurs as a result of pressure gradient changes across the AC membrane, and with diffusion, the molecules of oxygen move from the alveoli into the blood because there are fewer oxygen molecules in the blood, and the molecules of CO_2 diffuse from the blood into the alveoli because there are fewer CO_2 molecules in the alveoli.
- $\dot{V}/\dot{Q}$ mismatch factors, due to inadequate ventilation, perfusion, or both, can affect the diffusion of gases across the membrane. The mechanics and dynamics of breathing use the concepts of elastance, compliance, resistance, and pressure gradients.
- The total lung capacity of a normal male adult is between 5 and 6 L of gas, which can be further divided into different lung volumes and capacities.
- Diseases that result in difficulty moving air out of the lungs are classified as obstructive diseases, including asthma, COPD, cystic fibrosis, and bronchioectasis, and involve an increase in airway resistance.
- Diseases that result in difficulty moving air into the lungs are defined as restrictive diseases, are the result of loss of chest or lung compliance, either individually or together, and include occupational lung diseases (the pneumoconioses) and idiopathic pulmonary fibrosis, pneumonia, atelectasis, chest wall deformities and injuries, and all the neuromuscular diseases that affect breathing.
- Patient assessment includes visually inspecting the patient with a focus on the chest, using auscultation sites to check breath sounds, assessing respiratory patterns and breathing rate, and using palpation to check the status of the lungs, skin, subcutaneous tissues, and chest expansion. Prior to transport, the

CCTP needs to review the patient care report carefully, noting any medications, underlying disease, or trauma that might compromise ventilation.

- The functioning of the respiratory system is measured using ABG levels, in which blood is obtained from a superficial artery and then analyzed for pH, $Paco_2$ (partial pressure of CO_2 in arterial blood), Pao_2 (partial pressure of oxygen in arterial blood), Hco_3^- (concentration of bicarbonate ion), base excess, and Spo_2 (oxygen saturation of the hemoglobin molecule).
- Respiratory insufficiency is the inability of the respiratory system to keep up with the metabolic demands of the body. Respiratory insufficiency can lead to respiratory depression. Respiratory failure, either oxygenation failure or ventilatory failure, occurs when the respiratory system fails to meet the body's metabolic needs.
- Basic airway skills are the starting point in the initial assessment and treatment of the patient by the CCTP and include positioning the nontrauma victim in the recovery position, using the head tilt–chin lift or the jaw-thrust maneuver or using airway adjuncts such as the OPA or NPA.
- Both the OPA and the NPA are used along with manual airway maneuvers to provide a patent airway.
- Suctioning to remove debris (vomit and blood) and turning the patient to his or her side while using a large-bore suction device can restore airway patency and minimize the potential for aspiration.
- Patients should receive some form of supplemental oxygen based on provider assessment, regardless of documented Spo_2.
- Oxygen delivery may take many forms, including mouth-to-mouth ventilation, barrier device/resuscitation mask, and bag-mask ventilation.
- Definitive airway management is considered to be the placement of an ET tube or tracheostomy tube within the trachea.
- In anticipation of a difficult airway, several factors need to be considered, including patient history and patient assessment. Also, the 3-3-2 rule should be used.
- Advantages of using the LMA are ease of insertion and superior oxygenation and ventilation compared with bag-mask ventilation. Disadvantages for using the LMA are the risk of aspiration and difficulty with obtaining an adequate seal. Use the LMA if the patient is in a deep coma or cardiac or respiratory arrest, or if ET intubation is not possible. LMA is contraindicated for use on patients with a gag reflex or facial or esophageal trauma, or if an airway obstruction by a foreign body is suspected.
- The Combitube, a dual-lumen, dual-cuffed supraglottic airway, may be placed blindly, and once proper placement is verified, the patient must be kept sedated and paralyzed. Use the Combitube when ET intubation is not possible or if intubation as an attempted rescue airway has failed or the patient is in a deep coma or cardiac or respiratory arrest, or to reduce the risk of gastric distention. Combitube contraindications include patients with a gag reflex, an upper airway obstruction or suspected foreign body obstruction, facial or esophageal trauma, known esophageal disease, or possible caustic ingestion. Children younger than 16 years or patients shorter than 4′ should not be intubated with a Combitube.
- When a patient cannot be intubated or ventilated, placement of a surgical airway via needle cricothyrotomy, surgical cricothyrotomy (either open or modified), or RSI is required, which involves the coadministration of both anesthetic agents and neuromuscular blocking agents to produce a state of unconsciousness and paralysis that allow tracheal intubation.
- Some pharmacologic agents used in RSI are sedative/induction agents that include short-acting barbiturates such as sodium thiopental, benzodiazepines such as lorazepam and diazepam, anesthetic

agents such as etomidate and ketamine, opiates such as fentanyl, and lidocaine and atropine.

- Other pharmacologic agents used in RSI are neuromuscular blocking agents, which are either depolarizing or nondepolarizing. Succinylcholine is the only depolarizing neuromuscular blocking agent used in the United States, but there are several nondepolarizing neuromuscular blocking agents used, including vecuronium, pancuronium, rocuronium, and cisatracurium, and they are primarily used either as pretreatment agents, as the primary paralytic, or more commonly, to maintain post-intubation paralysis.
- Mechanical ventilation is the use of a machine that provides ventilatory support, ranging from simple to complex and of varying degree, depending on the capabilities of the machine.
- All ventilators have several common characteristics, including power source, cycling, breath delivery, parameters, circuitry and interface, and alarms, but not all ventilators have the same availability of all these parameters, nor do all ventilators have the same range of features.
- The indications for the need for mechanical ventilation range from apnea, managing the work of breathing, improving distribution of inhaled gases, to ventilatory and respiratory failure.
- In normal spontaneous breathing, a breath is initiated by a drop in the transrespiratory pressure gradient. Negative-pressure ventilators work in this manner by transmitting a negative pressure from the ventilator to the chest wall, thereby allowing inspiration to occur. Negative-pressure ventilators are used mainly on patients in rehabilitation or long-term care facilities or in the home.
- Positive-pressure ventilators use positive-pressure ventilation, in which air is pushed into the respiratory tract. Positive-pressure ventilators are often described by which variable terminates the inspiratory phase of the breath, such as pressure ventilators, volume ventilators, flow-cycled ventilators, and time-cycled ventilators, with volume ventilators being the most common.
- Conventional ventilation requires invasive ventilation in the form of placement of an artificial airway, chiefly the ET tube. Noninvasive ventilation is any form of mechanical ventilation without an artificial airway, but includes other devices such as CPAP and BiPAP, which use nasal or full-face masks to deliver a certain pressure to the lungs during spontaneous breathing.
- Both CPAP and BiPAP are contraindicated in patients who cannot protect their airways, have unstable facial fractures, or are uncooperative.
- Ventilator modes describe the type of breath delivered to the patient (ie, whether the breath is a mandatory machine breath or a spontaneous breath), may ensure a particular minute ventilation, and control the work of breathing.
- PEEP, not a true ventilator mode, occurs at the end of a mandatory machine breath, and instead of exhaling back to ambient pressure, the ventilator is set to stop the exhalation at a prescribed pressure, which results in an increase in the air in the alveoli, thus increasing the functional residual capacity, from which gas exchange occurs, and improving oxygenation. PEEP is contraindicated in patients with untreated pneumothorax or with bronchopleural fistula. During patient transport, few ventilator changes need to be made except for necessary adjustments of FIO_2 to accommodate changes in oxygenation. If ventilation needs to be initiated in the field, the CCTP should follow the recommended guidelines after securing a patent airway. The CCTP is responsible for ensuring proper ventilator function and that the patient's ventilatory status remains stable.
- Indications for using a portable ventilator include respiratory failure, apnea, inadequate gas exchange, decreased work of breathing and oxygen cost.
- The CCTP should be able to troubleshoot ventilator function, and a manual resuscitator should

accompany every patient on a ventilator.

Vital Vocabulary

3-3-2 rule A method used to predict difficult intubation. A mouth opening of less than three fingers wide, a mandible length of less than three fingers wide, and a distance from hyoid bone to thyroid notch of less than two fingers wide indicate a possibly difficult airway.

adventitious breath sounds Abnormal breath sounds that are heard in addition to, or in place of, normal sounds.

agonal respirations Slow, shallow, irregular respirations or occasional gasping breaths; results from cerebral anoxia.

alveolar ventilation (V_A) The volume of air that comes into contact with the alveolar capillary membrane surfaces and participates in the exchange of gases between the lung and blood.

anatomic dead space (V_D) Space in airway structures such as the trachea, bronchi, and bronchioles that does not participate in gas exchange. It is defined physiologically as ventilation without perfusion.

apnea The absence of respiration.

apneustic breathing A condition in which lesions in the respiratory center of the brain stem lead to a breathing pattern characterized by prolonged, gasping inspiration, followed by extremely short, ineffective expiration.

arterial blood gas (ABG) Analysis of the following characteristics of blood: pH, partial pressure of carbon dioxide (in arterial blood), partial pressure of oxygen (in arterial blood), concentration of bicarbonate ion, base excess (indicating whether the patient is acidotic or alkalotic), and oxygen saturation of the hemoglobin molecule.

auto-PEEP The nonintended increase in end alveolar pressure due to air trapping.

barotrauma Injury to the chest or lungs as a result of increased intrathoracic pressure.

bi-level positive airway pressure (BiPAP) The use of two separate pressures—inspiratory positive airway pressure and expiratory positive airway pressure—to raise the breathing baseline above ambient pressure. The pressure gradient enhances ventilation, and the reduced expiratory pressure makes exhalation easier and increases patient tolerance.

Biot's (ataxic) breathing Breathing characterized by three patterns: (1) slow and deep, (2) rapid and shallow, and (3) apnea. Causes include meningitis, increased intracranial pressure, and central nervous system dysfunction.

bradypnea A respiratory rate that is slower than normal.

bronchovesicular sounds A combination of the tracheal and vesicular breath sounds, heard in places where airways and alveoli are found, the upper part of the sternum and between the scapulae.

capnograph The measurement of exhaled CO_2, which in most cases correlates with the CO_2 levels in arterial blood, can be done with two different types of devices—an electronic monitor that displays a waveform or a colorimetric device that should turn yellow during exhalation, indicating proper tube placement.

capnometry The measurement of exhaled CO_2, performed the same way as capnography, but which provides a light-emitting diode readout of the patient's exhaled CO_2.

central neurogenic hyperventilation A pattern of very deep, rapid respirations at rates of 40 to 60 breaths/min, caused by a midbrain lesion or dysfunction.

Cheyne-Stokes respiration A cyclic pattern of increased respiratory rate and depth with periods of apnea. Causes include increased intracranial pressure, renal failure, meningitis, drug overdose, or hypoxia secondary to congestive heart failure.

cluster breathing An abnormal respiratory pattern in which a cluster of irregular respirations that vary in depth are followed by a period of apnea at irregular intervals.

continuous positive airway pressure (CPAP) A means of raising the breathing baseline above ambient pressure. The increased pressure across the entire breathing cycle increases the mean airway pressure, stents the airway, and increases the functional residual capacity, thereby improving oxygenation.

crackle Breath sound produced as fluid-filled alveoli pop open under increasing inspiratory pressure; can be fine or coarse.

esophageal detection device (EDD) A bulb or syringe that is attached to the proximal end of the ET tube; a device used to confirm proper ET tube placement.

eupnea Normal breathing at a rate of 12 to 20 breaths/min in the adult patient.

expiratory reserve volume (ERV) The air that can be expelled from the lungs after a normal exhalation.

face-to-face intubation Performing intubation at the same level as the patient's face; used when the standard position is not possible. In this position, the laryngoscope is held in the provider's right hand and the ET tube in the left.

flow-cycled ventilator A positive-pressure ventilator that ends inspiration when a predetermined flow rate is achieved.

flow rate The speed of the gas at which the tidal volume is delivered.

fraction of inspired oxygen (F_{IO_2}) Percentage of inhaled oxygen expressed as a decimal. For example, 40% oxygen = F_{IO_2} of 0.40.

functional residual capacity (FRC) The amount of air remaining in the lungs after normal expiration; the sum of residual volume and expiratory reserve volume.

hyperpnea A breath that is deeper than normal; can lead to low levels of CO_2.

hypopnea A shallow breath; can lead to increased CO_2 levels and decreased oxygen levels.

I time The time frame for the delivery of the tidal volume.

I:E ratio An expression for comparing the length of expiration to inspiration. The normal ratio is 1:2. This means that expiration is twice as long as inspiration. It is not measured in seconds.

inspiratory capacity (IC) The maximum amount of air that can be inspired; the sum of inspiratory reserve volume and tidal volume.

inspiratory reserve volume (IRV) The amount of air that can be inhaled after a tidal volume is inhaled.

invasive ventilation Application of mechanical ventilation through an artificial airway such as a tracheostomy or ET tube.

Kussmaul's respiration A fast and deep respiratory pattern without any periods of apnea. The rate and depth are greater than the normal rate expected; breathing is labored, with periods of deep breaths punctuated by sighs.

Mallampati classification A system for predicting the relative difficulty of intubation based on the

amount of oropharyngeal structures visible in an upright, seated patient who is fully able to open his or her mouth.

mean airway pressure The amount of positive pressure in the airway, averaged over the inspiratory and expiratory phases of the breathing cycle.

mechanical ventilation The application of a device that provides varying degrees of ventilatory support.

minute volume Total volume of air breathed in and out in 1 minute. It is calculated by multiplying the respiratory rate per minute by the tidal volume.

mode The particular way in which a spontaneous or mechanical breath is delivered.

negative-pressure ventilator A mechanical ventilator that operates using pressure that is less than ambient (atmospheric) pressure.

noninvasive ventilation Application of mechanical ventilation through a mask, mouthpiece, or other interfaces other than an artificial airway.

obstructive diseases Diseases that result in difficulty with moving air out of the lungs, such as asthma, COPD, cystic fibrosis, and bronchioectasis.

peak airway pressure The amount of positive pressure generated by the ventilator to deliver the tidal volume.

pleural friction rub The result of an inflammation that causes the pleura to thicken, decreasing the pleural space and allowing the surfaces of the pleura to rub together.

positive end-expiratory pressure (PEEP) The amount of pressure above ambient pressure present in the airway at the end of the respiratory cycle.

positive-pressure ventilator A mechanical ventilator that operates using pressure that is greater than ambient pressure.

pressure ventilator A type of positive-pressure ventilator that ends the delivery of the tidal volume based on a predetermined pressure; therefore, the volume is variable.

residual volume (RV) The amount of air remaining in the lungs after the expiratory reserve volume is exhaled.

respiratory depression A low respiratory rate (< 12 breaths/min in adults) for a prolonged period of time; also called hypoventilation.

respiratory failure A situation in which the respiratory system fails to meet the body's metabolic needs. If not reversed, it leads to respiratory or cardiopulmonary arrest.

respiratory insufficiency The inability of the respiratory system to keep up with the metabolic demands of the body.

restrictive diseases Diseases that result in difficulty moving air in to the lungs such as occupational lung diseases, idiopathic pulmonary fibrosis, pneumonia, atelectasis, chest wall deformities and injuries, and neuromuscular diseases that affect breathing.

retrograde intubation A technique in which a wire is placed through the trachea and into the mouth with a needle via the cricoid membrane. The ET tube is then placed over the wire and guided into the trachea.

rhonchi Rattling vibrations produced as air flows through mucus or around obstruction in the larger airways.

Seldinger technique A technique for obtaining vascular or other hollow organ access that uses a hollow

bore needle inserted percutaneously followed by placement of a soft-tipped guide wire. The needle is removed and a dilator is temporarily placed. The dilator is removed and the desired catheter is placed over the guide wire. The guide wire is then removed. The catheter is then secured.

sensitivity Ventilator control that regulates the amount of negative pressure required by the patient to initiate or "trigger" a breath.

shunt Perfusion without ventilation.

Spo_2 The noninvasive pulse oximetry measurement of oxyhemoglobin saturation by means of a beam of light applied to a superficial capillary bed such as the digits or ear lobe.

stridor High-pitched sound representing air moving past fluid or mechanical obstruction within or immediately above the glottic opening.

tachypnea An abnormally fast respiratory rate.

tidal volume (V_T) The volume of air moved into and out of the lungs with each respiratory cycle.

time-cycled ventilator A type of positive-pressure ventilator in which the ventilator ends inspiration after a selected inspiratory time has been achieved.

total lung capacity (TLC) The maximal amount of air that can fill the lungs; the sum of tidal volume, inspiratory reserve volume, expiratory reserve volume, and residual volume.

tracheal breath sounds Breath sounds heard by placing the stethoscope diaphragm over the trachea or over the sternum; also called bronchial breath sounds.

transillumination The act of using a light source placed within the trachea to visualize through the thin tissue that covers the trachea.

ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch A state of inadequate ventilation, perfusion, or both, in which there is inadequate gas exchange.

vesicular breath sounds Softer, muffled sounds in which the expiratory phase is barely audible.

vital capacity (V_c) The maximal amount of air that can be exhaled following a maximal inspiration; the sum of tidal volume, inspiratory reserve volume, and expiratory reserve volume; approximately 80% total lung capacity.

volume ventilator A type of positive-pressure ventilator in which the breath ends when the predetermined tidal volume is achieved.

$\dot{V}/\dot{Q}$ ratio The relationship between alveolar ventilation and alveolar capillary perfusion. The normal value is 0.8.

wheeze A high-pitched musical sound caused by airflow through a narrowed or constricted airway.

Bibliography

Alspach J. *Core Curriculum for Critical Care Nursing*. Philadelphia, PA: WB Saunders Co; 1998.

Butler TJ, Close JB, Close RJ. *Laboratory Exercises for Competency in Respiratory Care*. Philadelphia, PA: FA Davis; 1998.

Cottrell GP. *Cardiopulmonary Anatomy and Physiology for Respiratory Care Practitioners*. Philadelphia, PA: FA Davis; 2001.

Gausche-Hill M, Henderson D, Goodrich S, et al. *Pediatric Airway Management for the Prehospital*

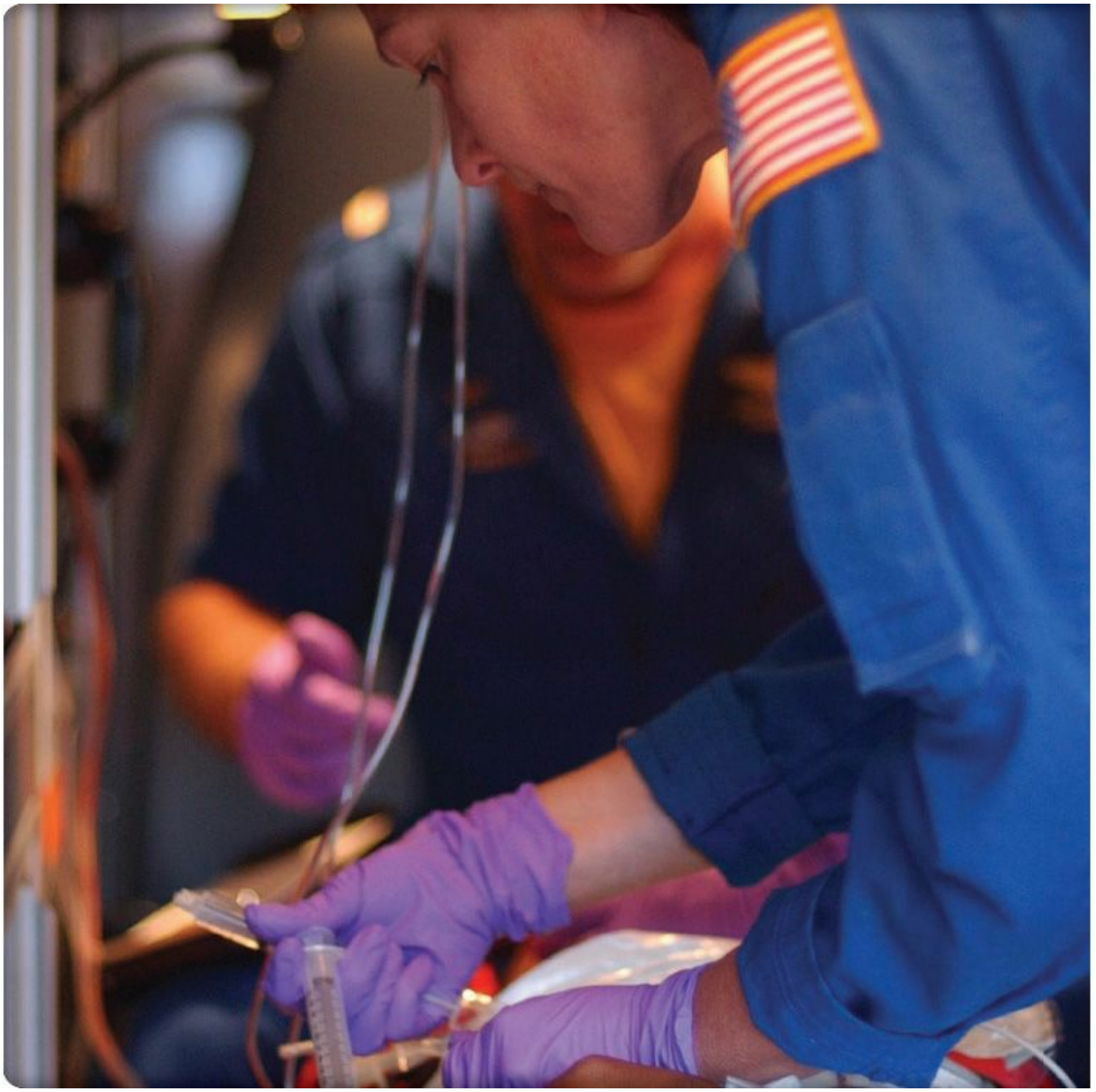
Professional. Sudbury, MA: Jones and Bartlett Publishers; 2004:48,52,82.

Margolis G. *Airway Management: Paramedic*. Sudbury, MA: Jones and Bartlett Publishers; 2004.

Scanlan CL, Wilkins RL, Stoller JK. In: Egan DF, ed. *Egan's Fundamentals of Respiratory Care*, 7th ed. St Louis, MO: Mosby Year Book; 1999.

Walls RM, Murphy MF, Luten RC, eds. *Manual of Emergency Airway Management*, 2nd ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2004:70,71.

Wilkins R, Krider SJ, Sheldon R. *Clinical Assessment in Respiratory Care*, 4th ed. St Louis, MO: Year Book; 2000.



Pharmacology

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Objectives

1. Outline the principles of medication administration for CCTPs, including patient and medication selection, predicted and desired responses, absorption and elimination principles, side effects or adverse medication reactions, and transport and monitoring considerations (p 188–194).
2. Identify and discuss reliable resources for medication and pharmacology information (p 188).
3. Discuss the significance of medication pharmacokinetics and pharmacodynamics in the critical care transport setting (p 189–190).
4. Identify airway management medications used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 195–197).
5. Identify respiratory management medications used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 197–201).
6. Identify cardiovascular system medications used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 201–210).
7. Identify medications for neurologic conditions that are used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 210, 214–215).
8. Identify medications affecting the gastrointestinal system that are used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 215, 217–218).
9. Identify any miscellaneous medications that are used during critical care transport, including indications, contraindications, dosages, side effects, and interactions (p 218–222).
10. Understand the sequence for medication infusion and know how to infuse medication with an infusion pump during transport and through changes in altitude (p 222, 224).

Introduction

Pharmacology is defined as the study of the preparation, properties, uses, and actions of medications. Modern health care and modern society are heavily dependent on medication for the management of illness and the alleviation of medical conditions or symptoms. Pharmacology plays a significant role in pre-hospital and critical care medicine and is often an essential component of patient care and transport. The practice of transporting critical care patients requires an enhanced understanding of all aspects of pharmacology in order to provide optimal patient care without causing further patient injury or death. Although this chapter covers a brief review of important basics, it assumes a general understanding of the subject of pharmacology. Many specific medications and medication groups are discussed. This list, however, is not comprehensive. As new medications and approaches to treatment of various conditions emerge, it is essential to balance this new information with proven treatment strategies. As always, follow local regulations, policies, and protocols and consult reliable, current sources when administering any medication.

Pharmacology and Medication Information

CCTPs should stay abreast of newly approved medications and current research. Current, reliable medication information may be obtained from a variety of sources, many of which are available in print and electronic formats **Table 7-1**.

In recent years, the Internet has emerged as a readily accessible source for medication information. Take some time to familiarize yourself with reputable and reliable Internet resources. Software versions of some of these resources, such as Epocrates, are also available for notebook computers, personal digital assistants (PDAs), and pocket personal computers. Regardless of the format used, careful research of the source's accuracy will assist in obtaining reliable information for clinical practice.

A Practical Approach to Medication Administration

Medication administration is a delicate blending of art and science. Many factors assist with medication selection for a particular condition.

The optimal medication for a particular patient may or may not be immediately available during patient transport. Many specialized medications such as antidotes, thrombolytics, and antibiotics are not routinely carried by CCTPs. Expense, storage concerns, and infrequent use are common limiting factors. A sending facility or a transport program's sponsoring hospital pharmacy are potential resources when a necessary medication is needed emergently but not carried by the transport team. Before leaving a sending facility, transport crews should obtain a sufficient quantity of a specialized medication infusion to account for transport time, plus an additional amount to provide a safe margin against unanticipated delays. If a desired medication remains unavailable, the transport team must decide if there is an acceptable alternative.

TABLE 7-1 Sources of Drug Information

Source	Description
American Medical Association *Drug Evaluation*	A nonofficial compendium that provides another source of useful drug information for pharmacists and medical practitioners; includes generic and trade names; information may not be limited to drugs approved for use by the FDA.
Drug inserts	Printed document included in the packaging provided by the drug's manufacturer; generally the same information submitted and approved by the FDA; when available, serves as a valuable reference; should not be confused with the information provided by a pharmacy when a patient receives a prescription, which is useful in obtaining information pertaining to a drug but is not necessarily inclusive.
Hospital formulary	A list of approved drugs, dosage forms, package sizes, and drug strengths stocked by hospitals and pharmacies. Published as a quick reference to assist the physician, registered nurse, pharmacy staff, and adjunctive staff. Divided into four general sections: introduction, therapeutic index, drug monographs, and general reference.
Physician's	Compiles data on most medications available in the United States; uses the information on file with the FDA; includes information regarding indications, dosages, contraindications, and adverse reactions.

Desk Reference	The size of the book makes it difficult for use on an ambulance or aircraft, but CD-ROM and electronic versions make it more accessible in the field. This source provides a vast amount of material on each medication, which may make it difficult to obtain any specific information needed immediately by CCTPs.
USDA Center for Drug Evaluation and Research	The FDA mission is to ensure that safe and effective drugs are available in the United States.
Various commercial medication reference texts and materials	These materials are widely available in print and electronic formats. Educational institutions, industry organizations, and independent publishing companies provide medication information in formats ranging from a simple listing of medications and doses (*Pharmacopeia*), to complex compilations that include an exhaustive list of interactions, overdose/toxicity treatments, pharmacokinetics, and administration guidelines. Select materials may only include medications related to a particular specialty (such as obstetrics, psychiatry, and critical care).

Abbreviations: FDA, Food and Drug Administration.

The scope of practice and monitoring capabilities of the transport team must also be considered when selecting a medication. Many vasoactive medications are best administered with continuous hemodynamic monitoring and require infusion pumps for precise adjustments. The transport team must be adequately trained and equipped to recognize and effectively manage anticipated side effects or adverse reactions associated with a particular medication. Transport teams who do not perform adequate monitoring or are unable to properly administer a medication place that patient at grave risk.

Other considerations include the clinical status of a patient. If immediate, life-saving interventions are required, it would be best to postpone the administration of scheduled, routine medications. Additionally, significant safety or operational priorities related to an aircraft or ambulance overshadow routine medication administration.

CCTPs must consider factors specific to each medication before administration. Indications, pharmacodynamics, pharmacokinetics, side effects, and potential adverse effects must all be considered. A particular medication is indicated when a specific condition requires treatment. Indications are further based on Food and Drug Administration (FDA) approval of the medication. A medication may have numerous, often seemingly unrelated indications.

Pharmacokinetics

Pharmacokinetics is the study of the metabolism and action of medications within the body, with particular emphasis on the time required for absorption, duration of action, distribution in the body, and mechanism of excretion.

Absorption

Absorption of a medication occurs either immediately or shortly after its administration. The route of administration affects how quickly and how much of the dose is absorbed. CCTPs generally use

intravenous (IV), intramuscular (IM), subcutaneous (SC), oral (PO), transdermal, nasal, and sometimes rectal routes for medication administration. Other routes not usually performed by CCTPs include ocular, intra-arterial, otic, and vaginal.

The route of medication administration affects its **bioavailability**, the amount of an administered medication (fraction or percentage) that reaches systemic circulation without any alteration. IV medications consistently have 100% bioavailability. The bioavailability of medications administered orally ranges from 0% to 100%, depending on the substance. Lidocaine, for example, when given orally only reaches approximately 35% bioavailability compared with 100% when given intravenously. Differences in bioavailability illustrate how a dose of a particular medication may be safe when given orally but toxic or lethal when given intravenously. For example, significant differences between the oral and IV dose exist for beta-blockers and calcium channel blockers. Nasal, sublingual, and rectal routes provide safe, alternative access for select medications when IV access cannot be established. The bioavailability of medications administered by these routes and other nonintravenous routes is based on numerous factors, including perfusion, membrane pH, and surface area along with variations based on the type of medication, chemical, or method of preparation.

Distribution

Once absorption has occurred, medication distribution to the tissues is highly variable. Blood is the primary distribution vehicle. Substances will distribute into the body based on the size of the molecule, ionization, binding to tissues or plasma proteins, lipid or water solubility, and perfusion to a region. Placental and brain barriers restrict distribution of certain substances into fetal and brain tissue, respectively. Highly lipid-soluble medications tend to cross these barriers where other medications would not readily cross. The solubility of a medication also affects dosage calculations for weight-based medications.

First-pass effect (also called first-pass metabolism) occurs with orally administered medications, in which the concentration of a medication is reduced as it passes through the liver before it enters systemic circulation. The medication undergoes biotransformation, elimination, or both. The kidneys and urinary system are the main routes of elimination. Patients with acute or chronic renal disease require careful consideration when medications are administered. Other routes of elimination include the lungs (usually highly volatile substances or gases), sweat, saliva, stool, and breast milk.

Biotransformation

The liver is the primary site for biotransformation, but many body tissues and cells have the ability as well. Four possible events occur during biotransformation of a medication. An active drug may be changed into an inactive metabolite. This new substance causes no new effects and is then ultimately eliminated. An active drug can also be changed into an active metabolite, for example procainamide into *N*-acetylprocainamide, that causes continued or new clinical effects. An active medication may also biotransform into a reactive metabolite that has a greater potential for action than its parent substance (for example, acetaminophen into *N*-acetyl-p-benzoquinoneimine). Finally, an otherwise inactive substance undergoes biotransformation into an active, potentially toxic substance (codeine changed into morphine). Active (and reactive) metabolites can complicate the clinical situation and require special consideration when selecting certain medications. The presence of active (and reactive) metabolites becomes particularly important when considering a medication half-life or monitoring a medication plasma level. Larger tertiary care centers often have the ability to assess medication metabolite levels in addition to plasma levels of the medication itself.

Elimination

Substances or medications undergoing elimination follow one of two possible patterns. If a substance follows **zero-order elimination**, a fixed amount of substance is eliminated over a time period, regardless of the amount of substance present in the plasma. Ethanol follows zero-order elimination. Regardless of how much ethanol is present, only a relatively fixed amount gets excreted each hour. Most medications and other substances follow **first-order elimination.** In first-order elimination, the amount of substance eliminated over a period of time is proportional to the plasma concentration. Medication half-lives reflect first-order kinetics. A medication **steady state** occurs when administration of a medication equals the rate of elimination, which is desirable with antibiotics and anticoagulants.

Factors affecting biotransformation and elimination include perfusion, liver and kidney status, metabolism, and the manner and extent of absorption. Biotransformation and elimination are also influenced by chronic exposure to a particular medication or chemical.

Enzyme systems in the liver perform biotransformation. The cytochrome P450 system, present in the liver and in other tissues, is the main pathway responsible for biotransformation of many medications and chemicals. This system is also manipulated by many medications and chemicals, leading to altered patterns of biotransformation in select persons. Common agents such as nicotine, grapefruit juice, and broccoli have a significant impact on the functioning of the cytochrome P450 system. The list of medications that either promote or inhibit the action of the cytochrome P450 system on other medications is exhaustive.

Pharmacodynamics

Pharmacodynamics is the way in which a medication produces the response intended, also known as the **mechanism of action**. It encompasses the factors that may alter the intended response and any side effects or unexpected effects.

Theories of Drug Action

Medications are chemicals that are administered to cure or treat a disease, symptom, condition, or other anomaly. When these chemicals act upon the body to achieve a particular goal, four basic mechanisms may occur. A medication may bind with a receptor site on a particular cell to either promote or inhibit a specific activity of that cell. A medication may also affect the physical property of a cell, which in turn alters the functioning of that cell. A medication may combine directly with other chemicals within the body to alter or limit the effects of this chemical or allow it to be removed. Finally, a medication may alter a metabolic pathway in order to achieve a desired result.

Medications that bind to a receptor site are the most prevalent, particularly in the prehospital setting. Receptor sites are specialized proteins on a cell that receive chemical mediator messages to stimulate a particular response. For example, when acetylcholine attaches to receptor sites in the heart, it causes the heart rate to slow. Cellular responses can be wide ranging depending on the chemical mediator and the cells being stimulated. A medication molecule will have one of two effects when it attaches to a receptor site. It may stimulate the receptor site to cause the response it normally does (agonist), or it may block the receptor site from being stimulated by other chemical mediators and inhibit the normal response (antagonist). Some medications can also act as agonist-antagonists, or partial agonists, by performing both roles. In any event, the medication molecule must compete with the naturally occurring chemical mediator. In order for a medication to work effectively, the medication molecule must have a higher affinity for the receptor than the intrinsic chemical mediator does. In addition, more than one medication may vie for the same receptor.

Once the medication is bound to the receptor site, it initiates a chemical change that produces the expected effect by direct binding or by release of a second compound (known as a second messenger) that causes the intended effect. Cyclic adenosine monophosphate (cAMP) is the most common second-messenger chemical related to pharmacology. Once cAMP is produced in the cell, it triggers the release of still other enzymes, which then carry out their own functions.

The number of available receptors is inconsistent and can be affected by the actual number of sites present, the number already occupied by another chemical mediator, and the number occupied by another medication. As medication molecules bind to the receptor sites, the number of receptors decreases, a process known as down-regulation. However, some medications can increase the number of available receptor sites, a process known as up-regulation.

Drug-Response Relationship

Once the medication finds the target tissue, it needs to accumulate to a sufficient concentration to produce its desired effect. The drug-response relationship correlates the amount of medication given and the response it causes. Most of this information comes from the plasma-level profiles, which describe the length of onset, duration, and termination of action. These profiles allow pharmacologists to determine the minimum effective level of a medication and the level at which the medication would be toxic to the patient. When a medication is administered, the **onset of action** is defined as the amount of time it will take for the concentration of the medication at the target tissue to reach the minimum effective level. The CCTP also needs to know how long the medication can be expected to remain above that minimum level to provide the intended action (ie, the **duration of action),** which is defined as the amount of time that the medication can be expected to remain above that minimum effective level. The **termination of action** is the amount of time after the concentration level falls below the minimum level to the time it is eliminated from the body.

All of these factors affect the **therapeutic index**—the ratio of a drug's lethal dose for 50% of the population (LD_{50}) to its effective dose for 50% of the population (ED_{50}). In other words, the therapeutic index gives an indication of a medication's margin of safety. The plasma profile also provides information about the medication's biologic **half-life (T½)**—that is, the time it takes the body to eliminate half of the drug. Despite the name, a medication is not eliminated in two "half-lives." During the second half-life period, only half of what remained after the first half-life is eliminated. After three half-lives, approximately one eighth of the medication still remains. In many instances the duration of action of a medication is unrelated to its half-life. Aspirin, for example, has a half-life of 15 minutes, yet produces antiplatelet effects that last for 7 days. Typically, a medication has no effect after five to seven half-lives.

Factors Affecting Drug Responses

Patient characteristics affect the action of a drug. Patients differ with respect to how they react to medications. Age, gender, weight, heredity, and clinical condition each have an impact on medication choice, medication response, required dose, and elimination characteristics.

Age

Patients of different ages may have vastly different responses to the same drug. Older people, for example, tend to be much more sensitive to the effects of drugs and often require smaller doses than younger patients. Elderly patients frequently exhibit declining renal function and decreased muscle mass, which interfere with the reliability of creatinine clearance when determining an appropriate medication dose. Additionally, changes in body water content, relative to body fat content, change the distribution of water- and lipid-soluble medications, requiring dosage adjustments. Changes in or a reduction of certain

neurotransmitters further complicates medication dosing in the elderly. A person's metabolism will vary significantly at different ages throughout life. Pediatric patients generally have an increased metabolic rate compared with adult and elderly patients and often require an increase in either dose or frequency of administration. It is not, however, solely a matter of dose. Some drugs have different effects altogether in different age groups.

Sedative-hypnotic agents tend to exhibit a more profound or prolonged effect in older patients. Excitement, rather than sedation, is also possible when elderly patients receive certain medications. Barbiturates, for example, act as sedatives in most adults, but may produce excitement or agitation in older patients. Pediatric patients frequently experience paradoxical drug reactions in which sedative agents cause hyperactivity and agitation, further complicating the clinical situation. Diphenhydramine, chloral hydrate, and certain benzodiazepines are frequently the cause of paradoxical medication reactions in children.

Weight

Many drugs are formulated for an average adult, usually considered to be a 154-lb (70-kg) person. However, the ultimate drug concentration will be quite different if the same dose is administered to a 106-lb (48-kg) person and a 300-1b (136-kg) person. Therefore, body weight must be taken into consideration by calculating dosages in milligrams or micrograms per kilogram of the patient's body weight (mg/kg or µg/kg, respectively). The nature of a particular drug, including receptor sites and tissue distribution, will determine whether the medication dosage is based on a patient's ideal body weight rather than actual body weight.

Special Populations

Pediatric and geriatric patients often have slower medication absorption and elimination times, necessitating modification of the doses of many drugs administered to these patients. Pregnant patients are limited in the medications they can take because of the risk to the fetus.

Special Populations

Almost all medications require lower doses for elderly patients because of diminished renal clearance. CCTPs should be aware of this when administering drugs. Additionally, medications that have a high first-pass metabolism rate through the liver can require additional medication doses to obtain the optimal serum levels. Consult a specific, reliable reference for each medication that is being considered for oral administration, and be sure to rely on medical direction at all times.

For example, digoxin should be administered according to a patient's ideal body weight. Lidocaine should be administered according to a patient's actual body weight. Research is limited on many other weight-based medication dosages; in practice, critical care medications are administered and titrated based on effect. Clinicians should consider tissue binding, protein binding, and perfusion when administering weight-based medications. Ideal body weight in adults is calculated as follows:

- **Males (kg):** 50 + (2.3 times patient's height in inches over 5′)
- **Females (kg):** 45.5 + (2.3 times patient's height in inches over 5′)

Gender

General differences in body mass between males and females affect reactions to medications. Some

medications can have varying effects in the different sexes, particularly when they are based on hormones.

Environment

The environmental milieu can influence a medication's reaction because of the psychological and physiologic stresses imposed on the patient. In addition, a particular reaction to an environmental factor, such as seasonal allergies, can alter response to a medication.

Time of Administration

The time at which a patient is given a medication may affect the reaction. If a patient takes an oral medication immediately after eating, the time of onset is likely to be slowed compared with taking the same drug on an empty stomach or during physical activity such as exercise.

Condition of the Patient

The patient's overall health also affects the response to many drugs. If the kidneys are not working properly, for example, medications may not be excreted efficiently, and the concentration of a drug may build up inside the body until it reaches toxic levels. Liver and kidney failure significantly alter the metabolism and elimination of many medications. *Consult a reliable medication reference or seek medical direction before administering medications to patients with significant liver or kidney dysfunction*. If the patient is in shock, it may take hours before the patient absorbs a drug that has been injected. In a situation such as profound anaphylaxis, it may be necessary to administer medications intravenously that are normally administered intramuscularly or subcutaneously. Medications may also not be effective in hypothermic patients. IV medications are often withheld if a patient's core temperature has fallen below 80°F (30°C) and given at greater intervals when hypothermic patients have a core temperature above 80°F (30°C). Chapter 18 provides a more detailed discussion of medication administration for the treatment of hypothermia.

Genetic Factors

A patient's genetics may affect the reaction to a medication. If a patient lacks necessary enzymes or has a lower metabolism, the response to a medication might be significantly delayed. Glucose-6-phosphate dehydrogenase deficiency is a common genetic disorder affecting hundreds of millions of people worldwide. The administration of oxidant medications (such as antimalarials), sulfonamides, nitrofurantoin, and phenazopyridine and exposure to naphthalene (mothballs) cause a potentially life-threatening hemolytic anemia in these patients. Other genetic disorders such as primary pulmonary hypertension and sickle cell disease have specific concerns related to pharmacology and hemodynamics. Medications with vasoconstrictor properties will adversely affect lung perfusion in patients with pulmonary hypertension. Any medication that causes hypoxia, vasospasm, or dehydration can precipitate or worsen a sickle-cell crisis.

Pregnancy

Medication administration in pregnant patients has special risks. Medications pass from the pregnant woman's bloodstream into the placenta and ultimately affect the fetus. During pregnancy, patients also experience impaired respiratory excursion, hemodilution, and altered hemodynamic parameters. There is evidence that catecholamines such as norepinephrine and epinephrine have an adverse impact on placental perfusion, but these medications are generally limited to use in extreme situations, such as profound shock or cardiac arrest. Ephedrine, because of its beta-2 and alpha-1 properties, is the preferred vasopressor for maternal shock. The FDA has established five categories to indicate a drug's potential for causing abnormal prenatal development. **Table 7-2** lists the categories and their implications.

Psychological Factors

The relationship between psychological factors and medication administration during critical care transport cannot be overstated. In several research studies, subjects were given a placebo, a pill with no therapeutic or medicinal properties. Some subjects began to exhibit effects as if they were taking real medication. This is known as the placebo effect, which has been noted in up to 10% of the subjects in some studies.

Psychological factors need to be considered when medications are administered for analgesia and sedation. Patient positioning, temperature regulation, and proactive communication can optimize the effects of analgesia or sedative medications, possibly decreasing the overall amount of each medication required. Occasionally, patients with psychiatric disorders require excessive amounts of sedatives for the desired effect or demonstrate a paradoxical medication reaction similar to children with select medications, especially with benzodiazepines.

TABLE 7-2 FDA Pregnancy Risk Categories

Pregnancy Category	Implications
A	Controlled studies in women fail to demonstrate a risk to the fetus in the first trimester (and there is no evidence of a risk in later trimesters), and the possibility of fetal harm appears remote.
B	Either animal-reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women, or animal-reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester (and there is no evidence of a risk in later trimesters).
C	Either studies in animals have revealed adverse effects on the fetus (teratogenic or embryocidal or other) and there are no controlled studies in women, or studies in women and animals are not available. Drugs should be given only if the potential benefit justifies the potential risk to the fetus.
D	There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (eg, if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
X	Studies in animals or human beings have demonstrated fetal abnormalities, there is evidence of fetal risk based on human experience, or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.

Data source: FDA, Title 21: Food and Drugs, Code of Federal Regulations, Title 21, Volume 4, Part 200 to 299. Revised as of April 1, 1997.

Predictable Responses

Because of the extensive research that goes into developing and testing a medication before it is approved, generally there is a good idea of potential patient responses to a particular drug. CCTPs should also anticipate unexpected drug responses beyond the desired effect, called **side effects**. Side effects are reactions that can manifest as signs or symptoms that are not the desired effects, but nevertheless are expected based on how the medication works. All medications have some side effects—even oxygen.

CCTPs need to be able to evaluate the benefits compared with the risks associated with the side effects. If the patient's condition will improve more so than deteriorate from side effects, the CCTP will likely administer the medication. Conversely, if the risks for side effects outweigh the benefits of drug treatment, it is best to withhold drug administration. This concept is known as the risk-benefit ratio. The risk-benefit ratio is evaluated separately for each patient and his or her particular clinical situation. A side effect that is insignificant for one group of patients may have life-threatening implications for another group of patients, such as genetically susceptible, immunocompromised, or hemodynamically unstable individuals. For example, if a medication causes hypotension as a side effect, it should be avoided in patients with shock. If a medication alters immune response, it may place immunocompromised patients at significant risk. Patients with G6PD deficiency, a genetic disorder discussed previously, may experience hemolytic anemia as a side effect of sulfonamides and oxidant medications. The risks associated with medication side effects should not be underestimated in high-risk or critically ill patients.

Iatrogenic Responses

When the administration of drugs leads to symptoms that mimic naturally occurring disease states, it is known as an **iatrogenic response**. An iatrogenic response is an adverse condition inadvertently induced in a patient as a result of treatment. For example, a urinary tract infection may develop after insertion of an indwelling catheter, or Stevens Johnson syndrome is a potentially fatal complication of treatment that may develop with medications such as penicillin, sulfa antibiotics, anticonvulsants, and at least one cyclooxygenase-2 inhibitor. Although Stevens-Johnson syndrome is often tough to predict or anticipate, the consequences of this syndrome and other iatrogenic drug responses are devastating and potentially fatal.

Unpredictable Responses

Even though the doses of medications have been carefully tested and designed for specific conditions, some patients have adverse effects that have not been anticipated. The most common unpredictable response encountered in the prehospital setting is an allergic reaction. An allergic reaction occurs when the patient is extremely sensitive, or hypersensitive, to the medication, or one of the ingredients used in its particular form or preparation. Because the patient is hypersensitive, the drug activates the immune system. Allergic reactions are unpredictable—unless the patient has had an allergic reaction to the same drug in the past—and may lead to life-threatening anaphylaxis. Allergic reactions should be anticipated with any drug. Severe reactions to an antibiotic that is penicillin-based may indicate a greater chance of reaction to another antibiotic that is penicillin-based. Patients with egg or soy allergies are not candidates for sedation with propofol (Diprivan). An allergy to bananas or avocados is highly suggestive of a latex allergy. There are numerous other allergy patterns between food, environmental, and medication allergies that CCTPs should evaluate prior to administering a medication to patients with known allergies.

In rare cases, the patient may experience a completely unique response that is specific to that person; it is not seen in other patients. This situation is known as an **idiosyncrasy** and is unrelated to the pharmacologic action of a medication or the dose administered.

Patients who take a particular medication for an extended period can build up **tolerance** to it. In these cases, the patient will often require higher doses than normal to achieve the desired response. Patients may also develop a tolerance to one medication as a result of prolonged administration of another medication, known as **cross-tolerance**. This phenomenon is often seen in patients who take several pain medications. A patient who is taking a medication such as oxycodone may become tolerant to opiate-based medications. Therefore, if morphine is administered for pain, the patient may not have the desired response because of cross-tolerance. **Tachyphylaxis** is a condition in which the patient rapidly becomes

tolerant to a medication. With tachyphylaxis, increasingly higher doses of a medication are required to achieve desired effects. This can occur with just a couple of doses and is most often seen with **sympathomimetic** agents. It is important to keep in mind, however, that toxic thresholds may remain unchanged. With prolonged administration of a medication, a patient can become drug dependent. In this case, the person will have significant symptoms if he or she stops using the medication.

All medications need to reach a minimum concentration in the target tissue before they become effective; that concentration is reached by providing a specific dose. If several doses are given in a relatively short time, the patient may experience a **cumulative effect**, which is an increased effect when a medication is given in several successive doses, possibly resulting in therapeutic or nontherapeutic effects.

Many patients take multiple medications at one time. It is possible for the effects of one medication to alter the response of another medication, a phenomenon known as a **drug interaction**. The interaction may not always be anticipated. For example, if the patient is taking two sympathomimetics, it does not necessarily mean the patient will experience a more dramatic sympathetic response. It is possible to see the opposite response or a completely unrelated response. When the patient is taking more than one medication, one medication could block the body's response to another medication. This is called **drug antagonism**. For example, if a patient has taken an opiate-based drug such as morphine, another medication, naloxone, can be administered to block the response to the morphine.

A **summation effect** occurs when two drugs given to the patient have the same effect, which doubles the response exhibited by the patient. When the patient receives two drugs that have the same effect but produce a response greater than the sum of their individual responses, the result is known as **synergism**. At times, the interaction between two medications can cause one drug to enhance the effect of another, known as **potentiation**. For example, acetaminophen (Tylenol) and alcohol interact. It is well known that high doses of acetaminophen are damaging to the liver. When alcohol is ingested along with acetaminophen, more of the medication is taken up into the liver and may result in acute liver failure. This phenomenon is referred to as the alcohol-acetaminophen syndrome. Some potentiation effects are known and can be exploited to achieve a desired effect; in other cases, potentiation may occur unexpectedly. A direct biochemical interaction that takes place between two drugs is referred to as **interference**. **Inhibition** is the opposite of potentiation, where one medication limits the effects of another.

Avoiding Medication Errors

Medication errors related to selection, administration, or monitoring are responsible for thousands of deaths annually. The risk of a medication error leading to an adverse patient outcome is high. CCTPs, in addition to ensuring the right patient, right drug, right dose, right route, right time, and right formulation, must evaluate medication decisions made by sending facility staff for potential errors. It is essential that CCTPs confirm the accuracy of any medication infusion that they are continuing during transport. Proper labeling of transport medications and infusions is essential for CCTPs when accepting a patient and turning over patient care. Additionally, line incompatibility should be considered for any patient receiving multiple IV medications. Numerous medications will either chemically interact or precipitate (take on a solid form) when combined in the same IV line, causing unpredictable or dangerous consequences for the patient.

Medication Management

Medication Storage

Many medications can be altered due to extremes in temperature, exposure to direct sunlight, or excessive humidity. In most cases, the potency of the medication is decreased, and, in some cases, the actual molecular components can be degraded and made inactive. Each manufacturer must provide guidance on the proper storage for each medication that is approved by the FDA. This information can be found on the drug insert or in the *United States Pharmacopeia*. In general, medications should be kept out of direct sunlight and stored in temperatures between 55°F (13°C) and 85°F (29°C). During transport, CCTPs and vehicles are exposed to temperature extremes. It is essential that medications remain within their required temperature range. Medications such as cardizem (Diltiazem), various neuromuscular blocking agents (succinylcholine and rocuronium), and lorazepam (Ativan) require refrigerator storage. These medications are only stable at room temperature for a limited period of time. Consult the manufacturer's guidelines for adjusted expiration dates with room temperature storage and anticipate frequent replacement of these medications if they are stored at room temperature.

Transport vehicles require special consideration for medication storage. CCTPs should monitor the temperature of transport vehicles when they are exposed to extremes of heat or cold. Medications may need to be removed while the vehicle is parked. Smaller electronic devices are now available that can record the temperature of a vehicle and identify periods of deviation from safe medication storage temperatures. CCTPs should be conscious of these general guidelines, use their best judgment, and ensure that medications are stored in areas that are not routinely or constantly exposed to poor climate conditions. Every department should have a written protocol or procedure for the specific handling of all medications, fluids, or diluents on the vehicle or in the station as a guide on how best to store and handle medications used in daily practice. In addition, CCTPs should check the expiration dates on medications on the transport vehicle at least monthly and dispose of the expired lots.

Security of Controlled Medications

Nearly every medication used in the prehospital setting is a prescription medication; however, some are controlled substances and have more stringent control guidelines. State and federal governments heavily regulate the acquisition, storage, usage, and disposal of controlled medications. State pharmacy boards and the US Drug Enforcement Agency have specific regulations that CCTPs must follow in these situations. Minimum requirements for the storage of controlled substances include a securely locked, substantially constructed cabinet with no sign or any other indication that the cabinet is used for the storage of controlled substances. Cabinets constructed of materials that are fragile and/or allow visualization, such as glass or plastic, are considered inappropriate for this purpose. A controlled substance disposition record must be maintained, and thorough documentation must be completed for any use of a controlled substance, including the disposal of any leftover waste medication not administered during patient care. Any expired controlled substances must be returned to the agency or departmental officer in charge of maintenance and dispersal of controlled substances and must also be documented in the disposition record(s). These are general guidelines. Every department must have a written protocol and procedure for the use, storage, disposition, and documentation of controlled substances.

Specific Medications Used in Critical Care Transport

Individual medications and medication families common to critical care transport are examined here, but this list is far from complete. CCTPs should be prepared to encounter many additional medications in clinical practice and have a strategy in place to responsibly administer or monitor patients who have received these medications. Conversely, not every medication or medication family here may be relevant to providers who only perform critical care transport of specialty populations (for example, high-risk obstetric or neonatal populations). Each medication or medication family is organized based on its most

common use. It is common for medications to have accepted uses in multiple categories. The dosages, kinetics, and related materials for each medication may vary slightly from one reference source to another. As always, follow local system or organizational protocols regarding administration guidelines for each medication.

Medications Used in Airway Management

Definitive airway management in critical care transport combines techniques for airway device placement with medications designed to facilitate placement. Sedative-hypnotic agents are administered with chemical paralytics to create the best patient condition possible for invasive airway procedures. Ideally, the patient should have no gag reflex, no trismus, relaxed facial/airway muscles for maximal visualization, no awareness of the procedure, and no memory of the procedure. These goals can be accomplished with the proper use of the medications described in this section. Various airway management protocols also include mucolytics for secretion control, IV analgesic medications, and anesthetic use, which are discussed later in this chapter.

Ultra-Short-Acting Nonbarbiturate Hypnotic Agents

For patients who require endotracheal intubation, ultra-short-acting nonbarbiturate hypnotic agents are quickly becoming the gold standard. Etomidate (Amidate) is the drug of choice in this category for intubation because of its effective action and few side effects. Etomidate depresses the reticular activating system by stimulating the gamma-aminobutyric acid (GABA) receptors. It decreases oxygen consumption and cerebral blood flow, which makes it ideal in cases of increased intracranial pressure. Administered at a dose of 0.3 mg/kg, etomidate reaches peak effect in 1 minute and provides anesthesia for about 3 to 5 minutes. There are no absolute contraindications for the use of etomidate for rapid sequence intubation. However, a high incidence of uncomfortable myoclonic muscle movement is associated with its use. Recent studies suggest that even a single dose of etomidate may result in adrenal suppression, which has caused some to reconsider its use in critically ill patients.

Ketamine, a phencyclidine (PCP) derivative, is another option for rapid sequence intubation and general anesthesia. Ketamine is also ideal because of its rapid onset of 1 minute or less and duration of action of 10 to 15 minutes. The drug makes the patient unaware of his or her surroundings and fall into a trance-like state. It works by interrupting pathways between the cortex, which is responsible for higher functions such as language and perception, and the limbic system, which provides emotions and memories. By interrupting these pathways, ketamine is believed to provide its dissociative, analgesic, and sedative properties. Typically given at a dose of 1 to 4 mg/kg, ketamine produces anesthesia, analgesia, and amnesia. This drug is abused in the general population so increased security is required for this medication.

Ketamine tends to cause an increase in blood pressure, pulse, and cardiac output. This can be both a benefit and a drawback depending on the cardiovascular state of the patient. Patients with uncontrolled hypertension could be further compromised by the effect of ketamine, whereas patients who are in a state of hypoperfusion may benefit. Ketamine has also been found to have bronchodilatory effects. The exact mechanism of this effect is unclear, but it has been found to be beneficial in patients with bronchospasm. CCTPs should anticipate an emergence reaction, which includes hallucinations, agitation, irrational behavior, and delirium, following ketamine administration. These reactions can be minimized or prevented by premedication with benzodiazepines such as lorazepam (Ativan) or midazolam (Versed).

Sedative-Hypnotic Agents

Propofol (Diprivan) is another sedative-hypnotic agent used in airway management. The normal adult dose is 1 to 2.5 mg/kg for induction; some providers administer 40 mg every 10 seconds until induction is

accomplished. Propofol provides both anesthetic and amnesic effects beginning about 20 seconds after administration and lasting for 10 to 15 minutes. In certain states it is outside the registered nurse and paramedic scope of practice to use propofol as an induction agent, but acceptable for use as a maintenance sedative in the intensive care unit (ICU) and during patient transport. The maintenance dose ranges from 5 to 50 μg/kg/min. Many alternate dosing regimens are available for pediatric patients, elderly patients, general anesthesia, and select compromised patients or situations. Consult a reliable reference for specific dosing parameters for these patients. In addition to use as a sedative-hypnotic agent, propofol has some powerful anticonvulsant properties. It may be useful in patients with status epilepticus who have resisted frontline anticonvulsant medications such as phenytoin and benzodiazepines. Propofol may also be the maintenance sedative of choice for short-term use in hemodynamically stable neurologic patients who are predisposed to seizure activity.

Propofol decreases myocardial activity and systemic vascular resistance. Patients could experience a decrease in oxygen delivery to vital organs, most notably the brain (evidenced by significant drops in blood pressure). Propofol should be used with caution in patients who would experience the devastating impact of hypotension (such as traumatic brain injury). Decreased blood pressure from propofol administration may prove beneficial in patients who are experiencing hypertension from the effects of sympathomimetic drugs. Additionally, propofol has been shown to have a greater effect on depressing pharyngeal and laryngeal muscle tone, possibly facilitating endotracheal intubation.

Propofol requires a strict aseptic technique during preparation and administration. The lipid-based preparation serves as a great breeding ground for invasive microorganisms. Additionally, propofol infusions should not be continued for longer than 48 hours. High doses of propofol or prolonged infusions have caused patient fatalities from propofol infusion syndrome, which includes metabolic acidosis, rhabdomyolysis, cardiac and renal failure, and several other serious conditions. Propofol has numerous medication interactions, through either alteration of the cytochrome P450 system or action to potentiate other central nervous system (CNS) and respiratory depressant medications and chemicals.

Benzodiazepines

Many airway management protocols include the use of benzodiazepines either during the initial induction or as a maintenance sedative. Benzodiazepines include common critical care medications such as diazepam (Valium), lorazepam (Ativan), and midazolam (Versed) along with oral medications for long-term home use, including oxazepam (Serax) and clonazepam (Klonopin). These medications have anxiolytic, sedative, and anticonvulsant properties. The three common critical care benzodiazepines are all pregnancy class D, which indicates a strong risk to the fetus. These medications should only be used in life-threatening situations in which no safer medication alternatives exist. Benzodiazepines have been linked to fetal anomalies and neonatal floppy baby syndrome.

Benzodiazepines are believed to affect the inhibitory neurotransmitter GABA in the brain, although their exact mechanism of action is not fully understood. Benzodiazepine molecules bind to a receptor near GABA binding sites, which is thought to enhance their affinity for GABA. This increased affinity causes brain activity to slow.

Like propofol, benzodiazepines should be used with caution in patients with hypotension or potential hypotension.

Of the three most popular benzodiazepines, midazolam (Versed) is the most common choice for rapid sequence intubation or induction because of the relatively fast onset time when compared to other benzodiazepines at 1 to 5 minutes when administered at a dose of 2.5 to 10 mg IV in adults. Midazolam should generally be used as an adjunct to other sedative-hypnotic agents rather than as the sole medication for induction. It has a 60-minute duration of action and peaks in approximately 30 minutes. If midazolam is to be used as the sole induction agent, it must be administered 3 minutes prior to proceeding with any

chemical paralytic agents. In addition, midazolam has a beneficial potent amnesic effect, which inhibits the patient's ability to recall the procedure.

Midazolam is believed to carry out its effect by altering the neurotransmitter GABA. As such, midazolam acts as a CNS depressant, but it also has hypnotic and anterograde amnesic properties. Although patient recovery time is dose dependent, patients can take as long as 2 hours to recover. Some interfacility transport systems may use a narcotic premedication, such as fentanyl, when using midazolam in order to gain a potentiation effect.

Diazepam (Valium) is a moderately long-acting benzodiazepine with a duration of action of 30 to 90 minutes, but has a slower onset than midazolam, at approximately 5 minutes, when patients are given between 2 and 10 mg IV. Lorazepam (Ativan) is a benzodiazepine medication that is gaining popularity. This medication has a rapid onset of 1 to 5 minutes, but a variable peak effect.

Diazepam and lorazepam are excellent first-choice anti-convulsants. Midazolam works well, but not as well as diazepam and lorazepam for seizure control. In critical care transport it is possible that only one of these medications may be available for use. Benzodiazepines are administered emergently for active seizure activity and supplemented by other anticonvulsants in patients who do not initially respond.

Midazolam, diazepam, and lorazepam are used routinely at reduced dosages for anxiolysis before and during transport. Use caution when administering these medications to patients who are predisposed to paradoxical or bizarre medication reactions, such as children, the elderly, and psychiatric patients.

In the event a patient has excessive levels of benzodiazepines, the use of flumazenil (Romazicon) may be necessitated. Flumazenil is used to treat benzodiazepine overdoses by antagonizing the benzodiazepine receptors. Adult patients are given an initial dose of 0.2 mg by IV catheter over 30 seconds. After 30 seconds of observation, a second dose of 0.3 mg can be administered over 30 seconds. If the desired effect is not yet achieved, 0.5 mg can be administered every minute, up to 3 mg total. Pediatric patients can receive 0.002 to 0.02 mg/kg IV every minute. Extreme caution must be exercised with the use of this medication. Flumazenil should be avoided in patients who have overdosed on any medication with the potential to cause seizures, patients with any unknown drug overdoses, patients with a history of seizures, any patient likely to experience a seizure (such as traumatic brain injury or intracranial hemorrhage), and any patient who has been taking benzodiazepines long-term. With all of these contraindications, the use of flumazenil in critical care transport is limited.

Paralytic Medications

Chemical paralytic medications inhibit muscle activity (and resistance) in patients who require airway and respiratory muscle relaxation to facilitate airway adjunct placement or mechanical ventilation. Intubation success rates are improved as a result of increased visualization of airway anatomy following chemical paralysis. **Acetylcholine (ACh)** is the neurotransmitter that bridges the synapse at the neuromuscular junction. An influx of calcium in the nerves stimulates the release of ACh, which moves across the synapse and binds with the **nicotinic receptors** on the muscle cells. When ACh makes that connection, depolarization of the muscle takes place and the intended movement or action takes place. A paralytic medication can be grouped into one of two categories depending on how it interacts with ACh and the nicotinic receptors: depolarizing or nondepolarizing.

Special Populations

Remember to reference a length-based resuscitation tape (such as the Broselow tape) for resuscitation medication dosages for children. Medication dosages and other pediatric topics are covered in Chapter 23.

Depolarizing Agents

Depolarizing paralytics work by acting as an agonist in the nicotinic receptor and mimicking the activity of ACh. The medication stimulates muscle depolarization but then remains bound to the receptor, thereby preventing it from repolarizing and being triggered again. Succinylcholine (Anectine) is the only depolarizing chemical paralytic agent in widespread clinical practice. In addition to activity on the nicotinic receptors, succinylcholine also stimulates cardiac muscarinic receptors, leading to bradycardia, especially in children. At a dose of 1 to 2 mg/kg, patients will experience muscle relaxation in about 30 seconds, with total paralysis occurring in about 45 seconds. The rapid onset time, coupled with the duration of action at 7 to 10 minutes, makes succinylcholine an ideal paralytic agent for medication-assisted airway management.

Succinylcholine is not without its drawbacks. It has a tendency to increase serum potassium levels, which can cause significant problems in the presence of chronic or acute conditions in which potassium levels may be altered. Acute injuries such as burns, crush injuries, and spinal cord injuries should be treated with alternative medications if they are available. Chronic conditions affecting potassium, such as renal failure, should also receive an alternative medication. Succinylcholine can also cause a transient increase in intracranial pressure (ICP). Although the increased ICP from laryngoscopy is of greater concern, the medication-induced ICP must be considered and accounted for in cases of closed-head injury. The most common side effect is muscle fasciculations, which is the twitching activity seen as the muscles depolarize. Although the twitching may not appear overly dramatic, care should be given to ensure the patient does not experience any further injury or inadvertently pull IV lines. Many rapid-sequence intubation or induction (RSI) protocols include a reduced dose of a nondepolarizing muscle relaxer prior to succinylcholine to prevent or reduce any fasciculations.

Succinylcholine can cause malignant hyperthermia and therefore is contraindicated in patients who have a known history or family history of this condition. Malignant hyperthermia is discussed in greater depth in Chapter 20. Also, expect a much longer than normal duration of action of succinylcholine when it is administered to any patient exposed to acetylcholinesterase inhibitors, found in chemical nerve agents and pesticides. This is the result of inhibition of acetylcholinesterase, the enzyme that breaks down ACh. A nondepolarizing muscle relaxer may be preferred in these situations.

Nondepolarizing Agents

Nondepolarizing agents work by competitively antagonizing ACh at the postsynaptic receptors. In doing so, the ACh is unable to cause stimulation, and muscle depolarization cannot occur. A few different nondepolarizing medications are available, and some are more desirable for medication-assisted airway management than others. Overall, the major drawback in using nondepolarizing medications is that they tend to have a slower onset time and longer duration of action when compared with succinylcholine.

Rocuronium (Zemuron) has the fastest onset time of the nondepolarizing paralytics, at 60 to 90 seconds. It is generally administered at a dose of 0.5 to 1 mg/kg, but at that dose the medication lasts for as long as 45 minutes. A higher initial dose (1 mg/kg) permits the shorter onset time in RSI procedures. Subsequent maintenance doses can be reduced to 0.5 mg/kg. In the event that definitive airway management cannot be established, providers are then required to manage an apneic patient for an extended period of time. Heated debate persists whether rocuronium or succinylcholine is the preferred medication for emergent chemical paralysis during RSI procedures. The side effects of succinylcholine are weighed against the longer duration of rocuronium. Several other nondepolarizing muscle relaxers are available for ongoing chemical paralysis during patient transport or in a critical care unit.

CCTPs may encounter other nondepolarizing muscle relaxers. Vecuronium (Norcuron), pancuronium (Pavulon), cisatracurium (Nimbex), and atracurium (Tracrium) each have the same mechanism as rocuronium. The onset, duration, and certain side effects will vary among these medications. Long-term

chemical paralysis has many serious implications for CCTPs and other health care providers. Muscle weakness and neuropathies can persist long after chemical paralytic medications have been discontinued. These conditions can impair ventilator weaning, can prolong ICU admissions, and will further compromise patient recovery. It is essential that patients only receive chemical paralytic agents for the shortest duration and lowest possible dose. Intensive care providers can use a train of four measurement, post-tetanic count, or double-burst stimulation to assess for the correct level of medication in patients receiving long-term chemical paralysis. Special instruction is required before CCTPs perform or interpret these tests. Proper utilization of these tests will prevent excessive dosing of paralytic medications, which will minimize the risk and severity of long-term complications. *Train of four and other paralytic monitoring does not at all reflect the underlying level of sedation and analgesia in chemically paralyzed patients*. It is imperative that CCTPs and other health care providers carefully evaluate the level of sedation and analgesia in any patient who is chemically paralyzed. In many patients, particularly those with unstable hemodynamics, assessment of sedation and analgesia through vital signs is unreliable. Conversely, it is common for hypertension and tachycardia to be incorrectly treated with vasoactive medications when the underlying cause is undersedation and pain. *Inadequate sedation and analgesia in chemically paralyzed patients is commonplace in intensive care and critical care transport*. CCTPs must take careful steps to avoid this situation in any patient who is transported.

Patients undergoing chemical paralysis are also at high risk for decompensation or death when CCTPs do not carefully match ventilation settings with patient needs. Hyperventilation, evidenced by increased minute ventilation, is often a compensatory mechanism in critically ill patients. Once a patient is chemically paralyzed, disastrous consequences can occur if inadequate ventilator settings are used.

Airway medications used in critical care transport are summarized in **Table 7-3**.

Medications Used in Respiratory Management

Effective respiration requires both the movement (ventilation) of air into and out of the lungs as well as gas exchange at the alveolar-capillary level. When respiration becomes compromised, numerous medications are available to augment either of these two functions. Beta-agonist medications act upon receptors in the lower airways to improve airflow into and out of the lungs. Additional medications improve oxygenation and ventilation through further bronchodilation, inflammation reduction, and secretion control.

Various inhaled gases and exogenous surfactant appear to improve oxygen delivery and exchange in patients with respiratory disorders. The research continues regarding the safety and efficacy of these approaches. Specialized training and equipment are required for these medications and chemicals to be used in ICUs or during critical care transport, beyond the scope of this text.

Beta-Agonist Medications

Beta receptors play a significant role in the management of respiratory disease. There are two types of beta receptors in the sympathetic nervous system. Beta-1 receptors are found in the cardiac tissue and increase the heart rate, cause cardiac muscle to contract, produce automaticity, and trigger cardiac electrical conduction. Beta-2 receptors are found in the peripheral vasculature and in the respiratory tract. They stimulate vasodilation and bronchodilation. Stimulation of the beta-2 receptors allows treatment of asthma and other diseases that cause excessive narrowing of the bronchioles, such as respiratory syncytial virus, bronchitis, or emphysema.

TABLE 7-3 Airway Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Ultra-short-acting nonbarbiturate hypnotic agents: etomidate type				
Induction agent to facilitate airway device placement	Depresses the reticular activating system by stimulating GABA receptors	Etomidate (Amidate): IV	Adrenal suppression, apnea, hypotension, hypertension, tachycardia, bradycardia, myoclonus, nausea, vomiting	Multiple doses increase the likelihood of adrenal suppression. Avoid extravasation.
Ultra-short-acting nonbarbiturate hypnotic agents: ketamine type				
Induction agent to facilitate airway device placement	Interrupts pathways within the cortex and limbic system; prompts release of endogenous catecholamines	Ketamine (Ketalar): IV or IM	Hypertension, emergence reactions, increased ICP, vivid dreams, tremors	Consider premedication with benzodiazepines to prevent an emergence reaction. Ketamine may be the preferred induction agent in patients with respiratory failure.
Sedative-hypnotic agents				
Induction agent to facilitate airway device placement; short-term maintenance sedative for intubated patients; anticonvulsant medication used for refractory status epilepticus	Alkyl-phenolic compound with anesthetic properties; exact mechanism is unclear, but it is unrelated to other common opioid or sedative agents	Propofol (Diprivan): IV	Including but not limited to: hypotension, apnea, hypertension, cardiac arrhythmias, propofol infusion syndrome, iatrogenic infections, tachycardia, bradycardia	Use with caution in any patient who would experience devastating consequences to hypotension, even transient (such as in patients with traumatic brain injury). Use strict aseptic technique when handling medication or IV tubing. Avoid prolonged use. Administer at the minimum dose possible.
Benzodiazepines				
Sedative agents, often administered to facilitate airway device placement; maintenance sedative agents; anxiolytic agents; first-line anticonvulsant agents	Binds with benzodiazepine receptors on GABA neurons; enhances GABA inhibition of the CNS resulting in CNS cellular depression	Diazepam (Valium): IV, IM, PO, rectally Lorazepam (Ativan): IV, IM, PO Midazolam (Versed): IV, IM, PO	Sedation, respiratory depression, apnea, hypotension, ataxia, vasodilation	Benzodiazepines may cause paradoxical reactions in children. They are not ideal for use as a sole agent for anesthesia induction. They may produce desirable amnesia effects. The antagonist flumazenil (Romazicon) is available for use is situations involving benzodiazepine overdose. Use with caution in any patient with hypotension or potential hypotension.
Depolarizing muscle relaxant agent				
Short-term chemical paralytic to facilitate airway device placement	Activates, binds, and then occupies with acetylcholine receptor sites at the	Succinylcholine (Anectine): IV; may be given IM in extreme (emergency)	May cause or worsen hyperkalemia, causes a transient increase in	Consider pretreatment with atropine in patients prone to

airway device placement	receptor sites at the neuromuscular junction, preventing further activation, leading to chemical paralysis	extreme (emergency) circumstances. Onset: 30–45 s Peak effects: 1–3 min Duration: 7 to 10 min	transient increase in ICP; may cause bradycardia (especially in children), muscle fasciculations, or malignant hyperthermia	patients prone to bradycardia. Do not administer succinylcholine in repeat doses or as an IV drip. Avoid or use with caution in patients with acetylcholinesterase poisoning. Use with caution in patients with neuromuscular disease. Provides no analgesia or sedation; must be accompanied by analgesic and sedative medications.
Nondepolarizing muscle relaxant agents				
Short-term chemical paralytic to facilitate airway device placement; chemical paralytic agents for patients who require ongoing chemical paralysis for transport and certain modes of mechanical ventilation	Competitively antagonizes acetylcholine at receptor sites of the neuromuscular junction, causing extended chemical paralysis	Rocuronium (Zemuron): IV bolus or infusion. Onset: 60–90 s Duration: 45 min Vecuronium (Norcuron): IV bolus or infusion. Onset: 30 s Peak effects: 2.5–3 min Duration: 25–30 min Pancuronium (Pavulon): IV bolus or infusion. Onset: 30 s Peak effects: paralysis in 3–5 min Cisatracurium (Nimbex) IV bolus or infusion. Onset: 2–3 min Peak effects: 3–5 min Duration: 25–90 min Atracurium (Tracium): IV bolus or infusion. Onset: 2–3 min (dose dependant). Peak effects: 5–7 min Duration: 60–70 min	Apnea in unventilated patients, hypotension, hypertension, tachycardia, or bradycardia; atracurium may cause bronchospasm or excessive secretions	Use in caution in patients with neuromuscular disease. These medications provide no analgesia or sedation and must be accompanied by analgesic and sedative medications.

Abbreviations: CNS, central nervous system; GABA, gamma-aminobutyric acid; ICP, intracranial pressure; IM, intramuscular; PO, oral.

Beta-2 agonists may produce some beta-1 effects as well. As the dose of a medication increases, beta selectivity decreases. Beta-2 antagonism serves no clinical purpose; there are no circumstances in which the goal is to reduce the amount of air getting to the lungs via bronchoconstriction. Bronchoconstriction becomes a concern with beta-antagonist (beta-blocking) medications (discussed later) as the dose is increased and beta selectivity disappears.

Albuterol is a common sympathomimetic (mimics the body's sympathetic response) beta-agonist medication used in the treatment of respiratory disorders. Albuterol is considered a selective beta-2 agonist because it affects beta-2 receptors with little or no effect on beta-1 receptors. At a typical dose of 2.5 to 5 mg via nebulizer, albuterol relaxes the smooth muscles in the airway, thereby reducing constriction. Following inhalation or nebulization, the medication is deposited directly at the affected airway tissue, which provides a faster onset with reduced systemic effects. Caution must still be taken, however, because side effects, although fewer, may still occur. Although albuterol is a selective beta-2 agonist, we can still see beta-1 agonism in the way of increased chronotropic and inotropic effects. Cardiac manifestations occur frequently. These increased effects can increase myocardial oxygen demand in a situation in which there is a reduced oxygen supply. Cardiac manifestations range from mild tachycardia and hypertension to chest pain, arrhythmia, angina, myocardial ischemia, and even hypotension. Additionally, agitation, insomnia, dizziness, and irritability are common. In younger, healthy patients, the benefits tend to outweigh the drawbacks because the medication is intended to ultimately

increase oxygen supply. As patients get older with concomitant diseases, the risks and benefits of albuterol should be carefully considered. Despite the potential risks of the many side effects described, albuterol is well tolerated by most patients.

Patients with severe asthma, chronic obstructive pulmonary disease, or other reactive airway disease exacerbations are often placed on a continuous albuterol nebulizer regimen. Increased or prolonged administration places patients at risk for a variety of serious medical conditions, including metabolic acidosis, severe hypokalemia, and many of the cardiac manifestations previously discussed.

Levalbuterol (Xopenex) is a newer, more expensive alternative for patients at high risk for adverse reactions or side effects from albuterol. Headaches, nervousness, and dizziness are the most common side effects. Cardiac manifestations are relatively rare, with tachycardia occurring most frequently. Levalbuterol inhalation solution does not contain lecithin, a preservative used in some albuterol inhalation preparations that may trigger an allergic response in patients with a peanut allergy.

Terbutaline (Brethine) is a beta-2 agonist that is used as a bronchodilator and is commonly used as a tocolytic but is not FDA approved. As a beta-2 agonist, this medication also relaxes the airway smooth muscles to reduce constriction. With a dose of 0.25 mg SC every 20 minutes, this medication has been shown to begin improving respiratory status in as little as 5 to 6 minutes, but the peak effect is generally not seen for 20 to 30 minutes. The side effects are consistent with other beta-2 agonists, which include tachycardia, high blood pressure, trembling, and anxiety. Additionally, terbutaline may decrease serum potassium levels, which has the potential to cause cardiac arrhythmia, although this appears uncommon. Terbutaline should not be used for patients with tachycardia as a result of cardiac arrhythmias.

In more critical cases, it may be necessary to use a nonselective beta-agonist medication. In this case, epinephrine is used and can be administered subcutaneously or via a nebulizer. In either event, the dose should be 0.3 mg of epinephrine of a 1:1,000 (1 mg/mL) concentration. If nebulized, the medication should be diluted with 2.5 mL of normal saline. Racemic epinephrine is also available commercially as a single-dose ampule, premixed for nebulization. Subcutaneous administration requires no dilution. Although either route of epinephrine administration can be expected to have a rapid onset, both carry a significantly higher incidence of side effects. In such cases, the benefits must outweigh the risks of tachycardia, increased myocardial oxygen consumption, and peripheral vasoconstriction.

Mucokinetic and Bronchodilator Medications

In addition to the beta-agonist medication, a handful of other classifications of medications may be used to dilate the patient's lower airway.

The anticholinergic medication ipratropium bromide (Atrovent) is often used concurrently with albuterol. As an anticholinergic medication, ipratropium antagonizes the parasympathetic nervous system to reduce mucosal secretions in the airway and dilate the airway, although to a lesser extent compared with beta-agonists. Ipratropium tends to have a long onset time of 20 minutes and a half-life of 2 hours. It is most commonly administered via nebulizer at a dose of 0.5 mg every 20 minutes for 3 doses. The side effects are consistent with anticholinergic medications; they include nervousness, gastrointestinal (GI) upset, dizziness, and headaches. This medication should be avoided in patients with narrow-angle glaucoma.

Although not commonly used in the acute setting, theophylline is another non–beta-agonist option. The method by which theophylline functions remains uncertain. It is thought to stimulate a release of catecholamines and stimulate the diaphragmatic muscles. In critical care settings, the recommended dose is 6 mg/kg over 20 to 30 minutes for a loading dose, which is followed by a maintenance infusion of 0.5 to 0.7 mg/kg/h. Theophylline has numerous side effects and drug interactions. The most serious adverse reactions include respiratory arrest, seizures, and cardiac arrhythmias. Patients may also experience GI upset, headaches, agitation, tachycardia, and palpitations. Patients taking several types of antibiotics or

thyroid medications should not receive theophylline.

Corticosteroids

The treatment of inflammation, especially in the airway, is anchored by the use of corticosteroids. Corticosteroids work as anti-inflammatory agents but also have been found to increase the number of beta receptors in bronchial smooth muscle. A medication in this classification should be used in most severe and moderate cases of bronchial constriction. Pharmacologists remain unclear on exactly how corticosteroids carry out their functions, but do know that they reverse increased capillary permeability and thereby reduce inflammation.

Prednisone and methylprednisolone (Solumedrol) are commonly used in emergency and critical care settings. Both medications have a prolonged time of onset that can be as long as 4 to 6 hours. Corticosteroid treatment should be withheld in patients suspected of having many different types of infectious disease processes. A mild respiratory illness that accompanies or precipitates a reactive airway disease exacerbation is not generally a contraindication to corticosteroid therapy. The typical dose for prednisone is 40 to 60 mg/d, whereas the one-time dose for methylprednisolone is 125 mg. GI upset, hyperglycemia, and electrolyte imbalances tend to be the most common side effects.

Dexamethasone (Decadron) is also a corticosteroid used for inflammatory disorders, including airway edema, increased ICP, and high-altitude pulmonary edema. The use of dexamethasone has somewhat waned in recent years as the result of associated complications, including severe infections, hyperglycemia, hypokalemia, and myasthenia gravis. It is typically administered at a dose of 4 mg. Adverse reactions include appetite and mood changes, hyperglycemia, and anxiety.

Prednisolone is a corticosteroid that has the lowest percentage of placental crossing. It is sometimes used when absolutely necessary in pregnancy. The dose is 1 mg/kg per os (by mouth) in pediatrics and 40 to 60 mg per os in pregnant adults.

Respiratory medications used in critical care transport are summarized in **Table 7-4**.

Medications Affecting the Cardiovascular System

The cardiovascular system is complex. This section of the chapter discusses the variety of medications commonly encountered when treating the cardiovascular system. The cardiovascular system is made up of the pump (the heart), the container (the vasculature), and the fluid (the blood). The medications used to treat problems with each of the three components are examined.

When looking at the heart, CCTPs should have three main areas of concern: the **chronotropic** aspect, the **inotropic** aspect, and the cardiac rhythm. Each of these is vital to the effective functioning of the heart. Some medications have effects on two or three of the aspects, whereas others have effects limited to only one aspect. Occasionally clinicians and reference sources mention the **dromotropic** aspect, which relates to intracardiac conduction and can be altered by certain medications as well. Dromotropy becomes a concern when multiple cardioactive medications are administered simultaneously. Medications such as catecholamines, beta-blocking agents, calcium channel–blocking agents, and anticholinergic agents may simultaneously affect the heart and blood vessels. Other medications involved in the management of cardiovascular conditions include anticoagulants and thrombolytics, phosphodiesterase inhibitors, and diuretics.

Alpha-Adrenergic Antagonists (Alpha-Blockers)

Alpha receptors are present in vascular smooth muscle and cause vasoconstriction when acted upon by endogenous catecholamines, particularly epinephrine and norepinephrine. Numerous medications antagonize alpha receptors as an unwanted side effect. Haloperidol (Haldol is discussed later in this chapter) and trazodone (Desyrel) are two psychiatric medications that possess alpha-antagonistic

properties causing hypotension, often postural, as a side effect. The alpha-blocker class of medications has been developed to treat hypertension, benign prostatic hyperplasia, glaucoma, and pheochomocytomas (catecholamine-producing tumors) by blocking alpha receptors in the vasculature and select organs (such as the eyes, intestines, or bladder). Clonidine (Catapres) and phentolamine (Regitine) are alpha-blocking medications encountered periodically during critical care transport. Numerous other alpha-blocking oral medications are in use clinically and may be observed as a patient's personal medication. These include terazosin (Hytrin), doxazosin (Cardura), and methyldopa (Aldomet). Labetolol is a medication used in critical care medicine with both alpha-antagonistic and beta-antagonistic properties.

TABLE 7-4 Respiratory Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Beta-agonist medications				
Reactive airway disease, bronchospasm	Activate beta-2 receptors in the respiratory tract, causing bronchodilation	Albuterol (Ventolin): Nebulization, metered-dose inhaler Levalbuterol (Xopenex): Nebulization, metered-dose inhaler Terbutaline (Brethine): SC Epinephrine (Adrenaline): Nebulization, SC, IM, IV	Tachycardia, hypotension, hypertension, chest pain, CNS stimulation, irritability, nervousness, headache, palpitations	Prolonged use of albuterol may cause metabolic acidosis. Use with caution in patients with cardiovascular disease.
Anticholinergic bronchodilator medications				
Reactive airway disease, bronchospasm	Block acetylcholine at bronchial smooth-muscle receptor sites	Ipratropium (Atrovent): Nebulization, metered-dose inhaler	Palpitations, bronchitis	Administered only every 6-8 h.
Theophylline derivatives				
Reactive airway disease, bronchospasm	Block phosphodiesterase and stimulate the release of endogenous catecholamines	Theophylline or Aminophylline: PO and IV	Tachycardia, nervousness, restlessness, nausea, vomiting	Numerous food and medication interactions may occur.
Corticosteroids				
Inflammatory disorders, airway edema, cerebral edema, high-altitude pulmonary edema	Suppress immune response; restore normal capillary permeability; limit neutrophil migration	Methylprednisolone (Solumedrol): IM or IV Prednisone: PO Dexamethasone (Decadron): PO and IV Prednisolone: PO	Insomnia, nervousness, immunosuppression, hyperglycemia	Use with extreme caution or avoid in patients with many active infections. Numerous side effects may occur, especially with long-term administration. Numerous medication interactions may occur.

Abbreviations: CNS, central nervous system; IM, intramuscular; PO, oral; SC, subcutaneous.

Clonidine is given during a hypertensive crisis, 0.1 to 0.2 mg orally. Common side effects of clonidine include dry mouth, dizziness, drowsiness, weakness, and orthostatic hypotension. The onset is typically ½ to 1 hour with effects lasting from 6 to10 hours.

Phentolamine should be carried by any transport provider who administers any IV medication with alpha-adrenergic properties (such as norepinephrine, epinephrine, and dopamine). These medications ideally should be administered through well-placed central venous access. In many situations, only peripheral IV access is available or possible to obtain. If inadvertent peripheral extravasation of alpha-adrenergic medication occurs, the resulting tissue necrosis can be devastating. Diluted phentolamine is injected into affected tissues near the site of extravasation to prevent local vasospasm and cell or tissue death. Effective therapy is demonstrated by normal skin color return within 1 hour.

Labetalol (Trandate, Normodyne) is a medication with both alpha- and beta-blocking (antagonist) properties. It can be given orally or intravenously for the management of chronic or emergent hypertension, respectively. It is contraindicated in pregnancy, although it has been recommended for use in moderate to severe preeclampsia. Avoid administering beta-blockers, including labetalol, when a patient has a high-degree (greater than first-degree) heart block or sick sinus syndrome. Heart failure and cardiogenic shock are other situations in which labetalol is contraindicated. The initial IV dose is 0.25 mg/kg, with escalating doses up to 40 to 80 mg each to a maximum total dose of 300 mg. Labetalol can also be administered as a continuous IV infusion of 2 mg/min (also to a maximum total dose of 300 mg). Effects begin in less than 5 minutes following an IV dose and last up to 4 hours.

Angiotensin-Converting Enzyme Inhibitors

Angiotensinogen is a blood plasma protein component and is acted upon by renin, which is released from the kidneys in response to decreases in blood pressure, to form angiotensin I. Angiotensin-converting enzymes, also found in the bloodstream, transform angiotensin I into angiotensin II. Angiotensin II is a formidable vasoconstrictor, but its creation is slowed by medications that inhibit angiotensin-converting enzymes, or ACE inhibitors. ACE inhibitors also block the breakdown of the vasodilator bradykinin, which also improves blood pressure. These medications are contraindicated if patients have any history of an angioedema reaction to prior administration of an ACE inhibitor.

Captopril (Capoten) is a popular ACE inhibitor found in the critical care setting. In addition to its use for hypertension, captopril may be used for congestive heart failure and acute myocardial infarctions. Typically given at a dose of 12.5 to 50 mg orally, patients should be monitored for hypotension, dizziness, and electrolyte imbalances. The drug effects peak in 1 to 1.5 hours and have a half-life of less than 3 hours.

The critical care paramedic may also use enalapril (Vasotec) to control a patient's blood pressure. It is often given intravenously to patients with hypertension or heart failure. Enalapril is also a common ACE inhibitor and works similarly to captopril, but it has a much longer half-life at 11 hours. Enalapril begins working in approximately 1 hour and peaks in 3 to 5 hours. Enalapril is given at a dose of 0.625 to 1.25 mg IV every 6 hours and has side effects similar to captopril.

Antiarrhythmic Medications

Patients with cardiac rhythm disturbances create a considerable challenge for CCTPs. In extreme circumstances, these rhythm disturbances can deteriorate into a complete loss of organized cardiac activity. The management of cardiac arrhythmias involves several classes of antiarrhythmic medications, many of which create additional risk for catastrophic and lethal rhythm disturbances. For a review of cardiac action potential, see Chapter 13.

The classification of antiarrhythmic medications is a general, broad-based class that is broken down further based on the medication's mechanism of action according to the **Vaughan-Williams classification scheme**. Class I antiarrhythmic medications all work by blocking sodium channels. In doing so, the medication reduces the phase 0 slope and the peak of the action potential. Class I drugs are further divided into IA, IB, and IC based on their strength and degree of phase 0 reduction. Class IA is considered moderate, class IB is weak, and class IC has a strong reduction. The general principle behind sodium channel blockades is to reduce the rate and magnitude of depolarization by decreasing conduction velocity in non-nodal tissue. Recall that nodal tissue generates the initial electrical impulse. Non-nodal tissue receives stimulation from nodal tissues or neighboring cells to initiate depolarization. With the advent of newer medications with several simultaneous mechanisms of action (amiodarone, for example), the Vaughan-Williams classification system has significant limitations.

Class I Antiarrhythmic Medications

Class IA medications have been found to increase the effective refractory period (ERP). This term refers to the interval within the cardiac cycle during which cardiac cells (and muscles) are chemically unable to reenergize. During this time additional action is impossible. By increasing the ERP, the medication interrupts reentry mechanisms by decreasing the cells' ability to recharge and depolarize as quickly. The class IA medications tend to be anticholinergic medications such as quinidine, procainamide, and disopyramide. There are some significant drawbacks with this class, however, particularly pertaining to the effects on patients with preexisting infarctions or structural heart diseases. They also tend to have significant adverse reactions, including lupuslike syndromes (inflammation of the heart tissue including the valves and pericardium), in as many as 30% of long-term procainamide users.

Of the class IA medications, procainamide is the medication most commonly encountered in the acute setting. Given as an infusion, the typical dose is 20 to 30 mg/min, with a maximum dose of 17 mg/kg. When administering procainamide, the CCTP must also be mindful of the clinical endpoints of a 50% widening of the QRS complex, the arrhythmia being suppressed, or the patient becoming hypotensive. Along with the lupuslike syndromes, the patient may also experience widening of the PR and QT intervals, atrioventricular blocks, seizures, and CNS depression.

Class IB medications are slightly different in that they carry out their functions by decreasing the ERP and the action potential duration (APD). The APD is the amount of time that the action potential cycle takes to occur. When the ERP and APD are decreased, the automaticity is decreased, which reduces the tendency to depolarize in a disorganized fashion. These medications are indicated for ventricular fibrillation, ventricular tachycardia, and ventricular irritability. Lidocaine has a greater impact on ventricle cells with longer action potentials than atrial cells with shorter action potentials. Lidocaine is the prototypical class IB medication. At a dose of 1 to 1.5 mg/kg and a repeat dose of 0.5 to 0.75 mg/kg, lidocaine remains a treatment option for patients experiencing cardiac arrhythmias originating in the ventricles. Some clinicians and organizations such as the American Heart Association have decreased the emphasis on lidocaine in favor of amiodarone (discussed later) for treatment of certain ventricular arrhythmias. The medication reaches peak effect in about 5 minutes but has a highly variable duration of action. Caution should be exercised so as not to exceed the maximum dose of 3 mg/kg.

Class IC medications work by suppressing the phase 0 repolarization. Class IC medications differ from the other class I medications in that they have virtually no effect on the action potential. By suppressing repolarization, the conductivity is reduced through the heart. Class IC medications are used for life-threatening arrhythmias originating in the ventricles. Some studies have indicated a propensity for ST-segment elevation with the use of class IC medications. Flecainide is a prototypical class IC medication. This oral medication is not routinely used in critical care transport. Interestingly enough, medications in this class can be proarrhythmic as well. They can precipitate ventricular arrhythmias, heart failure, and cardiac arrest, seemingly in the case of an acute myocardial infarction within the previous 2 years.

Class II Antiarrhythmic Medications/Beta-Adrenergic Blocking Agents

The class II antiarrhythmics are comprised of the beta-blocking medications. The cardiovascular system is influenced by the sympathetic nervous system, particularly by epinephrine and norepinephrine binding to the adrenergic beta-1 and beta-2 receptors. Beta-blocking medications work to antagonize the beta receptors by competitively binding to the beta receptor sites to inhibit stimulation. Earlier beta-blocking medications were considered nonselective because they indiscriminately blocked both beta-1 and beta-2 receptors. More recent versions are relatively selective for beta-1 receptors, although some beta-2 binding occurs, particularly with higher doses. These sympathetic nervous system blocking medications, or sympatholytics, have several uses, including the treatment of arrhythmias, hypertension, acute myocardial infarctions, and congestive heart failure. In the event the medication causes an excessive

blockade, the patient may experience bradycardia, heart failure, hypotension, and atrioventricular blocks. Especially with the use of nonselective beta-blocking medications, bronchoconstriction and hypoglycemia may occur. Propanolol is a common nonselective beta-blocking medication.

Metoprolol (Lopressor) is a selective beta-1 blocking drug that decreases the automaticity of the heart tissue. This effectively decreases cardiac output and systemic blood pressure. Metoprolol should be given at a dose of 5 mg IV every 2 to 5 minutes for three doses. The medication should then be titrated to achieve the desired effect of a heart rate between 60 and 90 beats/min and a safe blood pressure level. Metoprolol should not be used for patients with severe congestive heart failure, bradycardia, or cardiogenic shock. In addition to close monitoring of the patient's pulse rate, blood pressure, and respirations, particular attention should be given to the blood glucose level because metoprolol may reduce signs and symptoms of acute hypoglycemia. Furthermore, patients withdrawing from this medication may experience severe thyroid complications.

When a patient with supraventricular tachycardia, A-fib/A-flutter, or hypertension needs a shorter-acting beta-blocking medication, esmolol (Brevibloc) may be an appropriate choice. The effects of esmolol last approximately 10 to 30 minutes (except following cumulative doses), which makes adjustments easier when compared with longer-acting medications. Esmolol should be given with a loading dose of 250 to 500 μg/kg over 1 to 3 minutes. The patient should then be administered 50 μg/kg/min over 4 minutes. The infusion is either maintained at 50 μg/kg/min or increased by 50 μg/kg/min every 4 minutes (or longer) until the desired effect is achieved, to a maximum esmolol infusion rate of 200 μg/kg/min. Patients with atrioventricular blocks and uncompensated congestive heart failure should not receive this medication. There are many medication interactions that the provider must consider when administering this medication, including nonopiate pain medications and most other cardioactive medications. Extravasation can cause tissue necrosis.

Class III Antiarrhythmic Medications

The third class of antiarrhythmic medications is composed of the potassium channel blockers. Potassium channels open immediately after cardiac cell depolarization and allow for repolarization to occur. Potassium channel–blocking medications work by blocking these potassium channels, thereby delaying the phase 3 repolarization of the cardiac cells. When this occurs, we find an increase in the action potential duration and the effective refractory period. Because the medication increases the refractory period, we tend to see prolonged QT intervals on the patient's electrocardiogram (ECG). This effect makes potassium channel–blocking medications particularly useful for treating re-entry tachycardias and arrhythmias occurring as a result of cellular depolarization due to excessive excitability.

Amiodarone (Cordarone) is the most common class III antiarrhythmic medication, even though it also has properties that are consistent with other categories. As with the other antiarrhythmic medications, the class III medications can be proarrhythmic as well as antiarrhythmic. Amiodarone can precipitate torsade de pointes, atrioventricular (AV) blocks, and bradycardia, which make it contraindicated in the presence of bradycardia or AV blocks. The initial dose for patients in cardiac arrest is 300 mg IV rapid by push (if no pulse is present) followed by a repeat dose of 150 mg IV. Patients who are not in cardiac arrest may benefit from an initial dose of 150 mg IV given over 10 minutes. Numerous other dosing patterns are used for different indications. The onset of action is variable, and peak effects may not occur for days or weeks. Consult a reliable medication reference source for dosing in other (noncardiac arrest) situations.

Class IV Antiarrhythmic Medications/Calcium Channel–Blocker Medications

To conclude the Vaughan-Williams classifications, the class IV antiarrhythmic medications consist of the calcium channel blockers. Calcium channel–blocking medications bind to the L-type calcium channels in the cardiac and vascular smooth muscle cells. L-type calcium channels play a primary role in cardiac

nodal automaticity and phase 0 of the action potential. In other words, the L-type calcium channels influence the impulse generating and depolarizing activities of the sinoatrial and atrioventricular nodes. By blocking the calcium channels, these medications cause vasodilation, negative inotropy, negative chronotropy, and negative dromotropy (decreased electrical conduction velocity). Although calcium channel–blocking medications decrease the automaticity of aberrant pacemaker sites, the greatest influence comes in their ability to decrease the conduction velocity through the AV node. This factor makes class IV antiarrhythmic medications useful for tachyarrhythmias originating in the atria.

Currently, there are only two calcium channel–blocking medications used for treating arrhythmias: verapamil (Calan) and diltiazem (Cardizem). Each medication is effective in reducing atrial tachycardias, but diltiazem has been shown to have less risk of hypotension than verapamil. Verapamil is typically given at a dose of 2.5 to 10 mg IV over 2 minutes. The dose can be repeated at 5 to 10 mg after 15 to 30 minutes. Diltiazem is given at a dose of 0.25 mg/kg IV over 2 minutes. Neither medication should be given to patients who have preexisting bradycardias, Wolff-Parkinson-White syndrome, or heart failure. Additionally, patients taking a beta-blocking medication may experience a potentiation effect and should not receive calcium channel blockers.

Adenosine

Adenosine is a different type of antiarrhythmic. It is classified as an endogenous nucleoside, meaning it is a naturally occurring protein that is a substantial part of cellular metabolism. This medication works to interrupt re-entry pathways through the AV node. The AV nodal conduction is slowed, and the AV nodal refractory period is prolonged. In doing so, it can restore a sinus rhythm in patients who have paroxysmal supraventricular tachycardia. Adenosine is not effective in converting atrial fibrillation, but may be used as a diagnostic tool when differentiation between atrial fibrillation and supraventricular tachycardia cannot be made because of the rate. Because adenosine is quickly absorbed by vascular endothelial cells, it has an exceptionally short duration of action of around 12 seconds. It must be given as a rapid IV bolus followed by a rapid IV bolus of at least 10 mL of normal saline through an IV site no more distal than the antecubital fossa. Once adenosine is administered at an initial dose of 6 mg, you can expect the patient to exhibit a brief period of profound bradycardia or even asystole. This pause, although disconcerting, is brief and usually well tolerated. Communicating with the patient and other bystanders to anticipate this occurrence will mitigate much of the stress. If successful, this period should be followed by a sinus rhythm. Unsuccessful conversion will result in the heart rate returning to near the original rate, at which time a second and third dose of 12 mg IV can be administered. The side effects of the medication include flushing, chest pressure, and light-headedness. Adenosine should be avoided or used with extreme caution in patients taking dipyridamole (Persantine) and carbamazepine (Tegratol) because of likely potentiation effects. Adenosine can be administered for paroxysmal supraventricular tachycardia associated with Wolff-Parkinson-White syndrome, but CCTPs should have a cardioverter or defibrillator immediately available if the patient's condition or rhythm suddenly deteriorates.

Anticholinergic Medications

Anticholinergic medications have a wide variety of clinical uses. Diphenhydramine, a potent antihistamine, and various histamine-blockers are discussed later. Other anticholinergic medications are administered therapeutically to treat nausea and vomiting, excessive secretions from a variety of organs, abdominal cramps, and excess vagal tone leading to bradycardia or asystole.

Atropine is classified as an anticholinergic medication because it blocks the ACh receptors in the parasympathetic nervous system. Under normal circumstances, the parasympathetic nervous system causes the heart rate to slow following activation of these receptors. Atropine antagonizes these receptors to allow the heart rate to increase in response to endogenous sympathetic stimulation already present and

available within the body. When endogenous catecholamines are depleted or when bradycardia is not a result of increased parasympathetic stimulation, atropine is not likely to be very effective. Atropine, administered at a dose of 0.5 to 1 mg, with a maximum dose of 0.4 mg/kg, is the best treatment option for patients who are not experiencing myocardial ischemia or acute hemorrhage. The drug can be expected to begin working immediately, with a duration of action of 2 hours. Patients who receive atropine must be monitored for dizziness, palpitation, GI upset, arrhythmias, anticholinergic effects (blurred vision, dry mouth, and urinary retention), and paradoxical bradycardia when the medication is administered at low doses or too slowly. Atropine remains the medication of choice because it has predominantly chronotropic effects and little to no inotropic effects. CCTPs should be aware that atropine is not likely to be effective for increasing the heart rate in patients who have second- and third-degree heart blocks. In second- and third-degree heart blocks, CCTPs should anticipate the need for cardiac pacing instead of using atropine or when atropine is determined to be ineffective. Many RSI protocols include atropine as a prophylactic medication to prevent bradycardia from succinylcholine administration and airway manipulation, especially in pediatric patients.

Atropine, in addition to its use as the treatment for bradycardia and asystole, is essential for the emergent treatment of acetylcholinesterase inhibitor toxicity following exposure to certain pesticides and chemical nerve agents. A patient with this condition may require hundreds of milligrams of atropine. Tachycardia is not a contraindication for additional atropine in this situation.

Catecholamines and Sympathomimetic Agents

Dopamine, epinephrine, and norepinephrine are substances produced within the body to activate the sympathetic nervous system, which in turn initiates the fight or flight response from body cells, tissues, organs, and systems. These substances stimulate alpha- and beta-adrenergic receptors, resulting in many changes in the body. In healthy people, blood vessels constrict, cardiac output increases, blood coagulation increases, skeletal muscles strengthen, and large airways increase in diameter along with a myriad of smaller changes designed to improve the performance and efficiency of the human body. Collectively, dopamine, epinephrine, and norepinephrine are referred to as sympathomimetics or catecholamines based on their chemical structure. Each endogenous chemical described has been replicated pharmacologically for use in critical care and during patient transport.

Dopamine is available commercially for use as an IV infusion. In its natural endogenous form, it is a precursor to both norepinephrine and epinephrine within the body. Dopamine has positive inotropic effects by stimulating beta-1 receptors. In addition, dopamine stimulates alpha-1 receptors when given at a higher dose, which causes peripheral vasoconstriction. Dopamine also affects dopaminergic receptors. Dopaminergic receptors are found in the CNS as well as the pulmonary artery and atrias of the heart. Stimulation of the dopaminergic receptors increases myocardial contractility and cardiac output. The wide range of effects found with dopamine allow for a variety of uses. Patients may receive dopamine at a low dose in order to achieve a renal effect, up to 3 μg/kg/min, although the benefits remain undocumented. The intent is to increase the renal output and decrease fluid volume in the body. Slightly higher doses, between 3 and 5 μg/kg/min, will optimize the beta effects to increase cardiac output. Still higher doses of 5 to 20 μg/kg/min will provide the alpha effects to increase and maintain blood pressure. Patients may exhibit adverse reactions, including cardiac arrhythmias, hypertension, and increased myocardial oxygen demand. Dopamine typically has a peak effect around 5 to 10 minutes, and the effect terminates moments after the infusion is discontinued. In the event extravasation occurs, dopamine may cause tissue necrosis.

Epinephrine is another sympathomimetic with widespread effects. It can be used to support respiratory function by opening up larger airways. It augments blood pressure by increasing vascular tone. Epinephrine has chronotropic effects, but has even more inotropic effects on the heart. During cardiac

arrest resuscitation efforts, it is given at a dose of 1 mg IV every 3 to 5 minutes to enhance myocardial perfusion. When given intravenously, the onset can be expected within a few moments, with the duration lasting less than 10 minutes. Patients may exhibit hypertension, arrhythmias, pulmonary edema, and anxiety with administration. In various clinical situations, epinephrine is given as an IV bolus, an IV infusion, inhaled, nebulized, administered IM or SC, or administered via an endotracheal tube. Consult a reliable reference for dosing instructions when epinephrine is indicated.

For patients in cardiac arrest, the first or second dose of epinephrine can be replaced with vasopressin. Vasopressin is a naturally occurring hormone referred to as an antidiuretic hormone. The medication, when given at high doses, up to 40 units IV, has significant peripheral vasoconstrictive effects. It also has a much longer duration of action; therefore, no repeat dose is necessary and epinephrine should not be resumed for 10 to 15 minutes. Some evidence shows that vasopressin may also cause coronary artery constriction; therefore, caution should be exercised when using the medication in patients with profound cardiovascular disease. Vasopressin is an additional option, although not FDA approved, for patients in severe shock resulting from vasodilatation (such as sepsis). A vasopressin infusion is initiated at 0.0005 U/kg/h and titrated to clinical effect. Vasopressin is also administered for the treatment of diabetes insipidus and certain types of gastrointestinal hemorrhage.

Norepinephrine (Levophed) is another sympathomimetic that has significant beta-1 and alpha-1 agonist effects. It also is most commonly used for blood pressure control, although it should not be used for patients who are hypotensive as the result of acute hemorrhage. When norepinephrine is administered, patients may exhibit arrhythmias, tachycardia, hypertension, and decreased peripheral blood flow. When the drug is administered at an infusion rate of 0.5 to 1.0 μg/min, the peak effects can be seen after about 3 minutes and last less than 1 minute after the infusion is discontinued. The maximum safe dose of norepinephrine is 20 μg/min, although some tertiary care centers titrate infusions up to 40 μg/min.

Phenylephrine (Neo-Synephrine) is another treatment option for increasing a patient's blood pressure. The medication is classified as an alpha-agonist and causes vasoconstrictive effects by binding to, and stimulating, the alpha receptors in the vascular smooth muscle. By definition, phenylephrine is a sympathomimetic, but not a catecholamine, because of its pure alpha-agonist activity. Caution must be exercised because the vasoconstriction includes coronary arteries. Patients with impaired coronary circulation may experience myocardial ischemia. Phenylephrine is typically given as an infusion at a rate of 40 to 60 μg/min. It may be necessary to start the infusion at 100 to 180 μg/min until the blood pressure increases. Phenylephrine is ideal for situations in which vasomotor tone is lost, without simultaneous impairment of myocardial contractility.

Dobutamine (Dobutrex) is a frequently used sympathomimetic in critical care transport. Dobutamine stimulates beta-1 receptors to increase myocardial contractility. The major difference between dobutamine and dopamine or norepinephrine is that dobutamine has minimal chronotropic effects when compared with the other two medications. Peak effect is reached after 10 minutes, and the effects last for about 2 minutes after the infusion of 2 to 20 μg/kg/min is discontinued. Caution must be exercised because dobutamine may cause an increase in infarction size in patients experiencing an acute myocardial infarction. It may also precipitate cardiac arrhythmias, hypertension, and premature ventricular complexes. Dobutamine is known for worsening an already tenuous blood pressure. Consider starting treatment with dopamine to stabilize the patient's blood pressure before administering dobutamine to improve cardiac output in patients who require inotropic support.

Direct Vasodilator Medications

Hydralazine (Apresoline) acts directly on arterioles to produce vasodilation and lower blood pressure. It appears to be safe for use as an emergent antihypertensive in pregnant patients with preeclampsia. Additional uses include congestive heart failure, primary pulmonary hypertension, and malignant

hypertension. Numerous reported side effects include angina, reflex hypertension, and tachycardia. Blood pressure reduction begins in 5 to 20 minutes following IV administration and persists up to 4 hours. The initial dose for adults with hypertension is a 10-mg slow IV push reduced to a 5-mg slow IV push in pregnant patients.

Diuretic Medications

Diuretic medications can remove excess fluid from the circulating blood volume, alter the distribution of fluids within the body, enhance urinary elimination of harmful substances, and increase (or maintain) adequate flow through jeopardized kidneys. Treatment with diuretic medications is desirable in situations such as fluid volume overload, cerebral edema, hyperkalemia, and rhabdomyolysis.

The most common diuretic is furosemide (Lasix). Furosemide is a loop diuretic, which means it carries out its functions on the loop of Henle as well as the proximal and distal convoluted tubule. Furosemide inhibits the reabsorption of sodium and potassium salts, which in turn remove (excess) water. There is also evidence that furosemide produces renal vasodilation, which theoretically would improve hemodynamics in patients with fluid overload, although similar research disputes this claim. Several modifications to the dosing recommendations for furosemide have occurred over the past several years; doses may range from a 40- to 120-mg IV bolus and may include a constant infusion. Caution needs to be exercised because of the possibility of dehydration, hypotension, severe electrolyte imbalances, and alkalosis. The IV onset of action occurs in approximately 5 minutes, and the effects typically last about 2 hours. Recent recommendations for the treatment of acute heart failure de-emphasize the use of diuresis, noting that up to 40% of patients with acute heart failure are volume depleted. The relatively rapid effects of loop diuretics do not appear evident in critically ill patients; the effects of furosemide may not be seen for up to 2 hours after administration. Ultimately, routine use of loop diuretics in acute heart failure results in a longer hospital stay and increased mortality.

Some patients may receive a different diuretic called bumetanide (Bumex). Bumetanide is a loop diuretic that works similarly to furosemide. It should be given 1 to 2 mg IV over 2 minutes and repeated in 2 to 3 hours to achieve effect. The dose of bumetanide must be individualized in accordance with an FDA warning regarding excessive electrolyte and fluid depletion. Patients with preexisting electrolyte imbalances should not receive bumetanide. Most of the side effects are related to the electrolyte and fluid imbalances.

Mannitol is a common treatment choice for increased ICP in patients who have experienced a traumatic brain injury. Mannitol is an osmotic diuretic and is used because it pulls water out of the brain tissue into the vasculature. From there, mannitol causes an increase in renal output to rid the body of excess fluid. This diuretic is also used for enhanced urinary elimination of certain toxins, treatment of anuria from acute renal failure, and as a genitourinary irrigant. Mannitol has a rapid onset and a duration of 2 to 6 hours. It is commonly administered at a dose of 0.25 to 2 g/kg every 4 to 6 hours. Caution should be exercised because this medication can cause significant changes in electrolyte levels, which can have negative effects. However, the effects are generally much less than those caused by loop diuretics such as furosemide.

Nitrates

A traditional first-line medication for ischemic heart disease is nitroglycerin. Nitroglycerin also treats hypertension. This nitrate works as a vascular smooth-muscle relaxant, thereby allowing for a decrease in preload on the heart and an increase in vascular space into which fluid can move. Nitroglycerin is primarily a venous dilating agent, with limited effects on arteries, including coronary arteries. Typically given at a standard dose of 0.4 mg sublingually during the initial phase of treatment, some small-scale studies have shown potential benefit in doubling that dose in acute cases that involve pulmonary edema.

Beyond the initial phase of treatment, a nitroglycerin infusion can be used to maintain the decreased preload and vasodilation, often at a dose of 5 to 200 µg/min. Adverse reactions include headache, hypotension, dizziness, and reflexive tachycardia.

Sodium nitroprusside is a nitrate medication. Nitroprusside is not as commonly used to control blood pressure as other hypertensive medications, as a result of the increased chronotropic effects on the heart, but it can be effective at maintaining vasodilation. Nitroprusside is an arterial and venous dilator, which makes it more potent than nitroglycerin. Patients receiving this medication at an infusion rate of 0.5 to 10 µg/kg/m in require close monitoring because it can cause a precipitous decrease in blood pressure. The adverse reactions are similar to nitroglycerin but also include muscle twitching, acidosis, and cyanide-type poisoning in cases of accumulation after prolonged infusions.

Phosphodiesterase Inhibitors (Inodilators)

Milrinone (Primacor) can be used by CCTPs to improve cardiovascular performance. It is classified as a phosphodiesterase enzyme inhibitor, which means that it inhibits the cyclic adenosine monophosphate (cAMP) phosphodiesterase. This medication increases cardiac output by increasing the inotropic effect (cardiac contractility). Increased output occurs without increasing the heart rate or chronotropic effect. The medication simultaneously dilates blood vessels, which is helpful in patients with heart failure. Milrinone is considered to be a safer formulation than inamrinone, with less tendency to cause bone marrow or liver toxicity. Each can precipitate arrhythmias. Milrinone is given with a loading dose of 50 µg/kg over 10 minutes followed by a maintenance dose of 0.375 to 0.75 µg/kg/min. There is no evidence to support the use of milrinone for more than 48 hours, although patients with endstage heart failure may be maintained on long-term continuous infusions of milrinone. Ventricular arrhythmias, including ventricular fibrillation, are possible side effects in addition to hypotension and supraventricular arrhythmias. Serum potassium levels should be monitored in patients receiving this medication.

Anticoagulants, Fibrinolytics/Thrombolytics, and Blood Components

CCTPs will encounter situations that require dissolution of blood clots with thrombolytics, prevention of blood clots with anticoagulants, or expansion of the circulating blood volume with crystalloid and colloid medications. Each medication has the potential for significant harm from improper administration or inadequate monitoring. It is essential that CCTPs consider atypical causes of clinical signs and symptoms before administering anticoagulant or thrombolytic medications. Aneurysms and hemorrhagic events frequently mimic the clinical presentation of thrombus and embolus occlusion of the same organ. Aneurysms can cause chest discomfort and changes on an ECG. A cerebral hemorrhage can mimic a cerebral thrombus or embolus. Anticoagulant or thrombolytic medication use in these patients would likely be fatal.

Anticoagulant Medications

In many instances, it is necessary to prevent the coagulation of blood and the subsequent development of blood clots. The use of anticoagulant medications is common in the transport of critical patients. Aspirin is considered to be a first-line anticoagulant medication and is technically considered an antiplatelet medication. Aspirin works by inhibiting the enzyme cyclooxygenase. Cyclooxygenase is responsible for producing thromboxane A2, which activates platelets. By inhibiting cyclooxygenase, platelets are unable to bind and form a clot. Aspirin does not dissolve a clot that is already present, however, but will aid in the prevention of clot expansion. Every patient being transported for acute coronary syndrome or myocardial infarction should be considered for aspirin administration if the patient is not already receiving the medication.

Special Populations

Older trauma patients who have received anticoagulation medication need special attention during critical care transport. These patients are at increased risk for serious bleeding if they are inadvertently injured from being dropped, falling, or becoming involved in a vehicle crash. Use adequate patient restraint systems and care when moving the patient from one stretcher or bed to another.

Patients who have been administered too much of an anticoagulant medication should not receive rapid reversal if levels become elevated. Unusual bleeding or bruising and elevated coagulation studies on laboratory test results indicate excessive amounts of anticoagulant medications. It is essential to consider the risks of over-anticoagulation compared with the risk of excessive reversal. In many cases, the risk of blood clot formation exceeds the likelihood of spontaneous bleeding.

Medication options are available for high-risk patients who are unable to receive aspirin. Ticlopidine (Ticlid) and clopidogrel (Plavix) reduce platelet aggregation by blocking APD receptors on platelets without affecting prostaglandin metabolism as aspirin does. Ticlopidine has considerably more significant side effects than clopidogrel and appears to be losing favor among clinicians.

Clopidogrel has also demonstrated significant mortality benefits in patients with myocardial infarctions and is often given in conjunction with aspirin therapy. Depending on the situation, clopidogrel is given either 75 or 300 mg orally. Antiplatelet effects begin in about 2 hours and persist for 3 to 7 days. Gastrointestinal side effects predominate, but numerous other CNS, cardiovascular, and hematologic side effects are possible.

Emerging IV antiplatelet medications are becoming increasingly popular in the treatment of cardiac conditions and critical care transport. Glycoprotein IIB-IIIA inhibitors further undermine platelet activity in patients with acute coronary syndrome, especially prior to revascularization procedures. They disrupt the final common pathway for platelet aggregation. At the risk of oversimplifying their role and mechanism, it may be helpful to consider IIB-IIIA inhibitors as "super-aspirins." Abiximab (Reopro), eptifibatide (Integrilin), and tirofiban (Aggrastat) are each administered via IV infusion after an IV loading dose. As with any antiplatelet medication, a careful risk-benefit analysis of bleeding and other risks is essential prior to administration. Bleeding is the most common or obvious concern with these medications. Hypotension, bradycardia, and mild CNS side effects occur with varied frequency in these three medications. Consult a reliable medication reference source of individual dosing and administration information.

Heparin is another common anticoagulant medication. Heparin works by inactivating several blood factors to prevent fibrin formation. It also inactivates thrombin to prevent activation of more blood factors, giving it both anticoagulant and antithrombotic properties. Heparin is infused at a rate based on U/kg/h, depending on disease process and treatment goals. Most institutions administer a loading dose prior to infusion, but CCTPs will occasionally encounter situations in which the maintenance infusion is given without a loading-dose bolus. There are drawbacks to heparin administration, including the need for frequent determinations of partial thromboplastin time (PTT) or heparin level, as well as the lack of available oral form. CCTPs should review the latest lab values and the PTT prior to transport. Great care must be exercised when monitoring the patient for bleeding problems because controlling such hemorrhage is extremely difficult and life threatening. Many health care institutions require that at least two clinicians confirm the order and dose before heparin is administered to a patient. Heparin is also used SC as deep vein thrombosis prophylaxis in immobile, hospitalized patients. Many long-term indwelling vascular devices are flushed with heparin when they are not in use, as are short-term, temporary hemodialysis catheters. Be careful not to inadvertently flush large quantities of heparin into a

patient when accessing these indwelling catheters. Changes in a patient's neurologic status during transport create the concern for spontaneous intracranial hemorrhage and are an indication to discontinue heparin and any other anticoagulant infusions during transport.

Heparin has serious additional side effects. Heparin-induced thrombocytopenia (decreased platelet count)/thrombosis (blood clot), also known as HIT/T, is a syndrome with potentially lethal consequences for patients receiving heparin therapy. The mortality rate exceeds 30% in unrecognized or untreated patients. HIT/T syndrome can be distinguished from nonimmune-mediated (mild) thrombocytopenia because HIT/T appears between 4 and 14 days after beginning heparin therapy. Nonimmune-mediated thrombocytopenia appears within 4 days of starting heparin therapy. Twenty percent of thromboses associated with HIT/T occur in arteries, resulting in acute limb ischemia, cerebrovascular occlusion, and myocardial infarction. A thrombosis also frequently occurs as an extension or increase in the size of the original thrombosis that was being treated by heparin, significantly complicating the clinical situation.

HIT/T should be suspected whenever the platelet count decreases by more than 50% following heparin therapy. The diagnosis is confirmed by specialty laboratory tests, but the platelet decrease is highly suggestive of HIT/T, especially in the absence of other potential causes. HIT/T can occur with low-dose heparin administered SC and enoxaparin therapy. Heparin (or enoxaparin) administration should be immediately discontinued when HIT/T is suspected. The treatment for HIT/T includes the use of lepirudin (Refludan) and argatroban alternate anticoagulants and is best monitored by clinicians experienced in managing this syndrome.

Enoxaparin (Lovenox) is a low-molecular-weight heparin and can be used as an adjunct to thrombolytic medications in patients experiencing myocardial infarctions. It can also be used for patients with unstable angina and several other conditions. Similar to the standard heparin, enoxaparin inhibits various blood factors that form fibrin as well as increase antithrombin activity. Enoxaparin is administered at a dose of 30 mg IV bolus followed by 1 mg/kg SC, not to exceed 100 mg per dose. Twelve hours later, a second dose of 1 mg/kg can be given SC. Alternate dosing patterns are also available for various clinical situations. As with other forms of anticoagulant medications, it should not be administered to patients with bleeding problems or thrombocytopenia. There is no need to monitor PT or PTT values in these patients.

Warfarin (Coumadin) is an additional anticoagulant medication. Warfarin works by inhibiting the creation of vitamin K–dependent coagulation factors in the liver. A single dose can have effects that last as many as 5 to 7 days. Given typically at a dose between 2 and 10 mg (sometimes higher), warfarin carries significant and potentially fatal bleeding risks. It should not be administered to patients with bleeding tendencies, and a cumulative effect can occur in patients taking other anticoagulant, antiplatelet, and thrombolytic medications. Warfarin levels are monitored by obtaining an international normalized ratio, which involves a lab test result that reports the time it takes for blood to clot and compares it to the average time, and PT measurements. Medication doses and administration patterns are continuously adjusted to a target range for either of these values.

CCTPs frequently encounter patients on warfarin who have experienced devastating effects from inadequate monitoring and dose adjustments. It is not uncommon for a cerebral hemorrhage to occur when the anticoagulant effects of warfarin are potentiated by numerous medications, chemicals, and nutritional products. In severe over-anticoagulation situations, phytonadione (vitamin K, aquaMEPHYTON) can be given to counteract some of the anticoagulant effects. This situation should be approached cautiously. The reversal of anticoagulation can be equally as dangerous as over-anticoagulation in high-risk patients.

Thrombolytic or Fibrinolytic Medications

Thrombolytic medications differ from anticoagulants in that these medications break down an existing clot rather than prevent propagation. Thrombolytics convert plasminogen to plasmin. Plasmin is a natural

fibrinolytic that breaks down the fibrinogen and fibrin that compose clots. These medications should not be administered to patients who have undergone recent surgery or experienced problems with intracranial hemorrhage. Relative contraindications include hypertension, GI hemorrhage, and recent trauma.

Alteplase, also referred to as tissue plasminogen activator, or tPA, is identical to natural human plasminogen activator, which is used by the body to prevent excessive thrombus development. Alteplase is commonly used to break down clots that caused acute myocardial infarctions and acute ischemic cerebral vascular accidents. Patients who should not be given alteplase include those who have had recent strokes, intracranial or other internal hemorrhage, and severe hypertension. The typical dosing schedule is 15 mg as an IV bolus followed by an infusion of 0.75 mg/kg over 30 minutes without exceeding 50 mg. That schedule is followed by a maintenance infusion of 0.5 mg/kg over 60 minutes without exceeding 35 mg. If the patient weighs more than 65 kg, the dose changes slightly. The initial loading dose remains 15 mg, but the following infusion is given at 50 mg over 30 minutes, which is subsequently followed by 35 mg over the next 60 minutes. The total overall administration of all three doses should not exceed 100 mg. Alteplase has a short half-life of only 5 minutes, which often necessitates the concurrent administration of heparin to prevent the recurrence of the thrombus.

A close relative to alteplase, retaplase (Retavase) works similarly by producing plasmin. Retaplase, however, has been shown to diffuse throughout the clot with greater efficiency than alteplase and also has a half-life that does not necessitate infusion. The medication is given at a dose of 10 units IV over 2 minutes and can be repeated after 30 minutes if needed. The side effects and contraindications are the same as those found with alteplase. It is common for the second dose of retaplase to be administered during critical care transport, as the patient is en route to a tertiary cardiac care center. Signs of serious bleeding require withholding the second dose of retaplase.

Tenecteplase (TNKase) is a single-dose, weight-based thrombolytic that CCTPs are also likely to encounter. The mechanism and side effects are similar to the other thrombolytics previously discussed. Tenecteplase is given in combination with aspirin and heparin.

Streptokinase, another thrombolytic medication option, does not activate plasminogen. Instead, it binds to existing plasminogen to create additional plasmin. Streptokinase is derived from streptococci and carries some significant untoward effects, such as fever. It has a half-life of about 20 minutes and cannot be safely administered more than once in a 6-hour period as a result of the high antigenic properties. The dose ranges from 250,000 to 1.5 million units over 30 to 60 minutes, depending on the thrombus being treated. The untoward effects often necessitate discontinuation prior to therapeutic levels being reached. This medication appears to be less popular with clinicians, so its use in critical care transport is limited.

Blood Volume–Expanding Solutions

When intravascular volume is lost, it is necessary to replace the lost blood and other plasma fluids in order to maintain adequate circulation and perfusion to vital organs. As of the writing of this text, prehospital use of blood products and blood substitute products is still in the investigational stage and interfacility products is not yet commonplace. CCTPs must rely on other fluids to replace missing blood volume. There are predominantly two options for fluid resuscitation: crystalloid solutions and colloid solutions.

Crystalloid Solutions

Crystalloid solutions remain the most common choice for fluid replacement. A crystalloid is defined as a substance that forms a true solution and passes freely through a semipermeable membrane. In other words, the concentration of water and electrolytes is consistent with that of typical blood concentrations. There are three types of crystalloid solutions: isotonic, hypertonic, and hypotonic solutions. The categories are

based on the fluid's tonicity, or the amount of solutes dissolved in the fluid.

Isotonic solutions have the same tonicity as human blood. As such, the osmotic pressure does not cause fluid or electrolytes to shift into or out of the intravascular space. Instead, the solution will diffuse equally throughout intravascular and extracellular spaces. Isotonic solutions are advantageous in that they have been found to be safe because they tend not to create pressure and concentration gradients between the various fluid compartments within the body. Additionally, they are inexpensive and readily available in virtually every acute health care setting. As long as an isotonic solution is applied within the appropriate parameters, it can produce numerous beneficial effects for patients, including replacement of lost circulating intravascular volume, which increases tissue perfusion, promoting renal excretion of waste products and toxins; and replacement of cellular and interstitial fluids, which promotes cell, tissue, and organ functioning. The body requires two to three times more isotonic solutions when compared with colloid solutions to achieve similar volume expansion. Because the osmotic pressure is equal, crystalloid solutions easily transition into the interstitial spaces, which will cause pulmonary edema in the case of overadministration or heart failure. Normal saline and lactated Ringer's solution are the most common fluids used in this category. Providers of different medical specialties tend to have a particular preference to one of these solutions over the other.

Hypotonic solutions have lower concentrations of electrolytes when compared to the human body. As a result of the lower concentrations, the osmotic pressure moves fluid out of the intravascular space and into the intracellular space. These fluids, such as 0.45% sodium chloride solution, are not useful for hypovolemia because they will cause further collapse of the cardiovascular system. These solutions are usually only used in select situations and require careful monitoring for unintended alterations of body fluid distribution. Hyponatremia, cerebral edema, and death are possible with inappropriate administration of hypotonic IV solutions. CCTPs should note that 5% dextrose in water becomes hypotonic when the dextrose component is consumed by body tissues.

Hypertonic solutions have higher concentrations of electrolytes when compared to blood. When the solutions are administered, the osmotic pressure causes fluid to move out of the intracellular and extravascular spaces and into the intravascular space. Sodium chloride solutions, 3% and 5%, are included in the hypertonic solution category, but the use is limited because of the decreased intracellular fluid volume and inconclusive evidence regarding benefit vs mortality rates. Hypertonic saline is being investigated for numerous purposes, including sepsis management, trauma resuscitation, and treatment of tricyclic antidepressant overdoses. The safety and efficacy in these situations is still being evaluated.

Colloid Solutions

Colloid solutions contain a higher concentration of dissolved electrolytes than do typical blood concentrations. The resulting increased osmotic pressure causes interstitial fluid to be drawn into the intravascular space, thereby increasing the net fluid volume beyond the actual amount administered. This can be particularly problematic in the presence of increased capillary permeability. With this condition, the electrolytes can pass into the interstitial space and draw fluid out of the intravascular space. Life-threatening conditions occur when the permeability increases across the alveolar capillary membranes.

The debate continues as to whether colloid solutions are beneficial, indifferent, or harmful. Several studies have been conducted on the topic, and all three options have been demonstrated at one point or another. Most of the current literature discourages the use of colloids, citing an increase in mortality when these solutions are used.

Blood Products

Albumin falls into another classification of volume-expanding fluids: blood products.

Albumin is a colloid solution derived from human blood donations, processed to prevent infection

and then used for hypovolemia, burns, and certain surgical procedures. Albumin increases intravascular oncotic pressure, drawing extravascular fluid into the blood vessels. The role of albumin in many critical illnesses remains heavily debated. When vascular permeability is increased with certain critical illnesses, albumin has a tendency to leach out of blood vessels into interstitial spaces. The direction of oncotic pressure is reversed, drawing intravascular volume into interstitial spaces, dramatically complicating the clinical situation. In patients who are otherwise well hydrated, albumin is typically administered in a 25% solution. In patients with poor hydration, the concentration should be diluted down to 5% because the albumin will draw fluid from the interstitial spaces. For cases of hypovolemia, adults are commonly given 100 to 200 mL of the 25% solution with the option to repeat the administration in 10 to 15 minutes if needed. The dose administered to burn patients is based on their current hemodynamic status. Side effects are rare, but may include urticaria and nausea. Profound anemia and heart failure are contraindications to albumin administration. The use of sterile water as a diluent can cause fatal renal failure or hemolysis. Acceptable diluents include normal saline and 5% dextrose in water.

Hetastarch is a synthetic colloid solution that is designed to expand the plasma volume. A variety of electrolytes are contained in hetastarch, which in combination retain fluid in the vasculature to substitute depleted blood plasma. When using hetastarch, consideration must be given to the possibility of pulmonary edema, fluid overload, and congestive heart failure. Adult patients typically receive 500 to 1,000 mL based on the hemodynamic state. Pediatric patients should not be given hetastarch. Hetastarch should also be avoided in individuals with allergies to corn or corn products.

Dextran is another volume expander that CCTPs may encounter. It is used in situations when blood products are not immediately available or to prime the tubing of various external medical devices that sequester large amounts of blood. Initial dextran therapy should not exceed 20 mL/kg during the first day, with decreased dosing on subsequent days. Infusion should not exceed 20 to 40 mL/min in adults. An infusion pump is required for dextran administration.

Cardiovascular medications used in critical care transport are summarized in **Table 7-5**.

Medications Used for Neurologic Conditions

Neurologic illnesses, diseases, and conditions are commonly seen during critical care transport. Severity can range from mild anxiety or discomfort to devastating situations such as paralysis, brain injury, intractable seizures, or full brainstem herniation. Neurologic conditions can present following trauma, a myriad of illnesses, or toxic or environmental exposures, or as a consequence of interventions performed before or during critical care transport. Analgesia, anxiolysis, sedation, and seizure control are the main purposes of neurologic medications administered by CCTPs. Various medications with these properties are discussed in other sections of this chapter as well. Benzodiazepines, discussed in the airway medication section, are extremely versatile, treating anxiety, controlling seizures, and providing sedation in a vast array of clinical situations. Propofol, also discussed in the airway section, has potent sedative and anticonvulsant properties. Mannitol, a powerful diuretic medication, is often used to treat cerebral edema and may be a last alternative to save a patient from impending brainstem herniation. Magnesium sulfate, discussed later in this chapter, is effective in controlling seizures in preeclamptic and eclamptic obstetric patients. Medications commonly used in critical care transport to manage or prevent various neurologic conditions are also discussed.

Anesthetic, Antianxiety, Sedative, and Hypnotic Medications

Patient sedation may be warranted in a variety of situations. Several classifications of medications that provide sedation are available, whether the medication is being administered to patients experiencing anxiety or those requiring endotracheal intubation, and each has advantages and disadvantages. Benzodiazepines, which were discussed in the airway medication section, should be considered first-line

anxiolytic and sedative medications during critical care transport.

Haloperidol

Haloperidol (Haldol) is an antipsychotic medication that can sedate combative patients and those experiencing a psychotic episode. Although the exact mechanism of action remains unknown, it is believed that haloperidol works by selectively antagonizing dopamine D_2 receptors to produce a tranquilizing effect. Haloperidol generally has an onset of 10 minutes, with the peak effect usually occurring between 30 and 45 minutes, but possibly occurring later. This medication is not approved for IV administration, but is often used intravenously off label when emergent control of agitation is desired. Usual intramuscular dosing is 2 to 10 mg. The goal is to achieve sedation with the lowest possible dose because of the potential for a fatal condition known as neuroleptic malignant syndrome, which is discussed in Chapter 20. Additionally, providers should be prepared for the potential onset of extrapyramidal symptoms, which are involuntary muscle contractions of the face, neck, trunk, and/or extremities. Treatment for this condition follows individual agency guidelines, usually including intravenous benzotropine (Cogentin), diphenhydramine (Benadryl), or benzodiazepine medications. Respiratory and cardiac compromise is possible with haloperidol, especially with higher doses, and particularly with IV administration Also, an increased risk for prolongation of the QT interval exists, which may lead to torsade de pointes ventricular tachycardia.

Dexmedetomidine

Patients can be given a medication that antagonizes the alpha-2 adrenergic receptors. These neurotransmitters are inhibited, causing sedation for patients who are already intubated and ventilated. Dexmedetomidine (Precedex) is an alpha-2 agonist that may potentially be encountered by CCTPs. In addition to its use in intubated patients, dexmedetomidine has also been approved for sedation in nonintubated patients who undergo various invasive procedures. One benefit of dexmedetomidine is the 6-minute half-life, which may permit easier ventilator weaning or extubation. It is reported that this medication provides the sedative effects without the cardiovascular and respiratory compromise seen in other classes of sedatives. The typical loading dose is 1 μg/kg over 10 minutes followed by the maintenance dose of 0.2 to 0.7 μg/kg/h. Adverse reactions, although less frequent than other medications, include cardiovascular (hypotension and occasional bradycardia or atrial fibrillation) and potential respiratory system compromise. Infusions of dexmedetomidine should not be given for longer than 24 hours.

Anticonvulsant Medications

Although the exact mechanism behind various anticonvulsant medications is not completely clear, these drugs are believed to work by inhibiting the influx of sodium into cells. This halt of sodium transport decreases the ability of cells to depolarize and propagate the seizures. Several other types of drugs fall into the category of anticonvulsants based on the use and intended effect, including benzodiazepines, barbiturates, hydantoins, and valproic acid.

TABLE 7-5 Cardiovascular Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Alpha-adrenergic antagonists				
Hypertension, unwanted vasoconstriction from exogenous or endogenous catecholamines, pheochromocytoma	Block alpha receptors in vascular smooth muscle, preventing vasoconstriction from catecholamines	Clonidine: PO Phentolamine (Regitine): SC, IM, IV Labetolol (Trandate): PO, IV	Hypotension, tachycardia, dizziness, weakness, dry mouth, flushing, weakness	Phentolamine is infiltrated into subcutaneous tissues affected by extravasation of vasoconstrictor medications to prevent tissue necrosis.
ACE inhibitors				
Hypertension, left ventricular dysfunction, congestive heart failure	Prevent conversion of angiotensin I to angiotensin II, a potent vasoconstrictor; reduce aldosterone secretion	Captopril (Capoten): PO Enalapril (Vasotec): PO, IV	Hypotension, tachycardia, chest pain, palpitations, cough, hyperkalemia, angioedema	Numerous medication interactions occur. Be alert for ACE inhibitors causing sudden, unexpected angioedema, even in patients who have been previously taking ACE inhibitors.
Antiarrhythmic class I medications				
Treatment of various atrial or ventricular arrhythmias (different class I agents are appropriate for different types of arrhythmias)	Block fast sodium channels; inhibit depolarization of neuronal cells, decrease myocardial conduction velocity and automaticity	Procainamide: IV Lidocaine: IV, endotracheal tube	Hypotension, nausea, vomiting, unwanted conduction disturbances, cardiovascular collapse, numerous others	Correct any electrolyte disturbances. Avoid use in patients with any significant cardiac conduction abnormality without an appropriately functioning pacemaker.
Antiarrhythmic class II medications (beta-adrenergic blocking agents)				
Treatment of certain cardiac arrhythmias, hypertension, acute myocardial infarction, acute coronary syndrome; also used for tremors, certain psychological disorders, migraines	Competitively block beta receptors in heart, blood vessels; may affect other beta receptors at higher doses	Metoprolol (Lopressor): PO, IV Labetolol (Trandate): PO, IV Esmolol (Brevibloc): IV	Hypotension, bradycardia, conduction abnormalities, dizziness, hypoglycemia	Avoid use in patients with cocaine toxicity due to risk of unopposed alpha stimulation. Beta selectivity is lost following high doses or overdose. Avoid administration of IV calcium channel blockers with beta blockers within a short period of time.
Antiarrhythmic class III medications				
Various atrial or ventricular arrhythmias	Block potassium channels, which delay phase 3 repolarization and increase the effective refractory period	Amiodarone : PO, IV	QT interval prolongation, hypotension, bradycardia, other conduction abnormalities, flushing, edema, numerous cardiac arrhythmias, cardiovascular collapse, numerous additional side effects	Amiodarone affects sodium, potassium, and calcium channels along with alpha- and beta-blocking properties. Amiodarone has an exhaustive list of side effects and medication interactions.
Antiarrhythmic class IV medications (calcium channel blockers)				
Hypertension, certain types of tachycardia, migraines; also used for myocardial	Block L-type calcium channels especially in the SA and AV nodes;	Diltiazem (Cardizem): PO, IV Verapamil (Calan): PO, IV	Hypotension, bradycardia, conduction	Avoid administration of IV calcium channel blockers with beta-

hypertrophy, certain psychological disorders, and tocolysis	inhibit calcium influx into cells in arterial walls, decreasing systemic vascular resistance		disturbances, edema, flushing	blockers within a short period of time.
Antiarrhythmic medications				
Paroxysmal supraventricular tachycardia with or without Wolff-Parkinson-White syndrome	Replicate endogenous nucleoside, which slows or interrupts reentry pathways through the AV node	Adenosine (Adenocard): IV	Flushing, headache, dyspnea, hypotension, ECG changes, hyperventilation; prolonged asystole is possible	Administer rapidly through the largest proximal vein possible. Expect a significant sinus pause immediately following IV administration. Warn patient, family, or other bystanders of this event. Several significant medication interactions occur with adenosine.
Anticholinergic medications				
Bradycardia, asystole, acetylcholinesterase inhibitor toxicity may be given to prevent bradycardia in patients undergoing rapid sequence intubation; may also be used as a mucolytic agent in certain circumstances	Block acetylcholine receptors in the parasympathetic nervous system; counteract excessive vagal stimulation	Atropine: IM, IV, endotracheal tube	Arrhythmias, flushing, tachycardia, dry mucous membranes (may be beneficial), various CNS changes	Extreme quantities may be required for severe acetylcholinesterase inhibitor toxicity. Tachycardia is not an indication to withhold atropine in patients with acetylcholinesterase inhibitor toxicity.
Catecholamines and sympathomimetic agents				
Bronchospasm, airway edema, cardiac arrest, allergic or anaphylactic reactions, profound hypotension, profound bradycardia; certain medications may also be used when inotropic support or renal perfusion is needed	Mimic various effects and functions of the sympathetic nervous system through stimulation of alpha, beta-1, beta-2, and dopamine receptors in blood vessels, tissues, and organs	Dobutamine (Dobutrex): IV Dopamine (Inotropin): IV Ephedrine: IV Epinephrine (Adrenaline): Nebulized, SC, IM, IV, endotracheal tube Phenylephrine (Neo-Synephrine): IV Norepinephrine (Levophed): IV	Hypertension, tachycardia, worsening CHF, increased myocardial oxygen consumption, cardiovascular collapse, peripheral vasoconstriction/ischemia, anxiety, nervousness, restlessness (numerous others)	Continuous hemodynamic monitoring is essential for any of these medications. Administer via central venous access whenever possible; peripheral administration may cause tissue necrosis if the IV line infiltrates. Many vasoactive medications will worsen myocardial performance in compromised patients. Avoid using sodium bicarbonate in the same IV lines.
Direct vasodilator agents				
Hypertension, congestive heart failure, primary pulmonary hypertension	Cause direct dilation of arterial wall smooth muscles	Hydralazine (Apresoline): PO, IM, IV	Tachycardia, hypotension, rebound hypertension, vascular collapse, flushing, nausea, vomiting	Certain individuals may experience more profound hypotensive effects than others.
Diuretic medications				
Fluid volume overload, cerebral edema, various toxic exposures, hyperkalemia	Various depending on medication; may use either osmotic gradients or otherwise prevent	Furosemide (Lasix): PO, IM, IV Bumetanide (Bumex):	Hypovolemia, significant electrolyte abnormalities, dizziness, numerous	Closely monitor fluid volume and electrolyte status during and after administration. Do not

hyperkalemia, rhabdomyolysis	or otherwise prevent renal reabsorption of water, electrolytes, and waste products	PO, IM, IV Mannitol (Osmitrol): IV	dizziness, numerous additional abnormalities	administration. Do not use in patients who are physiologically unable to produce urine, such as those with severe chronic renal failure.
Nitrates				
Ischemic cardiac disease, hypertension; useful in patients who require preload reduction, afterload reduction, or vasodilation	Direct action on arterial and venous smooth muscles; nitroglycerin has primary effects on veins; each has effects on coronary and systemic arteries	Nitroglycerin (Nitrodur): Sublingual, transdermal, IV Sodium nitroprusside (Nitropress): IV infusion only	Hypotension, flushing, tachycardia, headache, light-headedness, dizziness, syncope	Thiocyanate toxicity is possible with sodium nitroprusside, especially during prolonged infusions. Monitor vital signs, acid-base status, and cyanide and thiocyanate levels during prolonged treatment.
Phosphodiesterase inhibitors				
Indicated in situations in which both vasodilation and inotropic support are required	Inhibit cAMP in myocardial and vascular tissues	Milrinone (Primacor): IV infusion (after loading infusion)	Hypotension, arrhythmias, nausea, vomiting	Do not use during or immediately after an acute myocardial infarction.
Anticoagulant medications				
Indicated in patients who are at high risk for the development of blood clots; also used to prevent extension of existing blood clots	Various: antiplatelet medications impair platelet aggregation or activation ability; other anticoagulant medications impair blood fibrin formation	Abciximab (Reopro): IV infusion Acetylsalicylic acid (Aspirin): PO Clopidogrel (Plavix): PO Enoxaparin (Lovenox): SC, IV Heparin (Hep-Lock): SC, IV Tirofiban (Aggrastat): IV infusion (after loading infusion)	Potentially life-threatening bleeding disorders, especially in high-risk patients; each medication can cause hematologic disorders; refer to a reliable medication reference prior to administration	Heparin and enoxaparin can each cause blood clot formation in addition to anticoagulation.
Thrombolytic medications				
Given in select situations in which coronary, cerebral, or systemic blood clots place patients at significant risk	Convert plasminogen to plasmin, which breaks down fibrinogen and fibrin in blood clots	Alteplase (Activase, tPA): IV bolus then infusion Reteplase (Retavase): IV bolus x2 Streptokinase (Streptase): IV infusion Tenecteplase (TNKase): IV bolus x1 Urokinase (Kinlytic): IV infusion (after loading infusion)	Potentially life-threatening bleeding disorders	A careful risk-benefit evaluation is essential prior to administration of any of these medications. Catastrophic or lethal complications can occur from uncontrolled hemorrhage following administration. Avoid any unnecessary invasive procedures or trauma in these patients.

Abbreviations: ACE, angiotensin-converting enzyme; AV, atrioventricular; cAMP, cyclic adenosine monophosphate; CHF, congestive heart failure; CNS, central nervous system; ECG, electrocardiographic; IM, intramuscular; PO, oral; SA, sinoatrial; SC, subcutaneous.

Initial pharmacologic treatment of seizures in prehospital and critical care transport should be done with IV benzodiazepines. Rectal diazepam is also an option if it is available for a patient without IV access. Diazepam and lorazepam are the preferred medications, but midazolam has unlabeled use as an anticonvulsant in status epilepticus as well. If seizures persist, if seizures are likely to recur, or if the patient has an illness or injury prone to seizures (such as toxic ingestion or traumatic brain injury), the patient should receive either phenytoin or fosphenytoin. Seizure activity, which is refractory to the previously discussed medications, should be treated with IV barbiturates (phenobarbital or pentobarbital) followed by propofol if necessary.

Phenytoin and Fosphenytoin

Dilantin, generically known as phenytoin, is an anticonvulsant medication prescribed to patients with seizure disorders. It can also be used in the patient with acute status epilepticus by administering 15 to 20 mg/kg IV, infused at a rate no faster than 50 mg/min or 1 mg/kg/min. It should not be used for patients with

cardiac arrhythmias, including AV blocks and bradycardia, though phenytoin is a treatment for arrhythmias in digoxin toxicity. Phenytoin can cause cardiac arrhythmias, including ventricular fibrillation and AV conduction abnormalities, and can cause hypotension as well. It may also cause significant tissue necrosis if extravasated. Extravasation of IV phenytoin causes a "purple glove syndrome," which has required limb amputation in severe situations. Oral phenytoin loading is much safer than administration of IV phenytoin and should be contemplated for conscious, stable, low-risk patients. Oral loading will take approximately 6 hours to reach a therapeutic plasma level. Meticulous dosing consideration is necessary when patients who are already taking phenytoin present following a seizure with a subtherapeutic plasma phenytoin level.

Fosphenytoin (Cerebyx) is a chemically modified form of phenytoin. It is designed to be modified by the body (biotransformed) into phenytoin, which makes it a prodrug of phenytoin. Because it transforms into phenytoin, the dose is documented as milligrams of phenytoin equivalents (PE). The dose for fosphenytoin is 15 to 20 mg PE/kg and should be administered at a rate of 100 mg PE/min. If seizure activity has not resolved, the dose can be increased to 30 mg PE/kg. The remaining information is the same as that for phenytoin. Plasma phenytoin levels will not be accurate until at least 2 hours after fosphenytoin administration.

Barbiturate Medications

Barbiturates are sedative medications, often used for anesthesia, with strong anticonvulsant properties. These medications are not routinely carried by CCTPs, but there is certainly the potential for CCTPs to monitor or administer these medications to patients with intractable seizures who require interfacility transport. These medications possess the ability to decrease cerebral metabolism and cerebral oxygen consumption in at-risk patients.

Phenobarbital is used as an anticonvulsant, although it falls into the barbiturate classification. It is given at a dose of 10 to 20 mg/kg IV for patients exhibiting status epilepticus. As a result of its barbiturate properties, it can cause respiratory and CNS depression. Effects begin in 5 minutes and peak in 30 minutes following IV administration. Both phenobarbital and phenytoin have small margins between therapeutic and toxic serum drug levels, which necessitate close monitoring.

In the event that the patient remains refractory to other medication treatments, pentobarbital may be administered as a last resort. Pentobarbital is a short-acting barbiturate that works as an anticonvulsant but also has sedative and hypnotic properties. Patients are administered a loading dose of 2 to 5 mg/kg and a maintenance dose of 0.5 to 3 mg/kg/h IV infusion. Patients with liver failure should not receive pentobarbital, and those who concurrently consume alcohol may experience an additive effect or death. Like most barbiturate medications, quick cessation of the medication should be avoided. These patients typically require endotracheal intubation for airway protection and ventilator support. Onset of this medication should occur in approximately 1 minute following administration.

Analgesic and Antagonist Medications

Pain is common during critical care transport. Patients experience pain resulting from an illness or injury, or as an unintended consequence of many therapeutic interventions performed by the CCTP. The use of morphine and other opiate-based medications is an essential component of patient care during these transports. Analgesia is the absence of the sensation of pain. Medications that relieve pain are referred to as analgesics. Opiate-based medications are not the only medications with analgesic qualities that are used by CCTPs. Ketamine, discussed earlier, acetaminophen, and various nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently available to CCTPs as well. Sometimes the analgesic itself is not sufficient to relieve pain, in which case an adjunct medication may be given to enhance the effects of the analgesic. When an opiate-based medication is used in conjunction with a benzodiazepine, the dose of

each drug should be lowered to avoid the risk of respiratory depression, unless the patient has an adequately protected airway and supported respiration.

The most common class of medications used for analgesia in the critical care setting is the opioid agonist class. Opioid agonists, either created synthetically or derived from the opium plant, bind to opiate receptors. By blocking these receptors, they prevent the neurons from sending pain signals. Opioid medications are widely abused in society for their euphoric properties.

These medications are also CNS and respiratory depressants. Opioid toxicity or overdose can readily be identified by the triad of respiratory depression, sedation, and pinpoint pupils. The diagnosis of opioid intoxication is frequently confirmed with reversal of these symptoms by an opioid antagonist medication, discussed later.

Fentanyl, Sufentanil, and Morphine

Fentanyl (Sublimaze) is a popular opioid agonist because it is rapid acting, is very potent, and has a relatively short duration of action. It is a synthetic opiate that is said to be anywhere from 75 to 200 times the potency of morphine and also has minimal cardiovascular effects, although bradycardia, tachycardia, and arrhythmias have been reported. Hypotension is also possible, especially when sympathetic stimulation from pain is artificially maintaining an increased blood pressure. It is common for hemodynamic, CNS, and respiratory status to decline when painful stimulation is suddenly eliminated by opioid medications. With high IV doses of fentanyl, chest wall "rigidity" has been reported. The actual incidence of this in clinical practice is debated, but CCTPs should be aware that it may occur. When given at a dose of 0.5 to 2 μg/kg, the patient will experience analgesia within about 90 seconds, with an effective duration lasting approximately 30 minutes.

Sufentanil (Sufenta) is a similar medication to fentanyl, which also has significant analgesic effects with minimal cardiovascular effects. It is most commonly used in surgical settings, but it is possible for CCTPs to encounter patients receiving this medication. The dose is started at 1 to 2 μg/kg (up to 8 μg/kg) for analgesia and increased up to the dose range of 10 to 25 μg/kg when used as an anesthesia induction agent in pediatric patients. Doses greater than 8 μg/kg are considered induction doses. As with other opioid agonists, patients with respiratory compromise should not receive sufentanil unless their airway and ventilations can be supported. The adverse reactions are consistent with fentanyl, including chest wall rigidity, cardiac arrhythmias, and respiratory depression.

Morphine has long been popular for the prehospital induction of analgesia. It is commonly administered at a dose of 0.05 to 0.1 mg/kg, and it alleviates pain through CNS depressant actions. It also suppresses fear and anxiety centers in the brain and brain stem respiratory centers. It has a tendency to increase peripheral venous capacitance and decrease venous return, which decreases preload and afterload and ultimately decreases myocardial oxygen demand. Although this decrease in oxygen demand is typically considered a benefit, care must be exercised to prevent hypotension. Morphine can also cause respiratory depression and somnolence. It should not be given to patients who have respiratory compromise, including patients with asthma, or those who are hypotensive or hypovolemic. Patients frequently report a flushed or itchy feeling with morphine, which is a histamine release, not a true allergic reaction. Morphine may also precipitate (or worsen) nausea or vomiting. It should be avoided in supine patients, in patients who are likely to vomit, and in turbulent aircrafts.

Other Opiate-Based Analgesic Medications

Hydromorphone is a potent analgesic medication that is also well known by the trade name Dilaudid. This medication has a particularly high abuse potential. There is also a notably higher risk of respiratory arrest and death when combined with alcohol or other CNS depressants.

Nubain, or generically known as nalbuphine, is an opioid agonist-antagonist that has a much shorter

list of miscellaneous side effects, although the typical respiratory depression, hypotension, and somnolence still exist. Nalbuphine is typically given at a dose of 10 mg IV, IM, or SQ every 3 to 6 hours.

Naloxone and Other Opioid Antagonists

Opioid antagonists reverse the effects of opioid drugs. They work by competitively binding with the opiate receptors in an antagonistic manner, meaning they do not stimulate the receptor to initiate its action. As a result of this binding, the opioid molecules cannot get to the receptor. The most common opioid antagonist used in the prehospital and critical care transport setting is naloxone (Narcan). Given at a dose of 0.4 to 2 mg, the medication takes effect quickly, but has a shorter half-life than most opiate-based products, which could necessitate repeat administrations. Many opioid overdoses require much more than 2 mg. After 10 mg total without improvement, opioid intoxication is unlikely to be the cause of apnea or unresponsiveness. Patients with apparent opioid intoxication should only receive enough naloxone to restore effective respiratory functioning. Excessive naloxone in opioid-addicted individuals will cause violent withdrawal symptoms, placing the patient, CCTPs, and other health care providers at risk, with potentially lethal consequences. Patients should receive small doses of naloxone at short intervals, while airway and respirations are supported, until adequate reversal of symptoms occurs. The risk of withdrawal symptoms in at-risk individuals should not be underestimated.

NSAIDs

NSAIDs are an alternative to opioid-based analgesic medications. The natural reaction to injury or infection in the body is to promote inflammation in the area(s) involved in an effort to build up the defense and rebuilding cells. This promotion is carried out by prostaglandins, which also have involvement in fevers and platelet activities. Prostaglandins are created by an enzyme found in the cells called cyclooxygenase, or COX for short. Several NSAIDs work by blocking the COX enzymes and thereby decreasing the inflammation-causing prostaglandins.

Ketorolac is the generic name for Toradol. Ketorolac is a medication used for pain management that is in the NSAID category. It is typically administered at a dose of 30 mg IV or 60 mg IM. It is an effective and popular pain medication, but several important details exist pertaining to the use of this drug. It should only be used for short-term or acute pain and not for minor or chronic pain complaints, and it should not be used for more than 5 days total. Ketorolac has been found to cause severe GI ulcers, bleeding, and/or perforations. It can precipitate significant cardiovascular events, including thrombosis, and should not be used for patients with advanced renal impairment. It is also strongly contraindicated for use in patients with hemorrhagic risks and patients in labor.

Neurologic medications used in critical care transport are summarized in **Table 7-6**.

Medications Affecting the GI System

The role and significance of GI medications in critical care transport is easy to underestimate. Aircraft or vehicle turbulence, certain toxic exposures, medication side effects, and numerous clinical conditions can cause nausea or vomiting that requires treatment during transport. GI bleeding is an often lethal condition, requiring critical care transport and the administration of specialized medications. During or after a severe illness or injury, many patients require parenteral nutrition that is often continued during critical care transport. CCTPs will find themselves frequently administering GI medications.

TABLE 7-6 Neurologic Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Anesthetic, antianxiety, sedative, and hypnotic agents				
Patients who require sedation for comfort or safety during transport	Haloperidol blocks postsynaptic dopaminergic receptors, depresses the reticular activating system, and slows the release of hypothalamic hormones. Dexmedetomidine antagonizes alpha-2 adrenergic receptors, inhibiting neurotransmission.	Haloperidol (Haldol): PO, IM, IV (IV is common but unapproved) Dexmedetomidine (Precedex): IV infusion (after loading infusion)	Sedation, hypotension, bradycardia, nausea. Haloperidol has numerous significant side effects, including paradoxical agitation, ECG changes, neuroleptic malignant syndrome, and extrapyramidal symptoms.	Numerous side effects and medication interactions associated with haloperidol significantly limit its usefulness.
Hydantoin anticonvulsants				
Patients in status epilepticus or those who are at high risk for seizure activity (ICH, TBI)	Decrease cellular sodium levels in the motor cortex of the CNS; increase effective refractory period in neural cells; also possess antiarrhythmic activity.	Phenytoin (Dilantin): PO, IV Fosphenytoin (Cerebyx): IM, IV	CNS depression, cardiovascular collapse, tachycardia, nausea, cardiac arrhythmias (especially with phenytoin), venous irritation, Stevens-Johnson syndrome.	IV fosphenytoin is considered safer for administration than phenytoin. IV phenytoin should not be infused any faster than 50 mg/min in adults. Monitor IV sites closely for signs of infiltration.
Barbiturates				
Used periodically for sedation or for status epilepticus in patients refractory to first-line anticonvulsants	Depress the sensory cortex, which slows cerebral metabolism and decreases cerebral oxygen consumption.	Phenobarbital: PO, IM, IV Pentobarbital: IM, IV	Bradycardia, hypotension, syncope, profound CNS depression, respiratory depression, Stevens-Johnson syndrome, nausea, vomiting.	These patients typically require airway management while receiving IV barbiturates.
Opioid analgesic medications				
Pain control or prevention in patients with discomfort from illness, injury, or invasive procedures, or as an adjunct to sedation in mechanically ventilated patients	Bind with opioid receptors in the CNS, preventing painful impulse transmission	Fentanyl (Sublimaze): Transdermal, IM, IV Hydromorphone (Dilaudid): PO, SC, IM, IV Meperidine (Demerol): PO, SC, IM, IV Morphine sulfate (Roxinol): PO, SC, IM, IV Nalbuthine (Nubain): SC, IM, IV Sufentanil (Sufenta): IV	Respiratory depression, apnea, hypotension, bradycardia, palpitations, flushing, nausea, vomiting, noncardiogenic pulmonary edema	Titrate medication doses and intervals to each particular patient and situation. Opioid overdose may be reversed with an opioid antagonist medication.
Opioid antagonist medications				
Reversal of opioid intoxication following accidental, intentional, or therapeutic overdose	Displace opioids and bind with opioid receptor sites in the CNS	Naloxone (Narcan): Endotracheal tube, SC, IM, IV, intranasally	May precipitate withdrawal symptoms in patients addicted to opioids	Carefully titrate naloxone intervals and dosing to reverse opioid-induced respiratory depression and sedation without precipitating withdrawal symptoms. Many opioids have a longer duration of action than naloxone, risking undetected return of toxic symptoms.
NSAIDs				
As adjunctive pain relief in patients who have already received or cannot receive opioid medications; various NSAIDs have antipyretic properties as well; decrease inflammation	Inhibit prostaglandin synthesis by impairing the function of cyclooxygenase	Ibuprofen (Advil, Motrin): PO Ketorolac (Toradol): PO, IM, IV	Abdominal discomfort, gastritis, hematologic abnormalities, gastrointestinal hemorrhage, Stevens-Johnson syndrome, renal insufficiency	Provide adequate hydration. Administer oral preparations with food.

Abbreviations: CNS, central nervous system; ECG, electrocardiographic; ICH, intracranial hemorrhage; IM, intramuscular; NSAID, nonsteroidal anti-inflammatory drug; PO, oral; SC, subcutaneous; TBI, traumatic brain injury.

Histamine-2 Blocker Medications

Histamine-2 (H_2) blocker medications are used clinically to minimize the effects of gastric acids on the stomach and digestive tract. Acids and enzymes are produced in the stomach by cells in the stomach lining, including the gastric parietal cells. Stimulation of these cells can occur by stimulating the H_2 receptor on the cells. The medications used to reduce acid production do so by antagonizing the H_2 receptors. These medications are particularly useful because they are very selective for the H_2 receptors,

have no effect on histamine-1 (H_1) receptors, and have no anticholinergic effects. Despite this, H_2 blockers potentiate the effects of H_1 (common antihistamine) medications, which are used to treat allergic reactions and related symptoms. This additive effect is beneficial when treating anaphylaxis and other significant allergic reactions.

Ranitidine (Zantac) is one such H_2-blocking medication used to treat gastroesophageal reflux disease. There is no proven benefit of IV ranitidine compared with oral doses of 150 mg. Particular caution needs to be used in patients with renal failure, and adverse reactions include diarrhea, constipation, dizziness, dry mouth, and dry skin.

Cimetidine (Tagamet) works the same as ranitidine, but the dose is higher at 400 mg. Cimetidine may increase the serum levels of some cardiac medications and tricyclic anti-depressants. Elderly patients may experience confusion, and young males can experience impotence when taking cimetidine. Serious adverse reactions include neutropenia, anemia, depression, psychosis, and hallucinations.

Famotidine (Pepcid) is similar to both cimetidine and ranitidine. It selectively blocks the H_2 receptor to reduce the release of acid in the stomach. The dose is 20 mg and has no significant contraindications. Famotidine can cause cardiac arrhythmias, but most other serious side effects are extremely rare.

Phenothiazine Antiemetic Medications and Metoclopramide

The general classification of antiemetic medications, those intended to treat nausea and vomiting, is comprised of more specific classifications. Nausea and vomiting are typically triggered in the medulla and can also be triggered by the vestibular system. The vestibular system has a high concentration of both histamine and acetylcholine. By using certain antihistamine medications, the activity of the histamine in the vestibular system can be inhibited, thereby suppressing the stimulation to the medulla. The same suppression can be accomplished by blocking the acetylcholine receptors as well. Meclizine and dimenhydrinate are two examples of antihistamines used as antiemetics. Scopolamine is another anticholinergic used to treat nausea and vomiting. Both of these groups have similar adverse reactions, with sedation, visual acuity changes, and, most notably, the worsening of narrow-angle glaucoma.

The vomiting center of the medulla can be triggered by the neurochemical transmitter dopamine when it activates the D_2 receptor. Medications that antagonize the D_2 receptors are effective in reducing nausea and vomiting. Promethazine (Phenergan) and prochlorperazine (Compazine) are phenothiazine-type antiemetics that are in this category. Promethazine is typically administered at a dose of 12.5 to 25 mg IV or IM, which is cut in half for elderly patients. Significant drawbacks exist, however, because these medications have been found to have major side effects. They can cause considerable sedation, orthostatic hypotension, and extrapyramidal effects. Promethazine should always be diluted with normal saline when injected intravenously.

Prochlorperazine (Compazine) is very similar to promethazine in that it blocks dopamine at the D_2 receptor in the medulla. The dose is 2.5 to 10 mg every 3 to 4 hours when given IV. It is contraindicated in patients who have bone marrow disease and severe liver or cardiovascular disease. This medication frequently causes Parkinson-like syndromes and can produce extrapyramidal effects. It is imperative that prochlorperazine is administered slowly or by infusion. Rapid injection frequently causes orthostatic hypotension. Both prochlorperazine and promethazine have the potential for seizures and may increase the likelihood of seizures in patients already predisposed to them. Use caution with these medications in elderly and other at-risk patients.

Metoclopramide, or Reglan, has similar action on the D_2 receptors but also has the added benefit of increasing GI motility. This is accomplished by enhancing ACh effects on receptors in the upper GI tract. Metoclopramide can cause drowsiness, dizziness, akathisia, and dystonic reactions, similar to other phenothiazine antiemetics. CCTPs should administer 5 to 10 mg IV or IM to most routine patients who

require an antiemetic. When nausea and vomiting control is essential in situations such as chemotherapy and oral antidote administration, metoclopramide can be administered in large doses up to 1 to 2 mg/kg. Many providers administer diphenhydramine (Benadryl) in these situations as prophylaxis against dystonic reactions. Rapid IV infusion can cause a profound dysphoria. IM and IV infusions are strongly preferred. The action of metoclopramide begins within 1 to 3 minutes and persists for 1 to 2 hours.

5-HT_3 Receptor Antagonist Antiemetics

The serotonin antagonists are believed to cause their effect by selectively blocking serotonin receptors in the medulla to suppress the vomiting centers. These medications have been gaining popularity as a result of the relatively low-level adverse reactions. The most common reported reaction is fatigue, which is followed by headache and diarrhea. Ondansetron is typically referred to by its trade name Zofran and is administered at 4 mg IV, IM, or PO. Granisetron (Kytril) and dolasetron (Anzemet) are other 5-HT_3 receptor antagonists that are gaining popularity. They each have a mechanism similar to ondansetron. Granisetron is usually administered IV during critical care transport, ranging from 0.1 to 2 mg/dose. Dolasetron is given at 0.35 mg/kg up to a maximum of 12.5 mg, although much higher doses are used for chemotherapy. Dolasetron, ondansetron, and granisetron work well in preventing emesis but have minimal to varied effectiveness in patients who are already vomiting. Clinician experience demonstrates more effectiveness in the treatment of active vomiting than the mechanism would suggest.

Antisecretory Agents: Somatostatin and Octreotide

Upper GI bleeding is a frequent, often fatal, medical condition. One hundred thousand hospital admissions occur each year for this condition, and patients with upper GI bleeding have an overall 10% mortality rate. Causes of upper GI bleeding include duodenal ulcers (approximately 35%), gastric ulcers (approximately 20%), hemorrhagic gastritis (18%), and esophageal varices (5% to 11%, depending on geographic region). Esophageal varices occur as a consequence of elevated portal venous pressure (portal hypertension), where portosystemic and gastroesophageal collateral blood pathways develop. Severe, potentially lethal, GI hemorrhage is the most significant risk associated with esophageal varices.

Somatostatin is a naturally occurring peptide chain that decreases intestinal fluid secretion, slows GI motility, and reduces portal blood flow with vascular smooth-muscle vasoconstriction. Somatostatin has been replicated pharmacologically for the management of bleeding from esophageal varices and other disorders, but it is extremely limited by its approximately 3-minute half-life.

Octreotide (Sandostatin) is a synthetic peptide with actions analogous to somatostatin with a longer, more clinically useful, duration of action. Octreotide is administered therapeutically for control of peptide-secreting tumors, certain acquired immunodeficiency syndrome–related diarrhea conditions, fistulas, and a variety of other GI conditions in addition to its primary use in critical care as a vasoconstrictor in patients with bleeding esophageal varices. Another novel and intuitively unrelated use of octreotide is for the treatment of hypoglycemia following an overdose of oral sulfonylurea (a hypoglycemic agent for control of type 2 diabetes).

Octreotide is given as a loading dose of 25 to 100 μg IV over at least 3 minutes, followed by an IV infusion of 25 to 50 μg/h. Common side effects include bradycardia, chest pain, fatigue, malaise, headache, diarrhea, and abdominal discomfort.

Total Parenteral Nutrition and Partial Parenteral Nutrition

Severe illness, major surgery, and GI tract dysfunction are widespread in critical care patients. Wound healing, hypermetabolic states, and physiologic stressors significantly increase nutritional requirements in these patients. When oral nutrition is inadequate or impossible, critical care providers use either partial parenteral nutrition (PPN) (administered other than through the digestive tract) or total parenteral nutrition (TPN). PPN is given to supplement enteral (into the digestive tract) feeding. TPN is administered as the

sole nutritional source. For purposes of this section, the terms TPN and PPN will be used interchangeably. Each type of nutrition is administered IV through a central venous catheter or a peripherally inserted central catheter, or PICC line. Parenteral nutrition can be administered concurrently with a lipid solution, although not in the same container.

Parenteral nutrition is customized for each patient based on estimated caloric and nutrition needs, adjusted for that patient's particular medical condition and any nutritional deficits. It is prepared in a hospital pharmacy and confirmed with the orders at the patient's bedside. H_2-blockers, electrolytes, insulin, glucose, minerals, and appropriate IV fluids are frequently present in TPN.

TPN is administered via an infusion pump at a precise rate. Unless line incompatibility problems or equipment limitations exist, parenteral nutrition should be maintained (continued) during interfacility critical care transport. No additional medications should be added to TPN or lipid solutions. No other medications should be administered into an IV line infusing TPN or lipids as well.

Any patient receiving insulin and/or dextrose should have frequent, scheduled determinations of blood glucose levels. This is important, even if the infusion has been discontinued during the transport. The effects of insulin will persist after the infusion has been stopped. CCTPs who use portable point-of-care testing devices such as the I-STAT should assess electrolyte and acid-base status during transport, especially if the transport period is extended. If this type of device is unavailable, it is important that CCTPs assess the most recent laboratory values at the sending facility and closely monitor the patient during transport for any signs of fluid or electrolyte abnormalities. In addition to glucose, fluid, or electrolyte abnormalities, parenteral nutrition may adversely impact the liver, gallbladder, and certain hematologic parameters.

Gastrointestinal medications used in critical care transport are summarized in **Table 7-7**.

Miscellaneous Medications Used in Critical Care Transport

The following section contains selected medications that do not easily fit into one of the broad categories previously described, or have widespread use for a variety of clinical situations. CCTPs should be familiar with these medications, including their indications, major side effects, and administration considerations.

Alprostadil

Alprostadil (prostaglandin E_1, [PGE]) is a vasodilator medication used to treat erectile dysfunction, but its major role in critical care transport is to maintain a patent ductus arteriosis (PDA) in newborn infants with certain congenital heart defects. The use of this medication in infants with ductal-dependent heart defects provides a life-sustaining bridge until major corrective surgery can be accomplished. Specialty consultation and training is required for CCTPs who carry this medication or who may be expected to administer or monitor this medication. Alprostadil is administered as an IV infusion at 0.05 to 0.1 µg/kg/min, which may be increased if results are unsatisfactory. Maintenance infusions can range from 0.01 to 0.4 µg/kg/min. The role of specialty consultation when giving this medication cannot be overstated. Neonatal medications should undergo additional scrutiny when being calculated or administered.

There is a greater than 10% chance of apnea when alprostadil is given intravenously. It is imperative that CCTPs consider early RSI or airway management in these patients rather than risk apnea occurring at a time when effective airway management is difficult or impossible, such as during severe aircraft turbulence. Flushing, fevers, bradycardia or tachycardia, hypotension, and hypertension are also frequent side effects of IV alprostadil.

TABLE 7-7 Gastrointestinal Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Histamine-2 blocker medications				
Gastric acid reduction in patients with ulcers, reflux, or excessive gastric secretions; adjunct to histamine-1 blockers in patients with allergic or anaphylactic reactions	Inhibition of histamine-2 receptors, in cells that release gastric acids	Famotidine (Pepcid): PO, IV Ranitidine (Zantac): PO, IM, IV Cimetidine (Tagamet): PO, IM, IV	Agitation, arrhythmias, confusion, vomiting, headache, dizziness, diarrhea, constipation	Modify the dose in patients with significant renal disease.
Phenothiazine antiemetics				
Nausea or vomiting; patients who would have catastrophic consequences from vomiting (ocular globe injury, patients on a backboard, various toxic exposures)	Block dopamine receptors in the cerebral medulla; have some antihistamine properties that also suppress nausea and vomiting	Promethazine (Phenergan): PO, PR, IV, IM Prochlorperazine (Compazine): PO, PR, IV, IM Metoclopramide (Reglan): PO, IM, IV	Hypotension, hypertension, bradycardia, tachycardia, sedation, akathisia, extra pyramidal symptoms, neuroleptic malignant syndrome, seizures, delirium, disorientation	Use slow infusion when prochlorperazine is administered IV. Monitor patients carefully for many possible side effects.
5-Hydroxytriptamine receptor antagonist antiemetics				
Nausea or vomiting; patients who would have catastrophic consequences from vomiting (ocular globe injury, patients on a backboard, various toxic exposures)	Selectively block serotonin receptors in the medulla to suppress vomiting	Dolasetron (Anzemet): PO, IV Granisetron (Kytril): PO, IV Ondansetron (Zofran): PO, IM, IV	Headache, drowsiness, fatigue, dizziness	Theoretically, these antiemetics work best before vomiting starts.
Antisecretory agents				
Bleeding esophageal varices	Decrease intestinal fluid secretion, decrease gastrointestinal motility, reduce portal blood flow	Octreotide (Sandostatin): SC, IM, IV	Exhaustive list of side effects with this medication	These agents may also be used for treatment of oral sulfonylurea overdose. Hypoglycemia or hyperglycemia may occur in diabetic patients.

Abbreviations: IM, intramuscular; PO, oral; PR, as required; SC, subcutaneous.

Drugs Used in Infectious Diseases

Critical care providers routinely transport patients with an active infectious disease or who are receiving treatment to prevent an opportunistic infection associated with their illness or injury. These patients require appropriate levels of personal protective equipment. Newer approaches to treatment such as early goal-directed sepsis management will only increase the role of antimicrobial medications in critical care transport. Early goal-directed sepsis management is a diagnostic and management strategy for patients with possible sepsis who receive prompt antimicrobial therapy and hemodynamic support when they meet specified criteria, designed to improve patient care and decrease mortality. In many instances, CCTPs will be expected to initiate appropriate antimicrobial therapies in high-risk patients at outlying community health care settings or during transport to a tertiary care center.

In most transport situations, antimicrobial therapy will have already been initiated before the CCTP arrives. It is essential that CCTPs determine whether any antimicrobial agents need to be administered during each transport. Depending on the pharmacodynamics and pharmacokinetics of a particular medication, a missed dose may alter the medication's plasma level and effectiveness for hours or even days. In severe cases, such as with gas-forming bacteria or profound sepsis, delays in initiating antibiotic therapy may have lethal consequences.

Antibiotics

Antibiotics, a subclassification of antimicrobial medications, are classified into several categories based on their composition and the types of bacteria they target. Not all antibiotics affect all types of infections. Antibiotics generally work by killing bacteria, or by preventing multiplication of bacteria, and thereby allowing the body's immune system to overcome the infectious invaders. Many patients are allergic to certain antibiotics. These patients may be allergic to multiple antibiotics or multiple groups of antibiotics. It is essential that CCTPs assess for the presence of medication allergy and consider the possibility of cross-reactivity of other antibiotics. In addition to allergic cross-reactivity, antibiotics and other antimicrobials can potentiate, inactivate, or otherwise alter the performance of other antimicrobials or medications. It is essential that each patient's medication profile be considered individually for medication cross-reactivity. Medication line incompatibility is a significant concern when multiple medications, especially antibiotics, are administered simultaneously. There are simply too many antibiotics and other antimicrobials that a CCTP may encounter to discuss individual indications and characteristics of each individual medication. CCTPs should develop the practice of evaluating each patient care situation to ensure proper administration and monitoring of antibiotics and other medications through the use of reliable, current reference materials.

Antifungal Medications

Fungal infections have evolved into a significant factor in critical care medicine. Immunocompromised patients, immunosuppressant medications, malignancy, and an increased prevalence of invasive procedures have promoted fungal infections to this dubious status. Five IV medications and numerous oral and topical antifungal medications are currently in widespread use in the United States. Each IV preparation has a long list of side effects and adverse reactions, including several with CNS and cardiovascular side effects occurring with a greater than 10% frequency. The CCTP should evaluate each medication when transporting patients receiving antifungal therapy.

Calcium Preparations

Calcium is an essential medication for CCTPs. Calcium chloride and calcium gluconate are the two IV preparations that are available. A 10-mL dose of calcium chloride provides approximately three times as much elemental calcium as 10 mL of calcium gluconate, although when identical elemental calcium doses are administered, the clinical effects appear identical. Each is typically supplied in a 10-mL vial or Bristojet. The IV dose is often tailored to a particular situation, to either replace a calcium deficiency or mitigate the toxic effects of another substance.

IV calcium is indicated in numerous clinical situations. Hypocalcemia is the most obvious indication. Hypocalcemia is identified either by hospital laboratory testing or on many of the portable point-of-care diagnostic devices being used in critical care transport. Normal adult serum calcium levels are between 8.2 and 10.2 mg/dL, or between 4.60 and 5.08 mg/dL for ionized calcium, which is the more clinically active of the two values. Calcium levels must be adjusted when patients have hypoalbuminemia, hemolysis, and certain blood transfusions. Hypocalcemia is observed with numerous cardiac arrhythmias, hypotension, prolonged QT intervals, or T-wave inversions on ECG. Noncardiac manifestations include muscle cramps, tetany, seizures, tremors, and ataxia.

Another indication for IV calcium is the prevention of cardiac arrhythmias associated with hyperkalemia. Severe hyperkalemia presents with characteristic ECG changes that are an indication for calcium as a myocardial cell membrane stabilizer until the excess potassium can be sequestered or eliminated.

Calcium is also given intravenously to treat hypotension and bradycardia associated with calcium channel–blocker overdoses. Verapamil (Calan) is often pretreated with IV calcium to prevent unwanted

hypotension. Additionally, IV calcium can improve blood pressure, but not pulse rate, in patients experiencing beta-blocker toxicity.

Calcium is the antidote for magnesium toxicity. When magnesium sulfate infusions are being administered to obstetric patients as a tocolytic or for pregnancy-induced hypertension, either calcium gluconate or calcium chloride should be readily available.

Hydrofluoric acid toxicity (discussed in detail in Chapter 20) is treated with aggressive calcium replacement. IV, oral, and/or topical calcium is administered in large quantities to these patients.

Use extreme caution when administering IV calcium. Extravasation may lead to significant tissue necrosis, especially from calcium chloride. When only peripheral access is available, calcium gluconate is the preferred agent for calcium replacement. Use a secure IV line, placed into a large vein, and monitor the infusion and site frequently for any signs of early infiltration. Hypotension, vasodilation, arrhythmias, and syncope are all possible side effects from IV calcium administration. Calcium can cause lethal arrhythmias in patients with cardiac glycoside toxicity (discussed in detail in Chapter 20).

Diphenhydramine

Diphenhydramine (Benadryl) is another medication carried by CCTPs with a myriad of therapeutic uses. It is an H_1 antihistamine, capable of anticholinergic effects, with a variety of beneficial clinical effects. Diphenhydramine is administered by itself or with a combination of additional medications for the treatment of allergic and anaphylactic reactions. Diphenhydramine is used as a mild sedative and sleep aid as an over-the-counter medication and can be used intravenously for this same purpose with often more profound results. This medication has mild antitussive (cough suppressant) and mild antiemetic properties in the setting of motion sickness. Diphenhydramine is extremely useful in the management of a dystonic reaction or in patients with extrapyramidal symptoms from various other medications.

Most CCTPs carry the injectable form of diphenhydramine (50 mg/mL). Adults receive 25 to 50 mg for most situations, but may receive up to 0.5 to 1 mg/kg for severe dystonic reactions. The adverse effects of diphenhydramine include tachycardia, palpitations, and chest pain, in addition to drowsiness, dizziness, and ataxia. Many other side effects are characteristic of anticholinergic medications, such as dry mouth, constipation, restlessness, agitation, and seizure activity. Despite the numerous potential side effects, diphenhydramine is well tolerated in most patients.

Glucagon

Glucagon is an endogenous peptide produced in the pancreas that has been replicated commercially for use in medicine and critical care transport. Glucagon is carried by CCTPs for use as either a rescue agent for acute hypoglycemia in patients without IV access or an antidote in select overdose or toxicity situations.

One milligram of glucagon is administered to larger children and adults who are hypoglycemic and do not have IV access. Ideally, IV dextrose would be administered to these patients, but without proper IV access, parenteral dextrose cannot be administered. Glucagon is given IM as a bridge until IV access can be established or the patient is able to receive oral sugars. Glucagon causes the increased production of cAMP, which in turn promotes hepatic glycogenolysis and gluconeogenesis, ultimately raising blood glucose levels. Onset begins in 30 minutes, and elevations in blood glucose levels can be observed up to 90 minutes following glucagon administration.

Glucagon receives widespread, but unlabeled, use as an antidote for both beta-adrenergic blocker and calcium channel blocker overdoses or toxicity. In these situations, glucagon also increases cAMP, which causes a potent increase in heart rate (chronotropy) and an increase in myocardial contractility (ino-tropy). In this setting, glucagon is administered 5 to10 mg IV followed by an infusion of 1 to 10 mg/h. These situations often require more glucagon than is readily available to most CCTPs.

Glucagon is usually packaged in powder form with phenol included for reconstitution. At high doses used for beta-blocker and calcium channel–blocker toxicity, sterile water, rather than phenol, should be used for reconstitution to avoid the risk or effects of phenol toxicity from the large amount required.

Magnesium Sulfate

Magnesium sulfate is another versatile IV medication frequently carried and encountered by CCTPs. Similar to calcium, the most obvious indication for magnesium sulfate is documented or presumptive hypomagnesemia, or low serum magnesium levels. Extracellular fluid contains approximately 1/20th the magnesium concentration of intracellular fluid. Only 1% of total body magnesium is present in extracellular fluids, making serum magnesium levels a poor indicator of actual body magnesium content. Despite this, the widespread use of serum magnesium monitoring continues. Clinical signs of hypomagnesemia include cardiac arrhythmias (atrial and ventricular), CNS hyperexcitability (psychosis, seizures, or disorientation), and neuromuscular symptoms (muscle cramps or hyperactive deep tendon reflexes). Causes of hypomagnesemia include impaired GI absorption, nutritional deficiencies, excessive GI secretions, dramatic fluid volume expansion, and uncontrolled renal excretion from pathologic or chemical toxicity.

Magnesium sulfate is used aggressively to treat ventricular cardiac arrhythmias, specifically torsade de pointes, ventricular tachycardia, and ventricular fibrillation. In these situations, 1 to 2 g of magnesium sulfate is infused over 5 minutes, which is a much faster treatment than most other situations.

The use of magnesium sulfate in high-risk obstetric patients is becoming controversial. Magnesium remains the first-line anticonvulsant in patients with preeclampsia and eclampsia. Patients with eclampsia who receive magnesium as an anticonvulsant have a lower recurrence of seizures than those treated with conventional anticonvulsants, such as benzodiazepines and phenytoin. The role of magnesium in pre-eclampsia and eclampsia is largely undisputed. Patients are typically administered a 4- to 6-g bolus over 15 to 20 minutes followed by an infusion at 2 g/h. It is essential that CCTPs closely monitor patient respiratory status, blood pressure, and deep tendon reflexes during magnesium sulfate infusions.

Magnesium sulfate as a uterine tocolytic agent is facing additional scrutiny. Its use continues to be widespread for this purpose in the management of preterm labor. Safety concerns and questionable efficacy have been cited in several exhaustive meta-analysis studies. Pulmonary edema, increased fetal mortality, and maternal respiratory arrest from magnesium toxicity are cited as reasons not to administer magnesium sulfate for this condition.

Finally, another indication for magnesium sulfate is asthma exacerbation.

Magnesium sulfate causes respiratory arrest or hypoventilation, particularly during IV infusions. Hypotension, vasodilation, cardiac conduction abnormalities, and a flushed feeling are also possible. IV calcium preparations (discussed previously) are the antidotes for magnesium toxicity. Hemodialysis is sometimes necessary for severe magnesium overdoses.

Potassium Chloride

Potassium is the major intracellular electrolyte, essential for nerve and muscle tissue function. Normal serum potassium values range from 3.5 to 5 mEq/L, reflecting only 2% to 3% of total body potassium stores. Large amounts (up to 500 mEq/d) are absorbed and eliminated each day. The kidneys are almost entirely responsible for potassium regulation in healthy persons. Many critical care patients have hypokalemia, or are at risk for hypokalemia, defined as a serum potassium level of less than 3.5 mEq/L. This serum value will not reflect actual body potassium levels in many situations such as acid/base alterations, hemolysis, and errors during common laboratory sampling techniques (pseudohyperkalemia).

Patients presenting with hypokalemia often have vague signs and symptoms. Abdominal pain, nausea, vomiting, paresthesias, and constipation are common symptoms. ECG changes, arrhythmias (especially

premature ventricular contractions and ventricular arrhythmias), hypotension, respiratory failure, and mental status changes are more severe manifestations. It is now possible to diagnose hypokalemia using portable point-of-care testing devices during critical care transport.

When potassium stores are depleted from impaired absorption, kidney dysfunction, or unintended elimination, potassium replacement is essential. Potassium chloride is available as oral and IV preparations. ECG monitoring is highly recommended for any IV infusions totaling greater than 10 mEq/h. Peripheral IV infusions should not be administered at a rate greater than 10 mEq/h. Central venous catheter infusions can be administered up to 40 mEq/h at concentrations no greater than 40 mEq/100 mL. Peripheral IV administration is likely to cause local discomfort. Hyperkalemia is the main adverse side effect of IV administration, demonstrated by ECG changes (especially peaked T waves), paralysis, palpitations, hypotension, and bradycardia. Avoid or use extreme caution when administering potassium chloride to patients with renal disease or impairment, patients with acid/base disorders, and patients receiving cardiac glycosides. If potassium levels fail to increase during repletion, it is important to assess magnesium levels. The kidneys depend on magnesium for renal potassium reabsorption.

Sodium Bicarbonate

Sodium bicarbonate is an electrolyte solution with powerful alkalinization effects. It is used as a pharmacologic therapy for certain (but not all) metabolic acidosis conditions. Sodium bicarbonate enhances sodium channel membrane functioning to stabilize myocardial functioning following tricyclic antidepressant and other medication overdoses. Salicylate removal is increased when sodium bicarbonate is used to alkalinize urine following an aspirin overdose. Other medications, particularly acidic formulations, have enhanced elimination when a patient's urine is alkalinized with sodium bicarbonate. Sodium bicarbonate promotes intercellular potassium shifts in patients with documented or presumptive hyperkalemia and may be administered when these patients experience cardiac arrest. Additionally, sodium bicarbonate protects renal tubules from damage by deposited myoglobin in patients with rhabdomyolysis. This is a powerful, capable medication carried by CCTPs.

Sodium bicarbonate is typically administered at 0.5 to 1 mEq/kg per dose IV push, ideally into a central venous catheter. Salicylate and tricyclic overdose situations and rhabdomyolysis are indications for a sodium bicarbonate infusion. IV infusions may also be used when certain variants of metabolic acidosis are present. Sodium bicarbonate is not recommended for routine use in cardiac arrest except for situations of documented metabolic acidosis or hyperkalemia.

Numerous electrolyte abnormalities (hypokalemia, hypocalcemia, and hypernatremia) and alkalosis are side effects of sodium bicarbonate. Cerebral hemorrhage, pulmonary edema, and congestive heart failure exacerbations are possible as a result of electrolyte changes and fluid volume overload. Alterations of the oxygen-hemoglobin dissociation curve occur following sodium bicarbonate administration, increasing hemoglobin oxygen affinity.

Tocolytic Agents in Critical Care Transport

Pharmacologic tocolytics are used clinically to prolong fetal gestation in order to minimize adverse medical conditions associated with prematurity or to allow time for maternal-administered corticosteroids to enhance fetal lung maturity in the event of a premature delivery. CCTPs are often requested to transport patients in preterm labor. At the time of this text, there are no current FDA-approved chemical tocolytic agents to suppress uterine contractions. Ritodrine, a beta-adrenergic agonist, had been approved for this purpose, but it has since been discontinued in the United States. Any other medications used as tocolytic agents have not been approved for this purpose.

Terbutaline and magnesium sulfate have been previously discussed. Magnesium sulfate has considerable risks to a pregnant woman and fetus but continues to receive widespread use. Magnesium is

generally administered as an IV bolus followed by infusion, which requires close monitoring by CCTPs during transport.

Terbutaline is a beta-2 agonist that is approved as a bronchodilator medication and is used frequently as a tocolytic medication. Terbutaline administration is often associated with hypertension, tachycardia, restlessness, and tremors. Pulmonary edema from terbutaline has led to at least 25 maternal deaths. A low-dose subcutaneous continuous or intermittent infusion appears to be effective for tocolysis without many of the side effects or tachyphylaxis issues. The FDA appears to be accepting the role of terbutaline as a tocolytic, but no new approvals for this use have been granted as of this writing.

The calcium channel blockers, nifedipine (Procardia) and nicardepine (Cardene), have tocolytic properties and are frequently used for this purpose. In general, the side effects with these medications are minimal, especially when given orally. Serious side effects, including pulmonary edema, have been reported in a small number of patients. It is advisable to avoid these medications in hemodynamically unstable patients and those with preexisting cardiovascular disease.

CCTPs may additionally encounter patients who have received indomethacin (Indocin) and various COX 1 and 2 inhibitors for tocolysis. These medications are in various phases of use and evaluation, but may be given to patients as a tocolytic agent; these patients then require transport. The safety and efficacy of these medications are still being debated.

Miscellaneous medications used in critical care transport are summarized in **Table 7-8**.

Medication Infusion Technique

Medication infusion is indicated when the patient requires continuous medication administration and has restricted fluid requirements. When altitude increases, IV drip rates will increase; conversely, when altitude decreases, IV drip rates will decrease. Flow rates should not be affected at 1,500′ to 2,000′. Patients should always be placed on pumps to ensure precise medication delivery during critical care transport.

A two-tone alarm from the infusion system indicates that infusion is complete or the battery is low. Four quick beeps and a corresponding red light alarm on the screen indicate a more serious problem. Silence the alarm and press the *Alarm channel* key if needed to access alarm information, then press *Start/stop* to restart fluids.

TABLE 7-8 Miscellaneous Medications for Critical Care Transport

Indications	Mechanism of Action	Commonly Used CCT Medications and Route of Administration	Side Effects Common to This Medication Class	Special Considerations
Alprostadil (prostaglandin E)				
Maintains patent ductus arteriosis in neonates with certain ductal dependent congenital heart defects	Prostaglandin, which causes direct relaxation of ductus arteriosis and vascular smooth muscle	IV infusion	Apnea, flushing, fever, bradycardia, tachycardia, hypotension, hypertension, cardiac arrest	Anticipate more than a 10% chance of apnea. Consider early rapid sequence intubation or airway management. Consult an appropriate specialist when managing neonatal patients who require alprostadil.
Calcium chloride and calcium gluconate				
Documented or presumed hypocalcemia, hyperkalemia with ECG changes, magnesium toxicity, hydrofluoric acid exposure	Replacement of lost or sequestered body calcium; membrane stabilizer that promotes normal cell function	IV	Hypercalcemia, tissue necrosis following extravasation, arrhythmias, hypotension, bradycardia	Avoid IV calcium in patients with cardiac glycoside toxicity. Closely monitor peripheral IV sites for signs of infiltration.
Diphenhydramine (Benadryl)				
Allergic or anaphylactic reactions, mild sedation, treatment of dystonic reactions, mild cough suppressant, mild antiemetic, extrapyramidal symptoms	Histamine-1 blocker with anticholinergic effects	IV, IM, PO	Palpitations, tachycardia, hypotension, seizures, insomnia, sedation, dry mucous membranes, decreased GI motility, nausea, vomiting, paradoxical excitement	The drug is well tolerated in most patients despite the wide range of potential side effects.
Glucagon				
Hypoglycemic patients without IV access; unlabeled use for beta-blocker and calcium channel-blocker toxicity	Endogenous peptide that increases production of cAMP, causing gluconeogenesis and glycogenolysis	IM, IV	Hypotension, hypertension, tachycardia, nausea, vomiting	Large quantities may be potentially required for severe beta-blocker or calcium channel-blocker overdose. Reconstitute with sterile water when large quantities of glucagon are administered.
Magnesium sulfate				
Hypomagnesemia, ventricular arrhythmias, anticonvulsant in patients with preeclampsia and eclampsia, unlabeled use as a tocolytic	Stabilizes excitable cell membranes, slows SA node conduction, decreases acetylcholine in motor nerve terminals, electrolyte essential for ion movement across cell membranes	IV bolus infusion or maintenance infusion	Flushing, hypotension, respiratory depression/apnea, vasodilation, cardiac conduction abnormalities	Have IV calcium available when administering magnesium sulfate. Monitor respiratory status, blood pressure, and deep tendon reflexes in patients receiving a continuous magnesium infusion.
Potassium chloride				
Documented or presumed hypokalemia	Electrolyte required for nerve, muscle cell, and tissue functioning; helps maintain acid-base balance	IV infusion, PO	Hyperkalemia, ECG changes, muscle paralysis, palpitations, hypotension, bradycardia	Administer no more than 10 mEq/h through peripheral IV sites. Use cardiac monitoring for patients receiving infusions totaling more than 10 mEq/h. Use extreme caution in patients with renal disease, patients with acid-base alterations, and those receiving cardiac glycosides. Consider point-of-care testing during transport if available.
Sodium bicarbonate				
Certain types of metabolic acidosis, for enhanced elimination of certain toxic exposures, severe hyperkalemia, rhabdomyolysis	Powerful alkalinizing agent that stabilizes various cell membranes, increases serum and urinary pH, and promotes renal excretion of weak acids and myoglobin	IV bolus or infusion	Cerebral hemorrhage, edema, worsening of congestive heart failure, hypernatremia, metabolic alkalosis, hypocalcemia, hypokalemia, altered hemoglobin oxygen affinity	Drug is not indicated for routine administration during cardiac arrest unless hyperkalemia or metabolic acidosis is documented. Monitor serum potassium during and after administration.

Abbreviations: cAMP, cyclic adenosine monophosphate; ECG, electrocardiographic; GI, gastrointestinal; IM, intramuscular; PO, oral; SA, sinoatrial.

The sequence for medication infusion is presented in **Skill Drill 7-1** and described as follows:

1. To set up a medication infusion system, start by pressing the *On/Off* key for 1 second, and then prime the tubing system **Step 1**.
2. Pull the white slide clamp on the cassette to the out position and insert the cassette into the appropriate channel **Step 2**.
3. After placing the tubing collar into the recessed area, select the appropriate channel by pressing *ABC*

on the soft pad that corresponds to the pump **Step 3**.

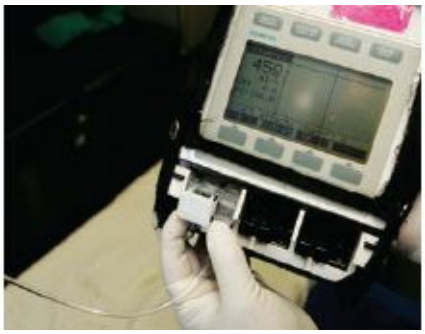
4. Set the rate and volume on the system and press the *Accept* button **Step 4**.
5. Press *Select* to advance the volume infused; press *Clear* to zero the amount infused **Step 5**.
6. Press the *Standard Display* key and then press *Start/Stop* to initiate infusion **Step 6**.

Fluids that are run using a dial-a-flow are not considered reliable and should be switched to IV infusion pumps.

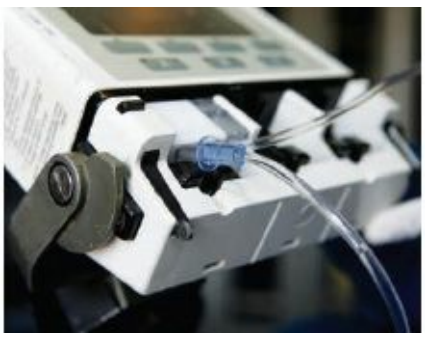
Skill Drill 7-1

Administering Medication With an Infusion Pump

1 Press the *On/Off* key for 1 second and then prime the tubing system.

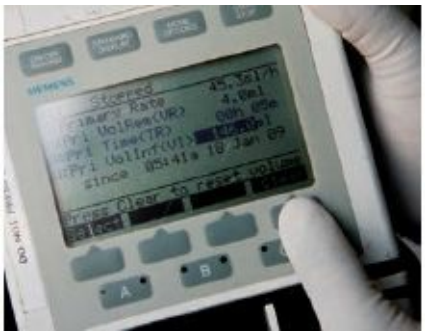


2 Pull the white slide clamp on the cassette to the out position. Insert the cassette into the appropriate channel.

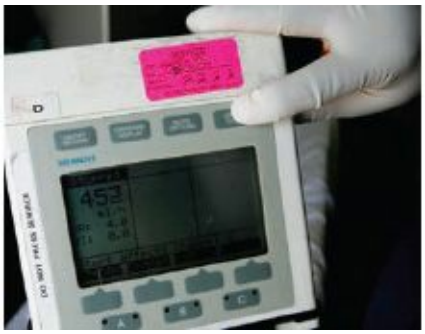


3 Place the tubing collar into the recessed area. Select the appropriate channel by pressing *ABC* on the soft pad that corresponds to the pump.

4 Set the rate and press *Accept*. Adjust volume and press *Accept*.



5 Press *Select* to advance the volume infused. Press *Clear* to zero the amount infused.



6 Press the *Standard Display* key. Press *Start/Stop* to initiate infusion.

Summary

The variety of medications used in critical care medicine is significantly greater than medications used in standard prehospital medicine. It is necessary that the CCTP have an intimate understanding of the medications used and the effects they may have when administered. Continued review and research are necessary to truly effectively treat critical care patients.

Case Study

YOU ARE CALLED TO A REGIONAL HOSPITAL EMERGENCY department for a male patient who is experiencing an active ST-segment elevation myocardial infarction (STEMI) and requires cardiac intervention and emergent air transfer to a tertiary care facility. Transport time by air will be 20 minutes.

Upon arrival, the staff reports that the 55-year-old, 122-kg man presented to the emergency department with chest pain with a rating of 7/10 and mild shortness of breath. He has had chest pain on and off for the last few days and awoke this morning with symptoms. His evaluation revealed an active

STEMI.

The patient's medical history and report per the referring registered nurse includes hypertension, coronary artery disease, and hyperlipidemia. This patient's prescription medications include Lopressor, Plavix, and aspirin.

In the emergency department, he was placed on the monitor and given oxygen at 4 L/min via nasal cannula. An 18-gauge angiocath was placed in the left forearm, and a 20-gauge angiocath was placed in the right hand. Blood samples were obtained. Both IVs were 0.9% normal saline. He received one nitroglycerin tablet sublingually, and a drip of 10 μg/min was initiated with a 100 mg/250 mL concentration running at 3 mL/h. Acetylsalicylic acid, 325 mg, was given, and 2 mg of morphine was administered as well. Serial ECGs revealed ST elevation in V_3 and V_4, which is indicative of an anterior infarct. Positive cardiac markers confirmed the ECG findings. A heparin drip of 1,000 U/h was initiated using a 25,000 U/250 mL concentration running at 10 mL/h. A 30.6-mg bolus of Reopro was given, and a drip using 9 mg/250 mL was initiated at 15.25 μg/min, or 17 mL/h. The patient also received 5 mg of metoprolol via IV push. His pain is currently a 4/10. His temperature is reported as 98.6°F (37°C). The referring nurse has no further report.

Your assessment reveals he is alert and oriented and consents to transfer. He reports chest pain at 4/10, but his shortness of breath feels resolved. Current vital signs at this facility are a blood pressure of 126/86 mm Hg, a pulse rate of 84 beats/min and normal sinus rhythm on the ECG, respirations of 16 breaths/min and nonlabored, and an oxygen saturation as measured by pulse oximetry (SpO_2) of 97% with 4 L/min oxygen via nasal cannula. The patient feels afebrile to touch. An examination of the patient's head, ears, eyes, nose, and throat reveals equal reactive pupils at 5 mm, a midline trachea, no jugular vein distention, and no audible carotid bruits on auscultation. His chest is symmetric with equal rise and fall, and lung sounds are clear with auscultation. His S_1 and S_2 heart tones are normal on auscultation, and no rubs or gallops are noted. He has a moderately obese abdomen and no organomegaly or pulsatile masses. His extremities are within normal limits, with no trauma, deformity, or other abnormality noted. Pulse quality is 2^+ (normal) on palpation. All IVs are infusing well, and no local redness or swelling is noted. The nitroglycerin is infusing in the right IV, and the heparin and Reopro is infusing in the left IV.

You duplicate flow rates and transfer his IVs to your pumps. The ECG, blood pressure cuff, and SpO_2 sensors are transferred to your monitors. Oxygen is transferred to your portable cylinder. The patient is transferred to your cot and secured, and IVs are transferred to the cot. You discharge the patient from the emergency department, obtain all records, and move to your helicopter. He is placed in the ship, and all oxygen and power needs are transferred to onboard systems. You lift off and transport is begun.

En route no changes in the patient's hemodynamic status are noted, as vital signs remain within the previously described range. He continues to complain of chest pain at 4/10, but has no shortness of breath. He denies having any nausea. Medical control and standing protocols are initiated. You arrive within the 90-minute door-to-balloon time standard at the tertiary care facility, and your patient is moved directly to the cath lab and transferred to staff without incident. You give your report and all paperwork to the charge nurse and retreat to complete your paperwork and reassemble your ship for the next call.

1. Are the medication doses correct?
2. Are any compatibility issues identified?
3. What further adjustments can be made to current medications to benefit this patient?

Analysis

The case study represents some of the most common scenarios a CCTP will face. The medication dosing is *not* correct. Although all the concentrations are within standards, the dosing for nitroglycerin is not

correct. The patient is receiving 20 µg/min with this concentration.

The administration of heparin and Reopro is under scrutiny. Although Reopro is not reported as having incompatibility issues in the same IV line, the manufacturer recommends that it is run separately in an IV. This could be accomplished by placing the nitroglycerin and heparin into the same IV.

This patient's condition may be better treated by increasing his nitroglycerin dose incrementally and monitoring his responses. Further doses of morphine may be used as well. Any adverse reactions may require discontinuing one or more of these medications.

This patient has had, and is receiving, multiple anticoagulant medications. He must be monitored for any bleeding because he is a substantial anticoagulation risk from his history. If bleeding is suspected, the CCTP should stop the infusions and immediately consult with medical control or follow established protocols. No prescreening information for anticoagulation was readily offered. It is always good practice to ask for this information because it may uncover a contraindication to medication use. Speed is essential in time-sensitive patients, but moving too quickly without paying attention to details is just as detrimental to the patient as moving too slowly.

There is a standard treatment for STEMI, but treatments and medications vary from region to region because of local physician preferences, drug availability and cost, and referral patterns related to facilities. The CCTP must be familiar with the protocols of his or her region and common treatment modalities. It does appear that the referring facility and the transferring crew all share a common ground in treating the patient's STEMI, preventing further ischemia, and rapidly transferring for interventions.

The identification of a wrong medication dose is of paramount importance. Although all health care providers strive to provide accurate care, errors can and do occur. The CCTP must ensure that a mistake is not repeated. Some of the most common errors involve mistaken concentrations, which result in dosing errors. This risk is increased when IV drips are hand mixed by the practitioner. The CCTP must ensure that concentrations are correct and match the dosing required. All medications administered using IV drips should be checked for flow rate and dosing accuracy. The CCTP must be well versed in many pharmacologic interventions. It is always recommended that up-to-date pocket guides or charts be consulted as well for accuracy as a result of the large number of medications you may encounter.

Another issue the CCTP must be alert to is compatibility. Although many medications are compatible with each other, many are not. Again, double-checking all medications for compatibility is a responsibility of the CCTP. Adjustments may be required to obtain compatibility in medications administered.

An understanding of pharmacology is a very challenging part of CCTP practice. The responsibilities of the CCTP can be as simple as ensuring the highest flow rates are properly placed in the IV lines to avoid line back-up and alarms, to making complex mathematic calculations to ensure that proper medication doses are being administered. It is the responsibility of CCTPs to maintain their knowledge of current and emerging pharmacology and to maintain due diligence in practice.

Prep Kit

Ready for Review

- Pharmacology is defined as the study of the preparation, properties, uses, and actions of medications.
- Current and reliable medication information may be obtained from a variety of print and electronic reference sources, including the Internet.
- Medications work in one of four ways: (1) by binding with a receptor site on a particular cell to either promote or inhibit a specific activity of that cell; (2) by affecting the physical properties of a cell and,

thereby, its functioning; (3) by combining directly with other chemicals within the body to either alter or limit the effects of a chemical or allow it to be removed; and (4) by altering a metabolic pathway.

- In down-regulation, as the medication molecules bind to their corresponding receptor sites, the number of receptors decreases. In up-regulation, the medication increases the number of available receptor sites.
- The drug-response relationship correlates the amount of medication given with the response it causes.
- The onset of action is defined as how long it takes for the concentration of the medication at the target tissue to reach the minimum effective level.
- The duration of action is defined as how long the medication can be expected to remain above that minimum level to provide the intended action.
- The termination of action is the amount of time after the concentration level falls below the minimum level to the time it is eliminated from the body.
- The onset of action, duration of action, and termination of action combine to determine the therapeutic index for a medication, which gives an indication of the medication's margin of safety (defined as the ratio of a drug's lethal dose for 50% of the population to its effective dose for 50% of the population).
- Patients of different ages may have vastly different responses to the same drug, owing to age-related factors such as renal function, muscle mass, body water/fat content, and metabolic rate.
- Both pediatric and geriatric patients often have slower absorption and elimination times, necessitating modification of the doses of many drugs administered to these patients.
- To correct for differences in weight between patients, drugs are dosed in milligrams or micrograms per kilogram of the patient's body weight.
- Other factors that may affect the dose-response relationship include gender, environmental factors (eg, stress), time of the medication's administration, the patient's overall state of health, and genetic factors.
- In pregnant patients, many medications will pass from the mother's bloodstream into the placenta and ultimately affect the fetus. For this reason, many medications are either contraindicated or used only after careful consideration in pregnant patients.
- Every medication has some side effects—that is, reactions that can manifest as undesired signs or symptoms but that are nevertheless expected based on how the medication works.
- An iatrogenic response is an adverse condition inadvertently induced in a patient by the treatment given. Some of these responses are devastating and even potentially lethal.
- Medication errors related to selection, administration, or monitoring are responsible for thousands of deaths annually—and the chaos often associated with critical care transport conditions makes such errors more likely.
- The most common unpredictable response encountered in the prehospital setting is the allergic reaction.
- The effects of one medication may alter the response of another medication in sometimes unexpected ways, a phenomenon known as a drug interaction.
- The critical care transport environment continually exposes personnel and vehicles to temperature extremes; it is essential that medications remain within their required temperature range.
- The optimal medication for a particular situation may or may not be immediately available during patient transport. For example, specialized medications such as antidotes, thrombolytics, and antibiotics are not routinely carried by critical care transport organizations.

- The transport team must be adequately trained and equipped to recognize and effectively manage anticipated side effects or adverse reactions associated with a particular medication.
- The route of administration affects how quickly and how much of an administered dose is absorbed into the patient's body. CCTPs generally use IV, intramuscular, subcutaneous, oral, sublingual, transdermal, nasal, and sometimes rectal routes for medication administration.
- The route of medication administration affects bioavailability—the amount of an administered medication that reaches the patient's systemic circulation without any alteration.
- A medication or other foreign substance in the body undergoes elimination, biotransformation, or both. The kidneys and urinary system are the main routes of elimination.
- Biotransformation may have any of four outcomes: (1) an active drug may be changed into an inactive metabolite and eliminated; (2) an active drug can be changed into an active metabolite that causes continued or new clinical effects; (3) an active medication may be changed into a reactive metabolite that has a greater potential for action than its parent substance; and (4) an otherwise inactive substance may change into an active, potentially toxic substance.
- Definitive airway management in critical care transport combines techniques for airway device placement with medications designed to facilitate this placement.
- To create the best patient condition possible for invasive airway procedures, the following classes of drugs are used: ultra-short-acting nonbarbiturate hypnotic agents, sedative hypnotic agents, benzodiazepines, and paralytic medications (depolarizing or nondepolarizing agents). Typically, a sedative-hypnotic agent is used in conjunction with a chemical paralytic agent.
- Beta-agonist medications act on receptors in the lower airways to improve air flow into and out of the lungs.
- Medications such as catecholamines, beta-blocking agents, calcium channel–blocking agents, and anticholinergic agents may simultaneously affect both the heart and the blood vessels and are used to ensure effective functioning of the cardiovascular system.
- Other medications involved in cardiovascular management include anticoagulants and thrombolytics, phosphodiesterase inhibitors, and diuretics.
- Clonidine and phentolamine are alpha-blocking medications encountered periodically during critical care transport; other alpha-blocking oral medications that may be observed as a patient's personal medication include terazosin, doxazosin, and methyldopa.
- Phentolamine should be carried by any transport provider who administers any IV medication with alpha-adrenergic properties (eg, norepinephrine, epinephrine, dopamine).
- Angiotensin-converting enzyme inhibitors inhibit vasoconstriction and block the breakdown of the vasodilator bradykinin.
- The management of cardiac arrhythmias involves several classes of antiarrhythmic medications, many of which create additional risk for catastrophic and lethal rhythm disturbances.
- Class I antiarrhythmic medications work by blocking sodium channels; they include anticholinergic medications such as quinidine, procainamide, and disopyramide.
- Class II antiarrhythmic medications (beta-blockers) work by competitively binding to the beta receptor sites to inhibit their stimulation. They are used to treat arrhythmias, hypertension, acute myocardial infarctions, and congestive heart failure.
- Class III antiarrhythmic medications (potassium channel blockers) are particularly useful for treating reentry tachycardias and arrhythmias occurring because of cellular depolarization as a result of

excessive excitability. Their use may produce prolonged QT intervals on the patient's ECG.

- Class IV antiarrhythmic medications (calcium channel blockers) cause vasodilation, negative inotropy, negative chronotropy, and negative dromotropy. Because these medications decrease the conduction velocity through the atrioventricular node, they are useful for treating tachyarrhythmias originating in the atria.
- Adenosine is not effective in converting atrial fibrillation, but may be used as a diagnostic tool when differentiation between atrial fibrillation and supraventricular tachycardia cannot be made as a result of the rate.
- Anticholinergic medications include diphenhydramine, a potent antihistamine, and other histamine-blockers. Other anticholinergic medications are administered therapeutically to treat nausea and vomiting, excessive secretions from a variety of organs, abdominal cramps, and excess vagal tone leading to bradycardia or asystole.
- Many RSI protocols include atropine as a prophylactic medication to prevent bradycardia from succinylcholine administration and airway manipulation, especially in pediatric patients.
- Dopamine (a sympathomimetic agent) stimulates alpha-1 receptors when given at a higher dose, thereby producing peripheral vasoconstriction. Stimulation of the dopaminergic receptors may have a myriad of effects: increased myocardial contractility and cardiac output, increased renal output, decreased fluid volume, and increased blood pressure.
- Epinephrine (a sympathomimetic agent) is administered to support respiratory function by opening up larger airways; to increase blood pressure by increasing vascular tone; and to enhance myocardial perfusion during cardiac arrest resuscitation.
- Other sympathomimetic agents include norepinephrine, phenylephrine, and dobutamine; all these agents are administered to improve cardiac output.
- Hydralizine is used with apparent safety as an emergent antihypertensive in pregnant patients with preeclampsia. Additional uses include congestive heart failure, primary pulmonary hypertension, and malignant hypertension.
- Diuretics are widely used by CCTPs for situations such as fluid volume overload, cerebral edema, hyperkalemia, and rhabdomyolysis. The most commonly administered diuretic is furosemide, although both bumetanide and mannitol are used for these purposes as well.
- The traditional first-line medication for cardiac ischemia is nitroglycerin (a nitrate). Nitroglycerin also treats hypertension. Nitroprusside (another nitrate) is both an arterial and a venous dilator, which makes it more potent than nitroglycerin; at normal doses, nitroglycerin is only a venous dilating agent.
- CCTPs often encounter situations that require dissolution of blood clots with thrombolytics, prevention of blood clots with anticoagulants, and expansion of the circulating blood volume with crystalloid and colloid medications.
- Aspirin is considered to be the first-line anticoagulant medication and is technically considered an antiplatelet medication.
- Glycoprotein IIB-IIIA inhibitors are becoming increasingly popular as antiplatelet medications, but carry a high risk of excessive bleeding.
- When a patient is taking heparin, the CCTP should review the latest lab values and the partial thromboplastin time prior to transport and monitor the patient closely for bleeding problems.
- Enoxaparin is a low-molecular-weight heparin that is used as an adjunct to thrombolytic medications in patients experiencing myocardial infarctions and in patients experiencing unstable angina.

- CCTPs frequently encounter patients taking warfarin who have suffered devastating effects (eg, cerebral hemorrhage) from inadequate monitoring and dose adjustments. This situation should be approached cautiously, because the reversal of anticoagulation can be just as dangerous as over-anticoagulation in high-risk patients.
- Thrombolytic medications are administered to break down an existing clot.
- Prehospital blood products and blood substitute products are still in the investigational stage, so CCTPs must rely on other fluids to replace the missing blood volume—namely, crystalloid solutions and colloid solutions.
- Three types of crystalloid solutions are used in the field: isotonic, hypertonic, and hypotonic solutions.
- Isotonic solutions have the same tonicity as human blood. Normal saline and lactated Ringer's solution are the most commonly used isotonic fluids.
- Albumin (a blood product) is a colloid solution that is used to treat hypovolemia, burns, and certain surgical procedures.
- Hetastarch is a synthetic colloid solution that is designed to expand the plasma volume. When it is used, consideration must be given to the possibility of pulmonary edema, fluid overload, and congestive heart failure.
- Dextran—another volume expander—is used in situations when blood products are not immediately available or to prime the tubing of various external medical devices that sequester large amounts of blood.
- Analgesia, anxiolysis, sedation, and seizure control are the main purposes of neurologic medications administered by CCTPs.
- Benzodiazepines are versatile, treating anxiety, controlling seizures, and providing sedation in a vast array of clinical situations.
- The initial pharmacologic treatment of seizures in prehospital and critical care transport should be done with IV benzodiazepines.
- Mannitol, a powerful diuretic medication, is often utilized for cerebral edema and may be a last-ditch effort to save a patient from impending brain stem herniation.
- Magnesium sulfate is highly effective in controlling seizures in preeclamptic and eclamptic obstetric patients.
- The use of morphine and other opiate-based medications is an essential component of patient care during critical care transport. Other analgesic medications used may include ketamine, acetaminophen, and various NSAIDs.
- A variety of gastrointestinal medications are used in critical care transport to counteract nausea or vomiting, control gastrointestinal bleeding, or provide parenteral nutrition during or after a severe illness or injury.
- Histamine-2 blocker medications are used clinically to minimize the effects of gastric acids on the stomach and digestive tract.
- Nausea and vomiting are typically triggered in the medulla and can also be triggered by the vestibular system, which is characterized by a high concentration of both histamine and acetylcholine. Antihistamine medications, which inhibit the activity of the histamine in the vestibular system, and medications that block the acetylcholine receptors are often used to treat nausea and vomiting.
- Medications that antagonize the D_2 receptors—such as the phenothiazine-type antiemetics—are

effective in reducing nausea and vomiting.

- The serotonin antagonists are believed to exert an antiemetic effect by selectively blocking serotonin receptors in the medulla to suppress the vomiting centers.
- Somatostatin is a synthetic peptide (based on a naturally occurring peptide) that decreases intestinal fluid secretion, slows gastrointestinal motility, and reduces portal blood flow with vascular smooth muscle vasoconstriction.
- Octreotide is a synthetic peptide with actions analogous to somatostatin, but a longer, more clinically useful, duration of action.
- Unless line incompatibility problems or equipment limitations exist, parenteral nutrition should be maintained (continued) during interfacility critical care transport. No additional medications should be added to total parenteral nutrition or lipid solutions.
- Any patient receiving insulin and/or dextrose should have frequent, scheduled blood glucose levels checked during transport because the effects of insulin will persist for a period of time after the infusion has been stopped.
- Alprostadil (prostaglandin E_1; PGE) is a vasodilator medication that is used during critical care transport to maintain a patent ductus arteriosis in newborn infants with certain congenital heart defects.
- CCTPs will be expected to initiate appropriate antimicrobial therapies in high-risk patients at outlying community health care settings or during transport to a tertiary care center.
- When selecting antibiotics, CCTPs must assess for the presence of medication allergy and consider the possibility of cross-reactivity of other antibiotics. In addition, they must assess the patient's medication profile individually, recognizing that antibiotics and other antimicrobials can potentiate, inactivate, or otherwise alter the performance of other antimicrobials or medications.
- Increases in the numbers of immunocompromised patients, immunosuppressant medications, malignancies, and invasive procedures have increased the prevalence of fungal infections in recent years. Five IV and numerous oral and topical antifungal medications are currently in widespread use.
- IV calcium is indicated in numerous clinical situations encountered by the CCTP—to treat hypocalcemia, for the prevention of cardiac arrhythmias associated with hyperkalemia, to treat hypotension and bradycardia associated with calcium channel–blocker overdoses, as an antidote for magnesium toxicity, and to counteract hydrofluoric acid.
- Diphenhydramine is administered by itself or with a combination of additional medications for the treatment of allergic and anaphylactic reactions, for use as a mild sedative, for use as a mild antitussive and antiemetic agent in the setting of motion sickness, and for the management of a dystonic reaction. It is also used in patients with extrapyramidal symptoms from various other medications.
- Glucagon is carried by CCTPs for use as either a rescue agent for acute hypoglycemia in patients without IV access or an antidote in select overdose or toxicity situations.
- Magnesium sulfate is another versatile IV medication frequently carried and encountered by CCTPs. It is used to treat hypomagnesemia, ventricular cardiac arrhythmias, and convulsions in patients with preeclampsia and eclampsia; it is also used as a uterine tocolytic agent.
- Potassium chloride is available as oral and IV preparations to treat patients in whom potassium stores are depleted from impaired absorption, kidney dysfunction, or unintended elimination.
- Sodium bicarbonate is an electrolyte solution that is used as a pharmacologic therapy for certain metabolic acidosis conditions. It is administered to stabilize myocardial functioning following tricyclic antidepressant and other medication overdoses, and to facilitate salicylate removal following

an aspirin overdose.

- When altitudes increase, IV drip rates will increase; conversely, when altitudes decrease, IV drip rates will decrease.
- Patients should always be placed on pumps to ensure precise medication delivery during critical care transport.

Vital Vocabulary

acetylcholine (ACh) Chemical neurotransmitter of the parasympathetic nervous system.

bioavailability The amount or percentage of a medication that reaches systemic circulation without being altered following administration.

chronotropic Altering the rate of contraction of the heart.

cross-tolerance A form of drug tolerance in which patients who take a particular medication for an extended period can build up a tolerance to other medications in the same class.

cumulative effect An effect that occurs when several successive doses of a medication are administered or when absorption of a medication occurs faster than excretion or metabolism.

depolarizing paralytics Medications that cause neuromuscular blockade by binding and briefly activating receptor sites at the neuromuscular junction, preventing further activation of these sites and causing chemical paralysis.

dromotropic Influencing the conduction rate within the heart.

drug antagonism Medications that bind with a receptor site and prevent activation of these receptors by other medications, without causing receptor activation or related clinical effects.

drug interaction The alteration of the action of a particular medication when combined with another medication.

duration of action How long the medication concentration can be expected to remain above the minimum level needed to provide the intended action.

first-order elimination The plasma concentration of a medication or substance is proportional to its rate of elimination.

first-pass effect Medications absorbed from the intestine that are partially inactivated as they pass through the hepatic circulation into the liver before entering the systemic circulation; also called first-pass metabolism.

half-life (T1/2) The time period required to eliminate one half of the plasma concentration. During the second "half-life," an additional 25% of the original plasma concentration is eliminated. After three half-lives, one-eighth of the original plasma concentration remains.

iatrogenic response An adverse condition inadvertently induced in a patient by the treatment given.

idiosyncrasy An abnormal (and usually unexplained) reaction by a person to a medication to which most other people do not react.

inhibition The presence of one medication that decreases the effect of another medication.

inotropic Affecting the force with which muscle tissue contracts, especially cardiac muscle.

interference A direct biochemical interaction between two drugs.

mechanism of action The way in which a medication produces the intended response.

nicotinic receptors Cholinergic receptors that bind with the neurotransmitter acetylcholine.

nondepolarizing agents Medications designed to cause temporary paralysis by binding in a competitive but nonstimulatory manner to part of the ACh receptor; they do not cause fasciculations.

onset of action The time needed for the concentration of the medication at the target tissue to reach the minimum effective level.

pharmacodynamics The branch of pharmacology that studies reactions between medications and living structures, including the processes of body responses to pharmacologic, biochemical, physiologic, and therapeutic effects.

pharmacokinetics The study of the metabolism and action of medications with the particular emphasis on the time required for absorption, duration of action, distribution in the body, and method of excretion.

potentiation The effect of increasing the potency or effectiveness of a drug or other treatment; may occur by administering two medications concurrently, where one increases the effect of the other.

side effects Reactions that can manifest as signs or symptoms that are not desired but are expected based on how the medication works.

steady state A point in drug administration at which the rate of administration (frequency and dose) is equal to the rate of elimination, resulting in a constant plasma medication level.

summation effect The process whereby multiple medications can produce a response that the individual medications alone do not produce.

sympathomimetic Medication that mimics the body's sympathetic nervous system response (fight or flight response); includes epinephrine and norepinephrine.

synergism An interaction of two or more medications that results in an effect that is greater than the sum of their effects if taken independently.

tachyphylaxis A condition in which the patient rapidly becomes tolerant to a medication.

termination of action The amount of time after the medication's concentration falls below the minimum effective level until it is eliminated from the body.

therapeutic index The ratio of a drug's lethal dose for 50% (LD_{50}) of the population to its effective dose for 50% (ED_{50}) of the population; a medication's margin of safety.

tolerance Physiologic adaptation to the effects of a drug such that increasingly larger doses of the drug are required to achieve the same effect.

Vaughan-Williams classification scheme Classification system for antiarrhythmic medications.

zero-order elimination Medications or chemicals are eliminated from the body at a constant rate, regardless of plasma concentration.

Bibliography

American Academy of Pediatrics. Ideal body weight calculation. Available at: http://www.pediatriccareonline.org/pco/ub/view/Pediatric-Drug-Lookup/153916/0/Ideal_Body_Weight_Calculation. Accessed February 2, 2009.

American Heart Association. Part 10.6: anaphylaxis. Available at: http://circ.ahajournals.org/cgi/content/full/112/24_suppl/IV-143. Accessed February 5, 2009.

Azer SA. Esophageal varicies. Available at: http://emedicine.medscape.com/article/175248-overview. Accessed February 18, 2009.

Azer SA. Esophageal varicies: treatment and medication. Available at: http://emedicine.medscape.com/article/175248-treatment. Accessed February 18, 2009.

Baglin TP. Heparin-induced thrombocytopenia thrombosis (HIT/T) syndrome: diagnosis and treatment. *J Clin Pathol*. 2001;54:272–274.

Barnet JC, Touchon RC. Short-term control of supraventricular tachycardia with verapamil infusion and calcium pretreatment. *Chest*. 1990;97:1106–1109.

Benowitz NL. Antihypertensive agents. In: Katzung BG, ed. **I** *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007.

Boersma E, Harrington RA, Moliterno DJ, et al. Platelet glycoprotein IIbIIIa inhibitors in acute coronary syndromes: a meta-analysis of all major randomized clinical trials. *Lancet*. 2002;359(9302):189–198.

Birnbuamer DM. Phenytoin loading in the ED: which method and agent is best? Available at: http://emergency-medicine.jwatch.org/cgi/content/full/2004/525/1. Accessed February 16, 2009.

Brubacher JR. Beta-adrenergic agents. In: Goldfrank LR, Howland ME, Flomenbaum NE, Hoffman RS, Lewin RS, Nelson LS, eds. *Goldfrank's Toxicologic Emergencies*, 7th ed. New York, NY: McGraw-Hill Co; 2002.

Carter SM, Gross SJ. Glucose-6-phosphate dehydrogenase deficiency. November 13, 2008. Available at: http://emedicine.com/article/200390-overview. Accessed February 5, 2009.

Campo-Soria C, Chang Y, Weiss DS. Mechanism of action of benzodiazepines on GABA receptors. *Br J Pharmacol*. 2006;149:984–990. Available at: http://www.nature.com/bjp/journal/v148/n7/full/0706796a.html. Accessed February 14, 2009.

Cornelius C. Drug use in the elderly: risk or protection? *Curr Opin Psychiatry*. 2004;17(6):443–447.

Corso L. Train-of-four results and observed muscle movement in children during continuous neuromuscular blockade. *Crit Care Nurse*. 2008. Available at: http://www.accessmylibrary.com/coms2/summaryo286-34707839.itm. Accessed February 14, 2009.

Cote CJ, Drop LJ, Daniels AL, Hoaglin DC. Calcium chloride versus calcium gluconate: comparison of ionization and cardiovascular effects in children and dogs. *Anesthesiology*. 1987;66(4):465–470.

Cunningham SJ, Hafeez W. Procedural sedation and pain management techniques. In: King C, Henretig FM, eds. *Pediatric Emergency Procedures*. Philadelphia, PA: Lippincott, Williams and Wilkins; 2007:417–418.

Drugs.com. First-pass metabolism. Available at: http://www.drugs.com/dict/first-pass-metabolism.html. Accessed February 6, 2008.

Erogul M. Pregnancy, preeclampsia: treatment and medication. 2008. Available at: http://emedicine.medscape.com/article/796690-treatment. Accessed February 15, 2009.

Fan E, Stewart TE. Albumin in critical care: SAFE, but worth its salt? *Critical Care*. 2004;8(5):297–299.

Flockhart DA. Drug interactions: cytochrome P450 drug interaction table. 2008. Available at: http://medicine.iupui.edu/flockhart/table.htm. Accessed February 13, 2009.

Fura A. Role of pharmacologically active metabolites in drug discovery and development [electronic version]. *Drug Discovery Today*. 2006;11(3/4):133–142.

Gaither JB. Toxicity, antidysrhythmic. July 9, 2008. Available at:

https://emedicine.medscape.com/article/813046-overview. Accessed March 5, 2009.

Garth D. Hyperkalemia. February 20, 2007. Available at: https://emedicine.medscape.com/article/766479-overview. Accessed February 20, 2007.

Gerber JG, Nies AS. Furosemide-induced vasodilation: importance of the state of hydration and filtration. *Kidney International*. 1980;18:454–459.

Greenblatt DJ, Harmatz JS, von Moltke LL, Shader RI. Pharmacokinetics and pharmacodynamics. Available at: http://www.acnp.org/GN401000085/CH084.html. Accessed February 6, 2008.

Grimes DA, Nanda K. Magnesium sulfate tocolysis: time to quit. *Obstet Gynecol*. 2006;108(4):986-989.

Gropper MA. IV albumin and sepsis. *Medscape Crit Care*. 2001. Available at: http://www.medscape.com/viewarticle/412357. Accessed February 16, 2009.

Gyetvai K, Hannah ME, Hodnett ED, Ohlsson A. Tocolytics for preterm labor: a systematic review. *Obstet Gynecol*. 1999;94:869–877.

Hamilton RJ, ed. *Tarascon Pocket Pharmacopoeia: 2009* [classic shirt pocket edition]. Sudbury, MA: Jones and Bartlett Publishers; 2009.

Harrington L, Leiker C. Dosing of emergency cardiovascular medications in obese patients. *Bariatric Nursing Surgical Patient Care*. 2007;2(2):131–139.

Harvey RP, Schocket AL. The effect of H1 and H2 blockade on cutaneous histamine response in man. *J Allergy Clin Immunol*. 1980;65(2):136–139. Hemmingway TJ, Savitsky EA. Wolff-Parkinson-White syndrome: treatment and medication. August 19, 2008. Available at: http://emedicine.medscape.com/article/761066-treatment. Accessed March 8, 2009.

Hodgson BB, Kizior RJ. *Saunders Nursing Drug Handbook*. St Louis, MO: Elsevier; 2008.

Hoffman BB. Adrenoceptor antagonist drugs. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007.

Hume JR, Grant AO. Agents used in cardiac arrhythmias. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:211–233.

Institute for Altitude Medicine. Healthcare provider information on prevention and treatment of altitude illness. Available at: http://altitudemedicine.org/providers.php. Accessed March 4, 2009.

Junger WG, Coimbra R, Liu FC, et al. Hypertonic saline resuscitation: a tool to modulate immune function in trauma patients? *Shock*. 1997;8(4):235–241.

Kass JE, Terregino CA. The effect of heliox in acute severe asthma: a randomized controlled trial. *Chest*. 1995;107(3):757–760. Available at: http://www.chestjournal.org/content/116/2/296.full. Accessed February 14, 2009.

Kearney TE. Sodium bicarbonate. In: Olson KR, ed. *Poisoning and Drug Overdose*, 5th ed. New York, NY: Lange/McGraw-Hill Co; 2007:423–424.

Kelley RE. Status epilepticus. Available at: http://lib-sh.lsuhsc.edu/fammed/intern/statepil.html. Accessed February 16, 2009.

Kim GH, Hahn DK, Kellner CP, et al. The incidence of heparin-induced thrombocytopenia type II in patients with subarachnoid hemorrhage treated with heparin versus enoxaparin. *J Neurosurg*. 2009;110(1):50–57.

King JF. Tocolysis and preterm labour. *Curr Opinion Obstet Gynecol*. 2004;16(6):459–463.

Klabunde RE. Alpha-adrenoceptor antagonists (alpha blockers). *Cardiovascular Pharmacology Concepts*. 2008. Available at: http://www.cvpharmacology.com/vasodilator/alpha.htm. Accessed

February 15, 2009.

Klabunde RE. Non-pacemaker action potentials. *Cardiovascular Physiology Concepts*. 2008. Available at: http://www.cvphysiology.com/Arrhythmias/A006.htm. Accessed February 15, 2009.

Klabunde RE. Vaughan-Williams classification of antiarrhythmic drugs. *Cardiovascular Physiology Concepts*. 2008. Available at: http://www.cvpharmacology.com/antiarrhy/Vaughan-Williams.htm. Accessed February 15, 2009.

Krause RS. Anaphylaxis: follow-up. October 6, 2008. Available at: http://emedicine.medscape.com/article/756150-followup. Accessed February 17, 2009.

Lacy CF, Armstrong LL, Goldman MP, Lance LL. *Drug Information Handbook*, 7th ed. Hudson, OH: Lexicomp; 2008.

Lam F, Elliot J, Jones JS, et al. Clinical issues surrounding the use of terbutaline sulfate for preterm labor. *Obstet Gynecologic Surv*. 1998;53(11):S85–S95.

Lameire N, Vanholder R, Van Biesen W. Loop diuretics for patients with acute renal failure: helpful or harmful? *JAMA*. 2002;288(20):2599–2601.

Lowson S, Gent JP, Goodchild CS. Anticonvulsant properties of propofol and thiopentone: comparison using two tests in laboratory mice. *Br J Anesth*. 1990;64(1):59–63.

Mansy WH. Toxicokinetics [King Saud University Web site]. Available at: http://faculty.ksu.edu.sa/73895/Documents/Kinetics%20of%20xenobiotics.ppt. Accessed February 6, 2008.

McQuaid KR. Drugs used in the treatment of gastrointestinal diseases. In: Katzung, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:702–703.

Mehta RL, Pascual MT, Soroko S, Chertow GM. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. *JAMA*. 2002;288(20):2547–2553.

Merck Manual Professional Edition. Altitude sickness. Available at: http://www.merck.com/mmpe/sec21/ch320/ch320a.html. Accessed February 17, 2009.

Merck Manual Professional Edition. Total parenteral nutrition. Available at: http://www.merck.com/mmpe/sec01/ch003/ch003c.html. Accessed February 17, 2009.

Parenteral. 2009. *Mosby's Medical Dictionary*. Available at: http://medical-dictionary.thefreedictionay.com/parenteral. Accessed February 17, 2009.

Pharmacology (phar). 2009. *Mosby's Medical Dictionary*. Available at: http://medical-dictionary.thefreedictionary.com/pharmacology. Accessed February 13, 2009.

Nicholl JS. Seizures in the emergency department. 2006. Available at: http://emedicine.medscape.com/article/1187711-overview. Accessed February 16, 2009.

Nochimson G. Toxicity, medication-induced dystonic reactions: treatment and medication. 2008. Available at http://emedicine.medscape.com/article/814632-treatment. Accessed April 1, 2009.

Nolte MS, Karam JH. Pancreatic hormones and antidiabetic drugs. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:702–703.

Novello NP, Blumstein HA. Hypomagnesemia. October 11, 2007. Available at: http://emedicine.medscape.com/article/767546-overview. Accessed February 19, 2009.

O'Donnell CJ, Ridker PM, Hebert PR, Hennekens CH. Antithrombotic therapy for acute myocardial infarction. *J Am College Cardiol*. 1995;25(7):S23–S29.

Oudiz RJ. Pulmonary hypertension, primary. August 29, 2007. Available at:

http://emedicine.medscape.com/article/301450-overview. Accessed August 29, 2007.

Parrillo SJ, Parrillo CV. Stevens-Johnson syndrome. January 23, 2008. Available at: http://emedicine.medscape.com/article/756523-overview. Accessed February 6, 2009.

Phillips TG. Hypothermia. March 28, 2008. Available at: http://emedicine.medscape.com/article/166840-treatment. Accessed February 5, 2009.

Pickkers P, Dormans TPJ, Russel FGM, et al. Direct vascular effects of furosemide in humans. *Circulation*. 1997;96:1847–1852.

Porter RJ, Meldrum BS. Antiseizure drugs. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:374–394.

Prince G. Best bets: which is the best calcium channel blocker for controlling the ventricular rate in patients with atrial fibrillation? Available at: http://www.bestbets.org/bets/bet.php?id=802. Accessed February 15, 2009. *RxList*. Noctec: side effects and drug interactions. Available at: http://www.rxlist.com/noctec-drug.htm. Accessed February 13, 2009.

Schneider RE, Caro DA. Neuromuscular blocking agents. In: Walls RM, Murphy MF, Luten RC, eds. *Manual of Emergency Airway Management*, 2nd ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2004:200–211.

Selleng K, Warkentin TE, Greinacher A. Heparin-induced thrombocytopenia in intensive care patients. *Crit Care Med*. 2007;35(4):1165–1176.

Sharma R, Mir S, Rizvi M, Akthar S. Efficacy of magnesium sulphate versus phenytoin in seizure control and prophylaxis in patients of eclampsia and severe pre-eclampsia. *J K Science*. 2008;10(4):181–185.

Sharma S. Shock and pregnancy. July 27, 2006. Available at: https://emedicine.medscape.com/article/169450-overview. Accessed February 6, 2009.

Sheppard D, Lampiris HW. Antifungal agents. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:781–789.

Sirven JI, Waterhouse E. Management of status epilepticus. *Am Fam Physician*. 2003;68(3). Available at: http://www.aafp.org/afp/20030801/469.html. Accessed February 16, 2009.

Spragg RG, Lewis JF, Wurst W, et al. Treatment of acute respiratory distress syndrome with recombinant surfactant protein C surfactant. *Am J Respiratory Crit Care Med*. 2003; 167:1562–1566.

US Food and Drug Administration. Alpha-adrenergic antagonists [FDA Heart Health Online]. Available at: http://www.fda.gov/hearthealth/treatments/medications/alphaadrenergicantagonists.html. Accessed February 15, 2009.

US Food and Drug Administration. Ritodrine [FDA-Approved Drugs]. Available at: http://www.fdaapproveddrugs.us/ritodrine-hcl.html. Accessed February 19, 2009.

van Heerdan PV. Sedation, analgesia, and muscle relaxation in the intensive care unit. In: Bersten AD, Soni N, eds. *Oh's Intensive Care Manual*, 5th ed. New York, NY: Butterworth Heinemann; 2003.

Venkatesh B. Acute calcium disorders. In: Bersten AD, Soni N, eds. *Oh's Intensive Care Manual*, 5th ed. New York, NY: Butterworth Heinemann; 2003.

Wagner LK. Diagnosis and management of preeclampsia. *Am Fam Physician*. 2004;70:2317–2324.

Walker M. Status epilepticus: an evidence based guide. *Br Med J*. 2005;331:673–677.

Wilkes NJ, Woolf RL, Powanda MC, et al. Hydroxyethyl starch in balanced electrolyte solution (Hextend) pharmacokinetic and pharmacodynamic profiles in healthy volunteers. *Anesth Analg*.

2002;94:538–544.

Worthley LI. Fluid and electrolyte therapy. In: Bersten AD, Soni N, eds. *Oh's Intensive Care Manual* , 5th ed. New York, NY: Butterworth Heinemann; 2003:885–895.

Zehnder JL. Drugs used in disorders of coagulation. In: Katzung BG, ed. *Basic and Clinical Pharmacology*, 10th ed. New York, NY: McGraw-Hill Co; 2007:542–559.

Laboratory Analysis and Diagnostic Studies

Authors

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Objectives

1. Understand the overall principles of laboratory analysis (p 236).
2. Understand the difference between sensitivity and specificity (p 236).
3. Discuss the difference between specimen culture and sensitivity (p 237).
4. Understand relevant basic chemistry and physiology (p 237–238).
5. Recognize the most commonly ordered laboratory tests done in the emergency department and in the intensive care unit (p 239–247).
6. Understand the importance of abnormal laboratory results as they relate to patient condition (p 239–247, 250–252).
7. Discuss the causes of abnormal laboratory results (p 239–247, 250–252).
8. Recognize blood typing and blood groups (p 248).
9. Understand the proper procedure for collecting blood specimens (p 249).
10. Identify the proper test tubes used for collecting blood specimens (p 249–250).
11. Identify the common errors in specimen collection (p 252).
12. Understand the basics of diagnostic imaging, including the standard radiograph, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound (p 252–253).

Introduction

CCTPs, when performing interfacility transports, are often responsible for patients who have undergone a battery of tests and have had examinations performed at the referring institution. These laboratory tests (sometimes called "labs") can be very useful in determining the seriousness of the patient's condition or aiding in anticipation of potential problems while en route. These tests will include laboratory examinations of the patient's blood, possibly urine, cerebrospinal fluid (CSF), or other body fluid.

Laboratory analysis serves many purposes in the critical care transport environment. A CCTP must be comfortable with not only the normal ranges for each lab value, but also the associated physiologic meaning of the test. Lab tests should not be used to satisfy one's curiosity, "complete the picture" for a particular panel of tests, or reassure concerned family or staff. Today's health care environment mandates a thoughtful and evidence-based approach to ordering tests, especially laboratory work.

Principles of Analysis

Laboratory analysis should run deeper than simply surmising whether the value is outside the normal range for that test. If the normal value range for creatinine is 0.6 to 1.2 mg/dL, for example, it will be clear that a patient whose creatinine value is 4.5 mg/dL is out of the normal range. The crucial thing to know is how this excess level affects the patient now and how it will affect the patient en route to the receiving facility. Certain principles that guide the analysis of all laboratory values must always be

present in a CCTP's mind.

An appreciation of a lab test's **precision** and **accuracy** is essential to the proper use of the particular lab test. If a particular lab test has a high precision, every time the test is performed, the value will be the same. For example, if a patient's serum sodium level was measured five times on the same sample, the values may range from 152 to 155 mEq/L. This test exhibits high precision (tightly packed values), but the results would be inaccurate if the patient's true serum sodium level is 140 mEq/L **Figure 8-1**. A test is considered accurate if the value (or average value) measured conforms to the standard or true value. Precision, on the other hand, is a measure of tolerance or variation within multiple measurements but does not reflect how well the measurements compare with the true value **Figure 8-2**. The scientists who design laboratory tests strive for tests that are precise and accurate.

As with their precision and accuracy, laboratory tests have differing levels of sensitivity and specificity. **Sensitivity** refers to the ability of a test to indicate whether a person does or does not have a certain condition. If a test is highly sensitive, most of the people with a particular condition would have a positive result. If a test has a low sensitivity, many people with the condition would have a negative result. Sensitivity is the proportion of people with the target disorder who have a positive test result. **Specificity**, on the other hand, is the proportion of people without the target disorder who have a negative test result. The **D-dimer test**, a test of hypercoagulability that detects a fragment, d-dimer, from the fibrinolysis process, is used to help diagnose and monitor diseases and conditions related to inappropriate clotting; for example, it can be used to test for deep venous thrombosis (DVT). This test provides a good illustration of sensitivity vs specificity. The presence or absence of DVT was determined by ultrasound (the "standard" in this example). The results are shown in **Table 8-1**.

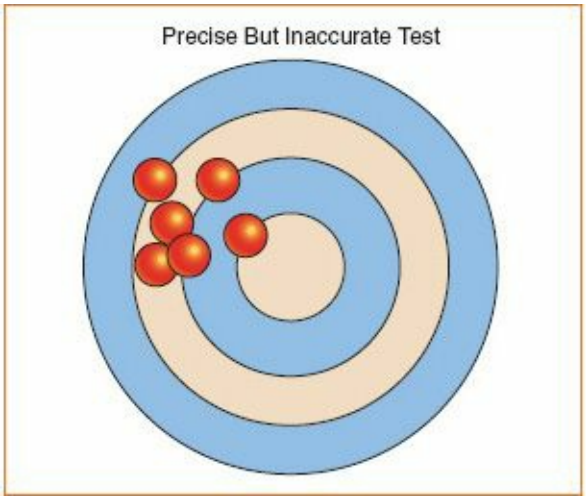


Figure 8-1 Precise (because results cluster together) but inaccurate (because results are out of the normal range [center circle]) lab test.

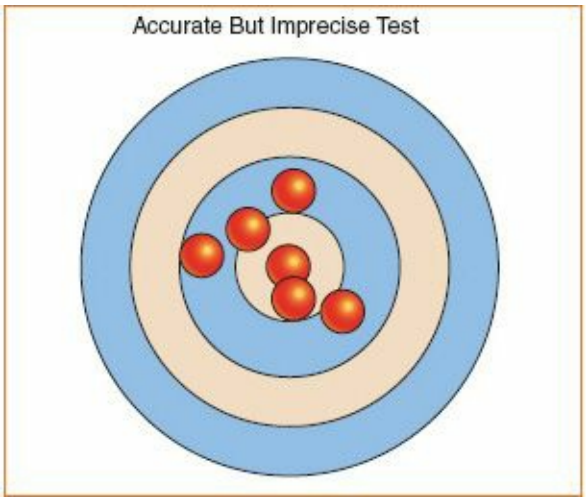


Figure 8-2 Imprecise (because results vary substantially) but accurate lab test.

TABLE 8-1 D-Dimer Test for DVT

D-dimer test result	Target disorder (DVT) present on ultrasound	Target disorder absent (no DVT) on ultrasound	Total
Positive	13	19	32
Negative	1	72	73
Total	14	91	105

Abbreviation: DVT, deep venous thrombosis.

The sensitivity of the d-dimer test for the presence of DVT is calculated by dividing the number of patients with a positive test result by the actual number of patients with the disorder:

$$13/14 = 0.928, \text{ or } 93\%$$

In other words, the d-dimer test will correctly identify a patient with a DVT 93% of the time.

On the other hand, the specificity of the d-dimer test for the presence of DVT is calculated by dividing the number of patients with a negative test result by the actual number of patients without the disorder:

$$72/91 = 0.791, \text{ or } 79\%$$

In other words, the d-dimer test will correctly identify a patient without DVT 79% of the time.

Certain tests lend themselves to certain levels of analysis. For example, often a serologic blood test examining for the presence or absence of the hepatitis B virus will show a result as positive or negative. This is a *qualitative* assessment because the result does not identify the specific level of hepatitis B virus within the blood. If the viral load of hepatitis B is desired, a *quantitative* test is performed to indicate the exact amount of the virus within the blood. Sometimes semiquantitative assessments are performed. For example, a urine test looking for blood in the urine may give the result as none, mild, moderate, or severe.

Novices in laboratory analysis can become overly concerned about the notion of normal vs abnormal lab values. One must remember that normal ranges were identified based on findings in healthy people—not the patients being transported by ambulance. By convention, a **normal range** is empirically derived and represents values that 95% of *healthy* people would have for the particular test. Thus, for every 100 healthy people tested, 5 will not have results that fall in the normal range, even though their particular level is "normal" for them.

When this idea of "normality" is transferred to dealings with patients who have acute diseases, one must be careful not to react too impulsively to spuriously high or low values. Not only is the normal range

for a particular lab test subjective in the emergency department (ED) or intensive care unit context, but an abnormal result may also be the desired effect of a particular treatment. Consider the intentional fluid depletion that is used with patients who have experienced a traumatic brain injury. Part of the therapy may be to decrease the intravascular volume, which will result in abnormally high serum sodium values. Trying to correct the "abnormal" serum sodium value would harm the patient. The message here is simple: Although abnormal lab values cannot be ignored, it is essential that they be put into context of the patient's entire clinical picture.

When dealing with an acute care patient, one must confirm abnormal findings by considering them part of the patient's entire clinical picture. If a particular lab test indicates a certain condition (eg, fluid depletion) but that finding is not confirmed by other tests or by other assessment methods (eg, increased heart rate or decreased blood pressure), the validity of the test or test result in that patient must be reexamined.

With all tests, it is important to keep in mind that errors in specimen collection, identification, labeling, and laboratory analysis can and do occur and may result in erroneous values being reported.

When evaluating laboratory results, it is critical to recognize that different laboratories have different normal ranges for the same tests. In fact, readers may find that many of the normal ranges listed in this text are different from the ranges they are accustomed to using in their local practice. It is likely that three different labs would have three different sets of normal ranges for each test they perform. It is essential to give careful attention to the normal values (indicated on report forms) when reviewing lab reports.

Specimen Cultures

Blood, urine, sputum, and other body fluid cultures provide information that is used to identify microorganisms and treat specific infections. For example, a sputum culture is obtained when a respiratory infection is suspected. It requires a conscious patient to be well hydrated and able to follow commands to produce sputum from a deep cough. An unconscious patient may be deep suctioned to obtain a sample. If a sample is not actual sputum, false results are likely, identified on the laboratory report as "mixed flora"; such results are not useful.

Sensitivity testing can be done as well (culture and sensitivity testing is sometimes called C and S). For bacterial infections, the sensitivity results indicate which antibiotic will most effectively treat the organism causing the infection. A specimen for culture should be obtained before antibiotic therapy is started so that the antibiotic does not influence the culture result and the proper antibiotic (one to which the microorganism is sensitive) can be selected. Usually, it takes 3 days for a laboratory to complete a culture and sensitivity report; an initial report is generated in about 24 hours, whereas a complete and final report will not be available for 72 hours.

Chemistry Review

Many consider the care of an acute patient to be akin to an advanced experiment in physiology. Physiology is built on the basics of chemistry and cellular biology, the principles of which should be part of the health care provider's educational background. Insightful test interpretation requires a firm grasp of these principles.

Ions

Ions are atoms that have gained or lost electrons. Recall that each electron has a single (–1) negative charge. If electrons are lost, the charge on the atom is "less negative," or the atom is now "more positive." The reverse is true if electrons are gained by the atom. A positively charged ion is referred to

as a **cation**, whereas a negatively charged ion is called an **anion**. If one electron is deleted from an atom, its charge is +1. If two electrons are deleted, its charge is +2. Conversely, if one electron is added to an atom, its charge is –1. If two electrons are added, its charge is –2. Often ions of opposite charge will join together with an **ionic bond**. Sodium chloride (NaCl—table salt) is an example of a molecule that is joined by an ionic bond. When molecules with ionic bonds interact with water (remember that plasma is 90% water), they often dissociate into the basic ionic forms **Figure 8-3**. This is why Na^+ and Cl^- exist as separate ions within plasma, rather than as the compound NaCl. The ionic components of plasma constitute the electrolytes, which include sodium (Na^+), potassium (K^+), chloride (Cl^-), calcium (Ca^{++}), and magnesium (Mg^{++}), among others.

Amounts of ions are expressed in moles or equivalents. A mole is a unit representing 6.02 × 1023 atoms, in much the same way that a "dozen" is used to specify 12 eggs. Equivalents (Eq) are used to measure amounts of charged particles (ie, ions). Essentially, 1 Eq is equal to 1 mole of ionic charges. Given that a single Na^+ atom has one charge, 1 mole of Na^+ atoms has 1 mole of charges (1 Eq). Similarly, calcium (Ca^{++}) has two charges, so 1 mole of Ca^{++} has 2 moles of charges (2 Eq). This concept applies only to charged particles; uncharged molecules, such as proteins, are electrically neutral overall.

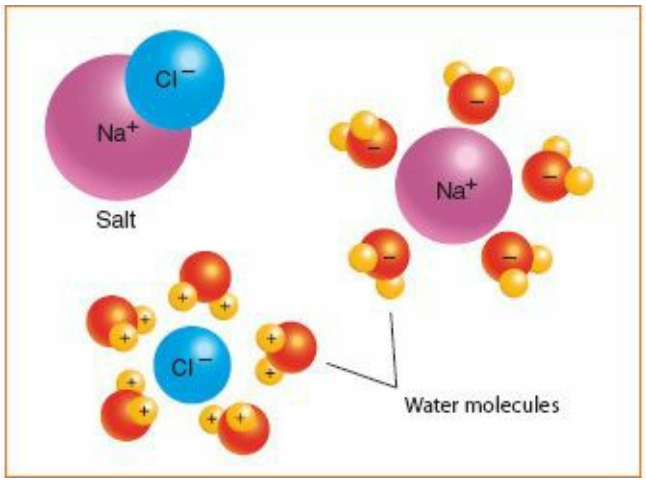


Figure 8-3 The ionic bonds of sodium chloride dissociate into their basic ionic forms when they interact with water.

Osmolarity

Laboratory testing often involves the analysis of certain fluid characteristics. One characteristic of interest to CCTPs is the **osmotic pressure** created by the blood, CSF, or urine sample. Because human cells are semipermeable membranes, **osmosis** (the diffusion of water across membranes) is a major mechanism of fluid movement between the body's different fluid compartments. The osmotic pressure is created in the space divided by the semipermeable membrane owing to differences in concentrations of solutes (dissolved substances) found in the solutions on either side of the membrane.

The osmotic pressure generated by the particles in the fluid is called the osmolarity or osmolality, depending on the method of measurement. Physiologic measurement of osmotic pressure in humans is measured as osmolality, often mislabeled as osmolarity. **Osmolarity** measures the amount of dissolved substance in 1 L of water, whereas **osmolality** measures the amount of dissolved substance in 1 kg of water. Because the volume (liter) of a fluid depends on the temperature of the fluid, osmolality is used because the weight (kilogram) of fluid is independent of temperature. The unit of measurement for osmolality is the osmole (Osm), which is the pressure created by 1 mole of particles in solution.

It is important to note a particular chemical property of osmosis: osmotic pressure depends only on the number of particles in the fluid, not on the size of the particles. A single protein or sugar molecule

induces the same osmotic pressure as a single ion of sodium (Na^+).

Ultimately, we want to know the amount of a particular ion or protein that is present in the sample. This amount is expressed as a **concentration**, which is the amount of the substance present in a given volume of fluid. For example, a patient's Na^+ concentration may be expressed as mmol/dL (millimoles of Na^+ per deciliter of blood). Because Na^+ has a single charge, the Na^+ concentration may also be expressed as mEq/L (milliequivalents of Na^+ per liter of blood). In the physiologic system, most concentrations are incredibly small. As a result, units of 1/1,000 (milli-) are often used to describe concentrations, including millimoles (mmol), milliequivalents (mEq), and milliosmoles (mOsm).

Laboratory values are reported in conventional units (such as milliequivalents per liter) or in Système International d'Unités (SI units; such as millimoles per liter). Many laboratories use SI values for some tests and conventional units for others, or they might use only conventional units. When reviewing results for a patient, CCTPs need to be sure that the reported units of measure are the same as the units of measure (that is, both conventional or both SI) given in the normal range.

CCTPs need to be aware that each laboratory has its own normal ranges that usually are printed on the laboratory report with the patient's values. In addition, normal ranges may differ from one instrument manufacturer to another. CCTPs should be familiar with the normal ranges for tests often used in the critical care transport setting, and they should be alert to normal ranges listed for all tests used for a specific patient, especially when comparing results from different facilities.

Biochemistry Review

Examining the basic ions and fluid properties provides only part of the story. To ascertain how well an organ (such as the liver) is functioning, it is necessary to look at various proteins and enzymes in a serum sample. Thousands of different kinds of proteins reside in the human body, and specific laboratory tests examine the amount of the various types of proteins present in a sample.

Enzymes are critically important proteins that act as catalysts for biochemical reactions. The formation of one biologic substance from another may proceed slowly when just the two substances are present; with the addition of the appropriate enzyme, however, the reaction rate may increase many times. Methods of measuring enzyme levels often rely on this basic catalytic principle. The beginning substrate (material) for the reaction is added to the sample, and the amount of product generated in a given time is measured. By knowing the beginning amount of substrate and the amount of product produced and by assuming normal enzyme function, one can then calculate how much enzyme is present. The function of enzymes is measured in units per liter (U/L), which is the amount of enzyme that catalyzes 1 micromole (μmol) of substrate per minute.

Lab Profiles

In laboratory testing, groups of related tests can be performed as a single unit, called a **panel** (or profile). The single unit comprising these related tests is often named for the tests' common link. For example, a liver panel consists of a set of tests that examine liver function, and a basic electrolyte and metabolite panel may be called a Chem-7. In a patient's hospital chart, these panels are written in the form of a matrix.

Lab Values in Blood Samples

Examination of the electrolytes and metabolites in the extracellular fluid constitutes one of the most basic and fundamental assessments done in the ED or intensive care unit. Often this is done as a standard panel

as mentioned earlier: the basic metabolic panel (or Chem-7) **Figure 8-4**.

Sodium

Sodium (Na^+) is the major extracellular ion; its serum concentration in a healthy person ranges from 136 to 142 mEq/L (mmol/L). This contrasts with intracellular concentrations of Na^+, for which the normal range is only 3 to 20 mEq/L. Dramatic changes in a patient's serum sodium level are possible and are primarily due to changes in extracellular water concentrations. As a patient becomes depleted of free (solute free) water, the serum sodium concentration increases (more sodium ions per liter of water). The introduction of excess extracellular water (without additional sodium) will, therefore, decrease serum sodium levels.

Serum sodium is a convenient marker for a patient's fluid status and is one of the key components of the serum osmolality calculation explained later in this chapter. Unlike with potassium derangements (discussed later), an abnormal sodium level will not manifest with electrocardiographic (ECG) changes. Most signs and symptoms revolve around neurologic sequelae owing to changes in osmolality.

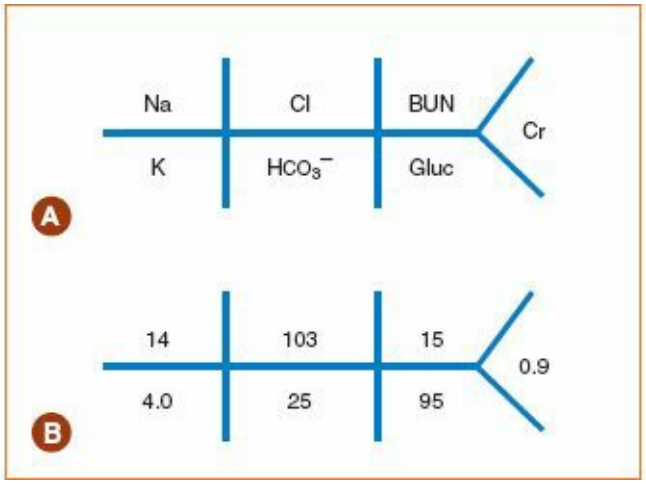


Figure 8-4 **A.** The items measured in a basic metabolic panel, shown in the format in which values are written. **B.** An example of how the values would be written, with each slot representing a particular lab value. These values are for a healthy person.

Elevated sodium levels are not uncommon in patients who are undergoing critical care transport. An elevated sodium level in a patient with a traumatic brain injury is likely a consequence of treatment at the referring hospital. Diuretics and restricted fluid intake are used in an effort to cause the excretion of more water than is taken in—essentially "drying out" the patient. This treatment is aimed at lowering intracerebral pressure. The serum sodium level in patients with traumatic brain injury can often exceed 170 mEq/L.

Hyponatremia (an abnormally low level of sodium in the blood) often results from an excess of free water or excessive sodium depletion. It can be found in patients with congestive heart failure, renal failure, or liver disease and in patients undergoing diuretic therapy. Although some debate exists about how fast a hyponatremic state should be corrected, slower (0.5 mEq/L per hour) is better to avoid causing subsequent neurologic problems.

Patients with a sodium imbalance (high or low) need to be monitored for intake and output, including oral and IV input. A sodium level of less than 125 mEq/L can result in behavioral changes, confusion, delirium, increased respiratory rate, muscle twitching, increased intracranial pressure, and cardiac abnormalities. Increased sodium levels can cause fluid retention and cardiac abnormalities.

Potassium

Whereas sodium is the major extracellular cation, potassium (K^+) is the major intracellular cation. The extracellular K^+ concentration (as typically measured in blood) is normally 3.5 to 5.0 mEq/L.

Of primary concern is the effect of an elevated K^+ level on cardiac cells, with cardiac arrhythmias occurring with **hyperkalemia** (an abnormally high level of potassium in the blood). A potassium level of 5.5 to 6.5 mEq/L can result in the classic peaked T waves, with flattened P waves occurring when the potassium level exceeds 7.0 mEq/L. Derangements in other electrolytes (for example, hypocalcemia, hypomagnesemia, and hyponatremia) can exacerbate hyperkalemia. Hyperkalemia in an acute care patient can occur as the result of excessive potassium supplementation, intracellular to extracellular fluid shifts with cellular lysis (seen with crush injuries and tissue necrosis), drug administration (angiotensin-converting enzyme inhibitors, for example), metabolic acidosis, and decreased excretion, as might occur in acute renal failure. CCTPs should be aware that many abnormally high potassium levels are actually reported in error—for example, cellular lysis (hemolysis) can cause an elevated potassium level, so if the sample is hemolyzed before analysis, an incorrect value may be reported.

Causes of **hypokalemia** (an abnormally low level of potassium in the blood) include cellular shifts (for example, as the result of insulin administration or hypothermia) or increased excretion of potassium (gastrointestinal [GI] tract or renal losses). Although younger patients may have a higher tolerance for decreased levels of potassium, older patients (especially patients taking a digitalis preparation) are more likely to have arrhythmias and ECG changes with low K^+ levels.

Low K^+ levels result in muscle pain, hyporeflexia, nausea, vomiting, and orthostatic hypotension. High K^+ levels can cause cardiac abnormalities, particularly atrioventricular and intraventricular blocks. Atrial arrest is likely when the K^+ level reaches 9 mEq/L.

For any patient with a cardiac history, altered renal function, liver disease, GI disturbances, or who is receiving insulin, a recent K^+ level should be available for review before transport.

Special Populations

Older patients with decreased potassium levels, especially those taking digitalis preparations, will have cardiac changes. Hyperkalemia can indicate digitalis toxicity.

Chloride

Chloride (Cl^-) is the major extracellular anion. Its single electrical charge is responsible for offsetting the positive charge of Na^+ and K^+, thereby maintaining electrical neutrality within the body. Serum concentrations range from 96 to 106 mEq/L in healthy people. Chloride is an important tool that the kidney uses to concentrate urine. Patients with **hypochloremia** (an abnormally low level of chloride in the blood) may have impending renal dysfunction. Care should be used with patients receiving diuretic therapy because they may also have abnormally low chloride levels. Strangely, patients with excess diuresis can also exhibit **hyperchloremia** (an abnormally high level of chloride in the blood).

In practice, chloride levels mimic sodium levels; when hypernatremia is present, hyperchloremia often is as well. Clinically, serum chloride levels help differentiate certain types of metabolic acidosis.

High chloride levels can result in circumoral numbness and tingling, muscle hypertonicity, decreased respiratory rate and depth, possible complaints of nervousness, and signs of central nervous system stimulation.

Carbon Dioxide

One of the most basic indications of the acid-base status of a patient is the *venous* bicarbonate level.

Confusing as it may seem, the carbon dioxide level (CO_2) is not a CO_2 level at all; rather, this test indicates the amount of bicarbonate (HCO_3^-) in the venous sample. Carbon dioxide and bicarbonate are in equilibrium according to the following equation:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Thus, CO_2 and HCO_3^- are related, although they are actually very different substances. Carbon dioxide is a gas, so when it is measured, the value should be expressed as a partial pressure. Bicarbonate is an ion and, like the other ions mentioned earlier in this chapter, the value is expressed as a concentration. A level below the normal range could indicate metabolic acidosis or respiratory alkalosis, whereas an elevated level could indicate metabolic alkalosis or respiratory acidosis. The normal HCO_3^- value is 21 to 28 mEq/L, and the normal CO_2 value is 22 to 28 mEq/L.

Blood Urea Nitrogen

A product of protein catabolism, urea is related to the amount of protein intake, protein metabolism, and rate of excretion. Urea is considered a useful marker for adequate kidney function because its level in kidney filtrate is often the same as in serum. The normal range for **blood urea nitrogen (BUN)**, which is the test used to measure urea, is 8 to 23 mg/dL. The BUN level tends to increase with age as a consequence of gradually declining renal function; a slightly elevated BUN finding of 28 to 35 mg/dL in older patients should not necessarily be a cause for concern owing to the slowing of renal function. Concern for an elevated BUN level in an elderly patient may vary depending on the results of other lab tests and the findings of the patient assessment. Any value of more than 40 mg/dL should be monitored closely.

When patients have high renal output levels, BUN values are a less valid means of assessing kidney health because of the inverse relationship between urine formation and urea reabsorption. Elevated levels of urea occur not only with decreased renal function, but also with consumption of a high-protein diet and in the presence of a high-protein catabolism state (for example, burns and crush injuries).

Creatinine

A major storehouse of intramuscular high-energy phosphate, **creatine** is degraded to **creatinine (Cr)** (a chemical waste product that results from muscle metabolism) at a relatively steady rate by the muscles. Within one person, the rate of Cr production typically varies by only 10% at any particular time. Creatinine is filtered from the blood by the kidneys and, without reabsorption, is excreted in the urine. Because of its steady rate of production, this by-product of metabolism can be used to assess kidney function. If urine function decreases, serum Cr levels rise above normal (normal range, 0.6 to 1.2 mg/dL).

An abnormal serum Cr level does not indicate exactly what the disease or cause is, but it does indicate some level of decreased renal function. Under certain circumstances, a patient may have an increased Cr level with adequate renal function, such as when the patient has had a large amount of muscle damage (owing to protein release from trauma, called rhabdomyolysis). Care must be taken when a geriatric patient has a Cr level slightly more than the upper normal value (near 1.5 mg/dL): Owing to the proportionately smaller amount of muscle mass and consequent lower Cr production in older people, a small elevation in the Cr level may reflect greater kidney dysfunction than would the same level in a younger patient.

If the cause of the elevated Cr level is not identified and corrected, damage to the kidneys will be permanent, requiring lifetime dialysis or kidney transplantation.

Creatinine clearance is identified as the most accurate measure of the glomerular filtration rate. The results of a Cr clearance test can be used to determine long-term management of patients with chronic

disorders of renal function and to calculate appropriate fluid and electrolyte replacement therapy. Creatinine clearance is calculated by multiplying the concentration of measured urine Cr by the volume of urine during a set time and dividing the product by the serum concentration of Cr. One test result does not give a great deal of information. However, serial results can be very useful in determining a patient's course of therapy.

Special Populations

Beware of geriatric patients with creatinine levels approaching 1.5 mg/dL. They may have more kidney damage than might otherwise be suspected, owing to the relatively smaller amount of muscle mass present, such that less creatinine is produced.

Glucose

Glucose is the most important carbohydrate within the body and is the lab measurement with which CCTPs may be most familiar. Glucose is commonly assessed in the field with a point-of-care testing device, and prehospital care providers often measure the blood glucose level of unconscious or semiconscious patients. Concentrations of glucose are normally in the range of 70 to 110 mg/dL. Maintaining a normal glucose level is an extremely important activity for CCTPs because patients may be receiving insulin drips or may have acute renal failure, which will affect the blood glucose level. If a patient is receiving an insulin drip, adjustments to the drip may be required if the patient's glucose level is steadily dropping. If the glucose level rises in a patient with renal failure, hypovolemic shock may occur.

High glucose levels that are not identified and corrected can potentially result in coma and death. Low glucose levels are generally easier to recognize early because of symptoms such as blurred vision, dizziness, nausea, vomiting, and shakiness. If left untreated, the patient can become unconscious, possibly injuring himself, herself, or someone else.

Total Calcium

Calcium is one of the most essential electrolytes found in the body. It is responsible for functions from muscle contraction to intracellular signal transduction. Physiologically, calcium is found in three states: free, chelated, and bound. The largest portion of calcium (47%) is found as a free calcium ion dispersed in the body fluids. Another 43% of calcium in the body is bound to proteins, mostly albumin. Naturally, the amount of calcium bound to proteins depends on the ability of the protein to complex with the calcium ion (Ca^{++}). Protein binding is a function of the shape of the protein, which in turn is heavily dependent on the surrounding pH. In abnormal pH states, the amount of calcium bound to proteins decreases, and, therefore, the amount of free calcium increases. Calcium that is chelated (bound) to other molecules, such as citrate, bicarbonate, lactate, phosphate, and sulfate, represents only 10% of the calcium in the body.

The sum of all calcium in the body is expressed as the total calcium level (Ca^{++}_{TOT}). The normal range for Ca^{++}_{TOT} is 8.2 to 10.2 mg/dL. The Ca^{++}_{TOT} is increased in elevated parathyroid hormone states such as hyperparathyroidism and parathyroid-secreting tumors. Low levels of Ca^{++}_{TOT} are seen with renal insufficiency, hypomagnesemia, and hyperphosphatemia and in patients who have received a massive blood transfusion or have decreased parathyroid hormone states.

Ionized Calcium

Only the calcium that is not bound or chelated (ie, free calcium) is physiologically active. In states in

which there are altered fractions of bound or chelated calcium, free calcium assessment is warranted. Examples of physiologic states that warrant assessment of **ionized calcium** include renal failure or nephrotic syndrome (hypoalbuminemia), acid-base derangements (particularly acidosis), and decreases or elevations in chelating compounds (citrate, bicarbonate, lactate, phosphate, and sulfate). The normal range for ionized calcium is 4.60 to 5.08 mg/dL.

Low levels of ionized calcium can cause serious arrhythmias and are especially pronounced in prolonged cardiac arrest. Although calcium administration has not been shown to be of long-term benefit to patients who have had cardiac arrest, its use for treatment may be warranted in patients with hyperkalemia, hypocalcemia, or toxic levels of calcium-channel blockers. CCTPs may find that patients with one of the aforementioned conditions are transported with a magnesium sulfate drip to decrease the likelihood of further cardiac arrhythmias or cardiac arrest.

Magnesium

Magnesium (Mg) levels can be affected by several body systems, in particular the GI and endocrine systems. The normal serum magnesium level is 1.3 to 2.1 mEq/L. High Mg levels are rarely encountered. Generally, the causes are renal defects, severe dehydration, overadministration of Mg, untreated diabetic coma, and aspiration of sea water. Low Mg levels are more commonly seen, as in GI distress, vomiting and diarrhea, hepatic cirrhosis, and pancreatitis.

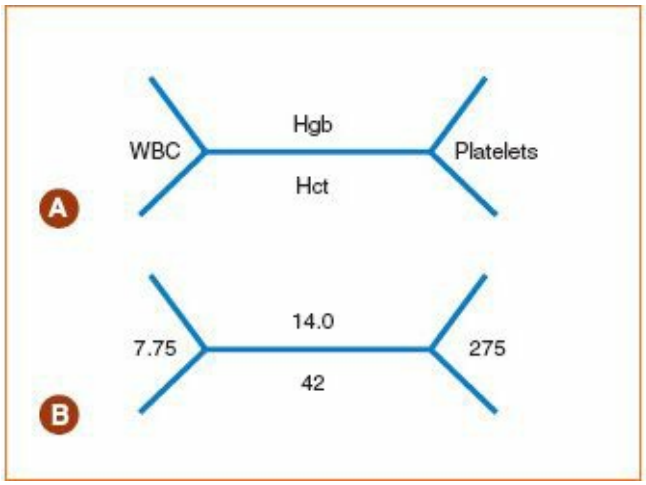


Figure 8-5 Complete blood count schematic (abbreviated notation). **A.** The format in which values are written. **B.** An example of values for a healthy person.

Blood Components

Figure 8-5 shows the schematic for abbreviated notation of a complete blood count (CBC) test. The schematic is intended to be used for quick reference of pertinent values. It is presumed that if one of these values is abnormal, the CCTP would go to the more complete medical record to ascertain other values.

Hematocrit

Hematocrit, sometimes abbreviated Hct, is one of the more general laboratory assessments. Its sensitivity for disease is very high, but its specificity is rather low. Thus, if a patient's hematocrit value is out of the normal range, disease may be indicated, but many different diseases or conditions could cause the abnormal value.

Hematocrit is the percentage of formed elements (that is, cells) in a venous blood sample. A hematocrit value of 45% indicates that 45% of the sample consists of cells or cellular debris, and the other 55% consists of plasma. The nature of this relationship leads to the lack of specificity for this test. For example, with an acutely ill patient, the health care provider is often concerned with a low hematocrit

value. A low level of formed elements, which are predominantly red blood cells, can indicate decreased capacity of the blood to deliver oxygen to the tissues. A patient who has an excess amount of plasma (such as from overzealous use of crystalloids) will also have a low hematocrit value. The normal range for hematocrit is 41% to 50%. For example, a patient with a hematocrit value of 22% could be exsanguinated or fluid overloaded **Figure 8-6**. Therefore, for a patient with an abnormal hematocrit level, further investigation must be done to identify the precise cause of the abnormality.

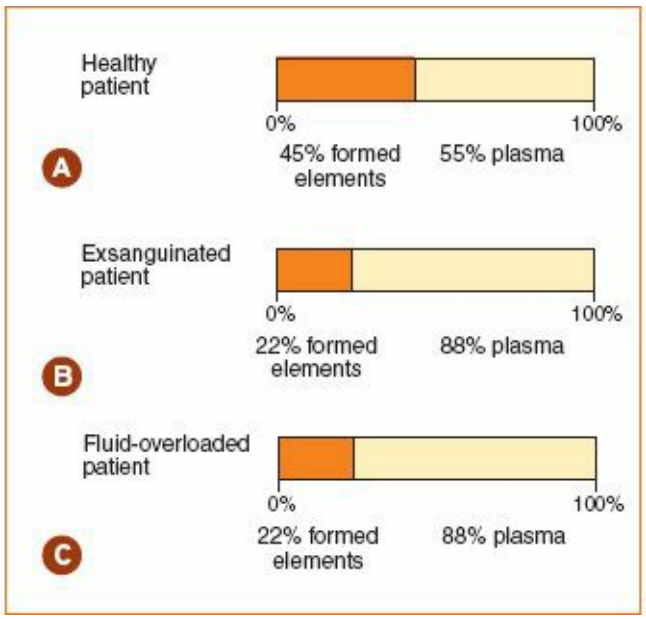


Figure 8-6 Examples of hematocrit levels and (B and C) potential causes. **A.** Healthy patient. **B.** Exsanguinated patient. **C.** Fluid-overloaded patient.

Hemoglobin

Hemoglobin (Hgb) is the protein responsible for carrying oxygen to the cells and, to a lesser extent, carbon dioxide back to the lungs. The abbreviations Hg and Hb are also used for hemoglobin. The level of hemoglobin varies by gender, with normal values falling in the range of 135 to 175 g/L (14.0 to 17.5 g/dL) for males and 120 to 160 g/L (12.0 to 16.0 g/dL) for females. Elevated levels are seen in people with **hemoconcentration** (decreased fluid in the blood, which means that concentrations of other blood components increase), which can result from dehydration, burns, or excessive vomiting. Of greater concern are low Hgb levels, which are typical of most types of anemia (microcytic, normocytic, and macrocytic).

Carboxyhemoglobin

Because carbon monoxide binds with hemoglobin irreversibly, the body's ability to transport oxygen can become extremely compromised when too much CO is present in the body. Although some **carboxyhemoglobin (COHb)**, a hemoglobin derivative, is always present in the vasculature, normal levels are thought not to exceed 0.02 (2%). The percentage is based on the amount of total hemoglobin. Assessment of COHb levels is useful in confirming a diagnosis of carbon monoxide poisoning, and it may be used to guide therapy as well. Caution should be used in assessing COHb levels in smokers, as their baseline COHb levels are normally higher than non-smokers. COHb levels must be requested specifically from the lab because the specimen is processed in a particular manner.

Red Blood Cell Count

The number of erythrocytes, or red blood cells (RBCs), per microliter of blood constitutes the RBC count. The normal range for **red blood cell (RBC) count** is 3.9 to 5.5 × 106/μL. Although an abnormal

number of RBCs does not necessarily mean the presence of disease, the RBC level can be elevated with hemoconcentration. Patients with elevated white blood cell counts may have an erroneously high RBC level owing to analysis errors. As expected, low levels are also seen in all types of anemia.

Leukocyte Count

The **white blood cell (WBC) count** is a measure of the total number of leukocytes (WBCs) in the blood. Because an "ordinary" WBC count does not differentiate among the various types of leukocytes in the sample, only so much clinical information can be inferred from this test. The normal range for WBC counts is between 4,500/µL and 11,000/µL. Low levels of WBCs are seen in patients with certain anemias (such as aplastic anemia), vitamin deficiencies, or sepsis. Although it might seem counterintuitive, low levels are also seen in sepsis: WBCs are mobilized to fight the infection and are ultimately destroyed at a rate that exceeds their production. **Leukocytosis** (elevated WBC count) is seen not only with inflammation or infection, but also in certain malignancies (leukemia and lymphoma) and vascular conditions (such as pulmonary embolism, acute myocardial infarction [AMI], and DVT), with steroid administration, and with the stress of trauma.

Platelet Count

Given that platelets fulfill essential roles in clot formation, the **platelet (Plt) count** is useful in assessing a patient's coagulation status. The normal range for a whole blood sample is 150 to 350 × 103/µL. Elevated levels of platelets are seen in patients with certain myeloproliferative disorders (a group of diseases in which the bone marrow produces too many RBCs, WBCs, or platelets; examples include polycythemia and chronic myelogenous leukemia), most notably after severe bleeding or following splenectomy. Decreased levels may occur when disease causes decreased production in the bone marrow. Often **thrombocytopenia**—an abnormally low level of platelets—is due to splenomegaly, disseminated intravascular coagulation (DIC), or high circulating levels of platelet antibodies (such as after massive transfusions).

Proteins

Total Protein

One of the most basic protein tests, **total protein**, examines the total quantity of protein in a blood sample. This quantity consists of mostly albumin and immunoglobulins. The normal range for a serum sample of total protein is 6.0 to 8.0 g/dL, and variations are almost always due to fluctuations in serum albumin levels. As a consequence, most clinicians focus on albumin levels.

Albumin

By far the most common protein in the body is **albumin**. Albumin serves many functions: It acts as a transport protein (for free fatty acids, bilirubin, hormones, and drugs), it is a free-radical scavenger, and it serves as the main source (70%) of protein-generated oncotic pressure. This is the osmotic pressure that allows for colloids to move from one side of the cell membrane to the other as needed. The normal range for albumin is 3.5 to 5.0 g/dL.

A low level of albumin may be due to increased catabolism of the protein (as seen in malnutrition), decreased production, or edema in the spaces between the cells in the tissue. Decreased production is seen in patients who have liver damage or other liver disease. **Hypoalbuminemia** (an abnormally low level of albumin) from increased interstitial sequestration can occur in high-vascular-permeability states and leads to diseases such as acute respiratory distress syndrome. High levels of albumin generally indicate only dehydration and are not pathologic.

C-reactive Protein

C-reactive protein (CRP) is an acute phase protein synthesized in the liver. It is an indicator of inflammation that begins to rise four to six hours after tissue injury. The normal range is 0.08 to 3.1 mg/L. A CRP level above 10 mg/L indicates significant inflammatory disease.

Myoglobin

Myoglobin is an oxygen-carrying heme protein present in high concentrations in the cytoplasm of cardiac and skeletal muscles. In cardiac patients, you may see an increase within two hours after the onset of chest pain. A positive myoglobin result after chest pain warrants further investigation; however, a negative myoglobin result can be used in ruling out a myocardial infarction. The normal range is 19 to 92 μg/L.

Lactate Dehydrogenase

In the absence of a functioning citric acid cycle (discussed in Chapter 9), pyruvate (the end product of glycolysis) is metabolized to lactate. The enzyme catalyzing this reaction is **lactate dehydrogenase (LDH)**. Found in almost all tissues of the body, the total form—LDH—is not sensitive or specific for any disease. The normal range of LDH is 100 to 200 U/L. Its usefulness in the clinical setting comes from analysis of its isoenzyme forms (LD_1 to LD_5). Various organs, tissues, and cells differ in terms of the amount of each isoenzyme they possess. For example, LD_1 is found largely in the heart, kidneys, and RBCs. Other tissues, such as skeletal muscle and the liver, contain relatively high amounts of LD_5.

Determination of the amount of each isoenzyme present may be used to aid in the diagnosis of cellular damage. In cases of liver disease, congestive heart failure, or skeletal muscle damage, the LD_5 level may be increased. The primary point of analysis of the LDH isoenzymes is to calculate the LDH_1/LDH_2 ratio. The normal range for LD_1 is 17% to 27%; and for LD_2, 27% to 37%. A ratio of more than 85% indicates possible AMI. This finding is useful if the AMI occurred more than 24 hours earlier or if the results of testing for the creatine kinase MB fraction are inconclusive. The LD_1/LD_2 ratio will often rise to levels exceeding 85% within 24 to 48 hours after myocardial injury. The LDH value was once considered useful to aid in the diagnosis of AMI but has been replaced in routine use by troponin analysis (discussed later in this chapter). Lactate dehydrogenase can also be helpful in assisting with the diagnosis of *Pneumocystis carinii* pneumonia and determining the severity of pancreatitis.

Creatine Kinase

Creatine kinase (CK) is found in muscle, liver, lung, GI, brain, kidney, and spleen tissues. If any of these tissue cells become damaged, CK is released into the vascular space. Elevated levels are indicative of muscle damage, which may or may not be specific to AMI; concomitant muscular tissue damage also leads to elevated CK levels. With myocardial damage, CK levels rise within 4 to 8 hours, peak within 12 to 24 hours, and return to normal within 2 to 4 days. The normal range for the total CK level is 40 to 150 U/L.

The MB fraction of CK, or CK-MB, refers primarily to CK in the heart muscle. The normal range for the CK-MB level is 0 to 7 ng/mL. With a level of less than 10 ng/mL, a myocardial event is unlikely. Results ranging between 10 and 12 ng/mL are not conclusive, and additional workup and serial lab tests are required. Results of more than 12 ng/mL are indicative of a myocardial event.

Troponin I

Troponin is a key protein involved in muscle contraction. This protein has three subunits: T, C, and I. The I subunit has three separate isoforms, one of which is found only in cardiac muscle. Troponin is expressed in the serum *only after* cellular necrosis releases the respective cellular contents. Thus, measurement of the troponin levels offers some advantage over measurement of the CK and LDH levels because CK and

LDH are normally present in the serum. The normal range for cardiac troponin I (often abbreviated cTnI) is 0 to 0.04 ng/mL, but elevations following myocardial injury are detectable in a serum sample after 4 hours; tests for cTnI are 97% sensitive and 95% specific at 6 hours after an AMI. Peak levels are expressed 8 to 12 hours postinjury, but the protein may be still be detected 5 to 7 days later. Not only is the cTnI level useful in detecting AMI, but it has also been shown to be predictive of adverse outcomes in patients with severe unstable angina. Normal values for the various troponins depend on the method used to do the test, but, in general, the normal range is 0 to 0.4 ng/mL for troponin I and 0 to 0.1 ng/mL for troponin T.

B-type Natriuretic Peptide

The **B-type natriuretic peptide (BNP)** level can be indicative of abnormal ventricular function and congestive heart failure if it is more than the normal range. The normal value for BNP is less than 167 μg/mL.

Aspartate Aminotransferase

Although not truly tests of liver function, elevated **liver function test (LFT)** results are indicative of liver damage. These tests measure enzymes that normally appear, to some extent, in liver cells. When the liver is damaged, these enzymes often spill out into the vasculature. These tests include aspartate aminotransferase, alanine aminotransferase, total bilirubin, direct bilirubin, and alkaline phosphatase.

Found in large amounts in the liver, the intracellular enzyme **aspartate aminotransferase (AST)**, previously called SGOT, is also found in skeletal muscle, the brain, RBCs, and the heart. Damage to cells of any of these organs causes them to release their contents, which can then be detected in a blood sample. Low levels of AST are not of serious consequence, considering that the normal range for this enzyme is 10 to 30 U/L. Elevated levels are seen in liver damage, especially with acute conditions such as acute hepatitis or biliary tract obstruction. Chronic liver damage (due to alcoholic cirrhosis, hepatitis, and liver cancer) also produces elevation of the AST level, although not as much as that of the **alanine aminotransferase (ALT)** level (described in the next section). Elevations in AST may occur in a setting of right heart failure, hypoxia (global or endorgan), or extensive trauma.

Alanine Aminotransferase

Like its counterpart AST, ALT, previously called SGPT, is found in large amounts in the liver, kidney, skeletal muscle, and heart. Similar to the findings with AST, the normal range for ALT is 10 to 40 U/L, with low levels considered to be of no consequence. The causes of an elevated ALT level mirror the causes of an elevated AST level, with the exception that the ALT level is higher than the AST level in acute processes and lower than the AST level in chronic processes. The ALT test is considered slightly more specific than the AST test for hepatic injury because fewer organs have ALT than have AST. The ratio of AST to ALT is an important diagnostic tool and is explained further in the calculation section in this chapter.

Although AST and ALT are found in the heart, neither is valid for diagnosing myocardial infarction. Elevated levels of ALT may be prolonged because this enzyme has a long half-life (between 36 and 60 hours). As a consequence, levels may appear elevated even after resolution of the hepatic dysfunction.

Total Bilirubin

All RBCs are metabolized eventually, with bilirubin being one of the by-products of their breakdown. This by-product is unconjugated and, therefore, not water soluble (**indirect bilirubin**). When bilirubin is conjugated in the liver, it becomes **direct bilirubin**, which is ultimately excreted in the bile. The normal range for the total bilirubin level is 0.3 to 1.2 mg/dL. A measure of liver health, the total bilirubin level (direct + indirect) is often elevated in patients with liver disease. Other causes of elevated levels of total

bilirubin include biliary tract obstruction and RBC hemolysis.

Direct Bilirubin

In the medical laboratory, bilirubin may be fractionated to indicate the respective levels of unconjugated (indirect) and conjugated (direct) bilirubin. Although such tests are unreliable at times, the cause of elevated total bilirubin levels may be derived from the fractionated levels. The indirect bilirubin fraction is often elevated in conditions when massive **hemolysis** (as in massive blood transfusions or blood transfusion reactions) has occurred. If only the direct or indirect bilirubin level is reported, the other value may be obtained by subtracting the reported level from the total bilirubin level. The normal values for direct and indirect bilirubin are 0.1 to 0.3 mg/dL and 0.2 to 0.9 mg/dL, respectively.

Alkaline Phosphatase

Alkaline phosphatase is found in almost all body tissues and is manufactured by the bone, liver, intestine, and placenta. It is essential for proper digestion and absorption through the mucous membrane of the GI tract. It is clinically useful for testing liver function and, in particular, for diagnosing a common bile duct obstruction. The normal range is 30 to 120 U/L.

Amylase

A key enzyme used by the body to metabolize carbohydrates, **amylase** is produced by the salivary glands and the pancreas. Tissues such as the ovaries, small and large bowels, and skeletal muscle also produce small amounts of amylase. The normal range for amylase is 27 to 131 U/L. Tests for amylase are used to assess for pancreatic insufficiency or damage: Levels of this enzyme are elevated not only with pancreatic disease (such as pancreatitis, pancreatic cancer, and diabetic ketoacidosis), but also with bile duct obstructions and head trauma. A sign of pancreatic insufficiency, low amylase levels are seen in people with cystic fibrosis.

Lipase

The pancreatic enzyme **lipase** metabolizes lipids. Generally considered more specific than amylase for identifying pancreatic disease, lipase is also produced by the liver, intestine, and stomach. The normal range for lipase is 31 to 186 U/L. In acute pancreatitis, the levels of lipase and amylase are elevated, but the lipase level stays elevated longer. Although measurements of lipase levels have poor sensitivity in terms of identifying chronic pancreatitis and pancreatic cancer, elevated levels are often seen with both diseases. Like amylase, lipase is prone to elevation in the case of bile duct obstruction or biliary disease.

Coagulation

Assessment of the coagulation system involves looking at the intrinsic and extrinsic pathways of the coagulation cascade, shown in Chapter 9. The intrinsic pathway (initiated within the body, such as by platelet damage) begins with the activation of factor XII and then factors XI and IX, ultimately resulting in the activation of factor X and initiating the common pathway of coagulation. The extrinsic pathway (initiated outside the body) following tissue injury begins with tissue factor and factor VII, and leads to activation of factor X, initiating the common pathway of coagulation. Fibrin is eventually produced, resulting in a clot. The coagulation cascade is discussed in greater detail in Chapter 9.

The enzymes involved in coagulation are synthesized in the liver. Some clinicians consider assessments of these enzymes to be the true “liver function tests” because they are products of the liver, rather than merely being markers of liver cell damage. Many coagulation assessments are functions of time and, therefore, are reported in units of seconds.

Prothrombin Time

One of the steps in coagulation involves the creation of thrombin from prothrombin by prothrombin activator, which is a collection of activated substances. The **prothrombin time (PT)** is the rate of conversion of prothrombin to thrombin in a blood sample, and it represents the function of the extrinsic pathway. The normal range is 10 to 13 seconds. The PT may be increased in liver disease or with warfarin therapy, and decreased with low levels of vitamin K, in DIC, and after massive transfusions.

Activated Partial Thromboplastin Time

The **activated partial thromboplastin time (aPTT)** indicates the health of the intrinsic and common pathways of the coagulation system. Genetic diseases, such as hemophilia A, hemophilia B, and von Willebrand disease, may cause elevated aPTT levels. The aPTT is often used to assess for DIC, in which the level is often grossly elevated from the normal range of 25 to 40 seconds. Patients involved in interfacility transport may be receiving heparin therapy or anticoagulant prophylaxis. The aPTT is used to assess for the correct therapeutic effect of heparin.

International Normalized Ratio

Owing to lack of standardization of measuring the PT within different laboratories (especially internationally), a normalizing index has been created by using the international sensitivity index. The normal range for the **international normalized ratio (INR)** is 0.9 to 1.3. Increased ratios are seen in the same diseases in which PT is increased and in persons receiving anticoagulants. The INR is often used to target anticoagulant therapy, with ranges varying by the reason and type of anticoagulant used. The typical anticoagulation target for the INR often ranges from 2.0 to 3.0.

Lactate

Adequate indicators of a patient's tissue and end-organ perfusion and oxygenation are a long-sought "gold standard" for laboratory tests. Growing in popularity as an indicator of a patient's tissue and end-organ perfusion and oxygenation is the venous **lactate** level. Once tissue perfusion and oxygen become inadequate, cells switch to anaerobic metabolism. This mechanism allows the cells to use glucose, but less efficiently than if there was adequate perfusion and oxygen. The result of this inefficient metabolism is production of lactic acid, which is measured as lactate. Perfusion and oxygenation of cells, tissues, and end organs is considered inadequate if the serum lactate level in the blood is more than normal (normal range, 5.0 to 15 mg/dL). Lactate levels are only a global indicator of perfusion and oxygenation and do not indicate which tissues are inadequately perfused or oxygenated. Apart from being nonspecific, the lactate level is slow to respond to adequate resuscitation with fluid and oxygen. Once tissue perfusion and oxygenation return to normal, anaerobic metabolism ceases and lactate is no longer produced. The remaining lactate is converted into nontoxic metabolic by-products in the liver and kidney and, eventually, to carbon dioxide and water.

Osmolality

The serum of the blood contains numerous components that contribute to its tendency to give or extract free water when blood is placed near a semipermeable membrane. As noted in the chemistry review section, this tendency is commonly called osmolarity, although it is measured as osmolality in clinical medicine. Physiologically, the normal range for osmolality is 275 to 295 mOsm/kg. If a patient has a low level of free water in his or her system (commonly referred to as the patient being "dry"), the osmolality will be high because there will be more particulate matter for a given volume of serum. If a patient is "wet," possibly owing to excess fluid retention, the osmolality will be low. This scenario is similar to the interpretation of the serum hematocrit value. The measured osmolality is often compared with the calculated osmolality to help determine the definitive diagnosis; this will be described in more detail

later.

Ethanol

Laboratory analysis may not only have patient care implications, but also legal ramifications. For example, ethanol (also called ethyl alcohol or EtOH) is not a normal physiologic product in the body; therefore, the value if the person has consumed no alcohol is 0 mg/dL (the normal level in medical literature is < 20 mg/dL). The consumption of alcohol, especially in excess, has neurologic implications and also raises serum osmolarity.

Most states define legal intoxication as an ethanol level of more than 80 mg/dL. Owing to the legal implications of ethanol measurements, precise protocols often govern the technique for drawing the blood specimen and its storage and analysis. Before obtaining a sample for measurement of the ethanol level, CCTPs need to refer to their protocols and guidelines.

Calculated Values

Sometimes in clinical medicine, the result of an individual lab test is also used in a formula for further analysis. For example, a particular lab result may be used only in relation to another test as part of a ratio or it may be part of a complex formula used to summarize many findings in one coherent manner. From a mathematical standpoint, it is extremely important to not change the order of the ratio variables. For example, a BUN:Cr ratio is not equal to a Cr:BUN ratio. If the ratios are not calculated correctly, deviations from normal cannot be properly interpreted.

AST:ALT

The **AST:ALT** is an easily calculated index that is useful for determining the cause of liver dysfunction. For example, this ratio may be used to distinguish between acute (viral) and cirrhotic liver disease. Although not 100% accurate, if the AST:ALT ratio is greater than 1 (especially 3:1 or greater), the etiology often involves alcoholic liver disease. If the AST:ALT ratio is less than 1, acute injury—often from a viral infection—is typically to blame.

BUN:Creatinine

An index to determine the cause of increased levels of two metabolites that indicate renal pathology—BUN and Cr—is the **BUN:Cr** ratio. A normal ratio is 13:1 to 19:1. If the ratio is increased, dehydration, GI bleeding, or increased catabolism may be at fault. A decreased ratio may be present in patients with acute tubular necrosis or a low protein intake, or following hemodialysis. However, many variables affect this ratio, and it is not generally useful in the transport environment.

Anion Gap

Disturbances in the overall electrical or acid-base balance of the body result from disease and can result in further deterioration in a patient's condition. The primary pathology is usually related to respiratory or kidney problems. The major contributors to the overall electrical charge are Na^+, Cl^-, and HCO_3^-. Their relationship is called the **anion gap (AG)** and is portrayed in the following equation:

$$\text{Anion gap} = Na^+ - (Cl^- + Hco_3^-)$$

The normal range for the AG is 8 to 16 mEq/L. An increased AG indicates that unmeasured anions (such as in lactic acid) are present.

Blood Gases

A typical **arterial blood gas (ABG)** panel assesses a patient's acid-base status (pH) based on CO_2 tension ($PaCO_2$), bicarbonate level (HCO_3^-), and base excess (BE). Measures of the patient's oxygen status, oxygen tension (PaO_2), and oxygen saturation (SaO_2) are also given as part of the ABG panel. There are many indications for assessing a patient's blood gas parameters, and any patient in the intensive care unit is likely to meet at least one of these criteria. For example, a patient's blood gas levels are typically analyzed when questions arise about the capacity of a patient to exchange oxygen and carbon dioxide, especially if the patient has an underlying cardiopulmonary disease or is in such unstable condition that the clinician needs to quickly assess if the patient is near death.

Acid-Base Status

The acid-base balance reflects how well the body's respiratory and metabolic systems are functioning. If they are working properly together, then there is a balance. When they are not, acidosis or alkalosis can occur.

Hydrogen Ion Concentration

A direct measure of the acid-base status of the patient, hydrogen ion (H^+) concentration, more commonly referred to as pH, quantifies the amount of unbuffered H^+ present. Physiologic pH is related to the amount of CO_2 and the amount of HCO_3^-. An increase in the PCO_2 will result in a smaller fraction component, which translates to a lower pH (acidic). Conversely, an increase in the HCO_3^- will cause a larger fraction and, therefore, will result in a pH increase (alkaline). The inverse of each of these disturbances will result in the respective opposite result on the pH. In other words, decreased PCO_2 will increase pH and decreased HCO_3^- will decrease pH. Based on the other components of the blood gas panel, the pH derangement (acidosis or alkalosis) can be classified as metabolic or respiratory, respectively.

The normal range for the arterial pH is 7.35 to 7.45; on the venous side, the normal range is slightly lower, 7.31 to 7.41. The body can handle minor variations in pH relatively well. By contrast, once deviations in pH become extreme—for example, 7.2 or less or 7.6 or more (arterial)—normal physiologic reactions may be drastically affected. With large derangements in serum pH, many proteins and enzymes cease to carry out their normal functions, and vasopressors are inactivated. For example, in severe acidosis, epinephrine is rendered physiologically inactive.

Partial Pressure of Carbon Dioxide

The natural by-product of cellular respiration, carbon dioxide, is transported back to the lungs by a number of mechanisms. Carbon dioxide exists in the blood as a soluble gas, which is measured as a partial pressure—called partial pressure of carbon dioxide ($PaCO_2$). Because alterations in respiratory function substantially affect the $PaCO_2$, this parameter is considered the "respiratory" component of the blood gas analysis. If the $PaCO_2$ is above or below the normal range of 35 to 45 mm Hg, a respiratory derangement is present. This derangement may be primary (that is, respiratory acidosis or alkalosis) or secondary (that is, compensated metabolic acidosis or alkalosis). The elevation above sea level indirectly affects the $PaCO_2$. At elevations above sea level, increases in respiratory rate as a result of lower partial pressure of oxygen (PO_2) will result in a lower-than-normal $PaCO_2$ in the blood.

Bicarbonate

In the blood gas panel, $PaCO_2$ represents the respiratory component, and **bicarbonate (HCO_3^-)** represents the metabolic component. If there is a metabolic aspect to an acidosis- or alkalosis-related condition, a

corresponding derangement in HCO_3^- will be seen—its level will be decreased in acidosis and increased in alkalosis.

The normal HCO_3^- is 21 to 28 mEq/L. The administration of intravenous bicarbonate to treat metabolic acidosis is controversial and not generally indicated unless the underlying cause of acidosis cannot be corrected or the pH is less than 7.20.

Base Excess

Base excess (BE), is a measurement of metabolic derangement that is included as part of the ABG panel. Base excess is also known as base deficit (BD) because the value can be either a positive number (excess) or a negative value (deficit). Healthy people do not have an appreciable BE. The BE is measured in units of mEq/L and has a normal range of –2 to +3. If there is an excess amount of acid or lack of base, the BE is negative. Conversely, if there is a deficient amount of acid or an excess amount of base, the BE is positive. Positive values beyond the normal range indicate an excess of bicarbonate (base) and negative values less than the normal range indicate a deficit of bicarbonate. The more extreme the metabolic derangement, the larger the BE value.

Some clinicians use base excess to assess for proper fluid resuscitation. If there is inadequate fluid present to support metabolic activities (as in hypovolemic shock), the patient will experience ineffective energy production with a concomitant increase in lactic acid. The BE value in such a case may be mild (–2 to –6), moderate (–6 to –15), or severe (–15 or less). If a patient has normal vital signs with a continued large BE, a source of continued shock should be sought.

Oxygenation Status

Partial Pressure of Oxygen

The **partial pressure of oxygen (Po_2)** measures the amount of oxygen dissolved in the blood. A healthy person has an arterial Po_2 in the range of 80 to 100 mm Hg. Other than in unique cases (such as carbon monoxide poisoning), patients do not benefit from having a Po_2 greater than 100 mm Hg; the most important consideration is simply that the Po_2 remain within the normal range. As the Po_2 falls below 80 mm Hg, the affinity of hemoglobin for oxygen decreases and oxygen is released more readily from the hemoglobin molecule, which results in hypoxia. Once again, the elevation above sea level affects the arterial partial pressure of oxygen, or Pao_2. At elevations above sea level, the environmental Po_2 is lower, resulting in a lower Pao_2 in the blood.

Oxygen Saturation

The Sao_2 measures the percentage of potential oxygen-binding sites on hemoglobin that are occupied by oxygen molecules. The oxygen saturation measured transcutaneously (Spo_2), using a saturation monitor and probe, differs from the oxygen saturation calculated from an arterial or venous blood sample (Sao_2) that is reported on a blood gas report. The Sao_2 is calculated based on other parts of the blood gas panel. The normal value for measured Sao_2 for a healthy person is greater than 93%. The calculated saturation from the blood gas values can give a falsely elevated saturation when abnormal hemoglobin variants, such as carboxyhemoglobin and methemoglobin, are present in the sample. This is particularly important because although the Sao_2 value may appear normal, the patient actually has a poor oxygen content because the abnormal hemoglobin variants cannot carry oxygen.

Obtaining an Arterial Blood Sample

Finally, this section explains how to obtain an arterial blood sample used for the measurements previously discussed.

Skill Drill 8-1 shows the procedure for obtaining an arterial blood sample, discussed below:

1. Appropriately identify the patient. Explain the procedure. Determine the appropriate arm from which to take the specimen **Step 1**.
2. Perform an Allen test. Have the patient raise the hand above the level of the heart. Then have the patient make a fist while you simultaneously compress the radial and ulnar arteries **Step 2**.
3. When the patient's hand turns white or pale, release compression of the ulnar artery and assess circulation. The color should return to normal in approximately 5 to 7 seconds **Step 3**.
4. Repeat while releasing the radial artery. Sluggish return of color may indicate occlusion in one or both of the arteries. Arterial puncture should not be performed on this extremity.
5. Ensure universal precautions.
6. If possible, position the patient's arm with a small towel roll beneath the wrist to slightly extend the wrist (do not overextend) **Step 4**.
7. Prepare the site per protocol.
8. Palpate the radial artery with your index and middle fingers (do not use your thumb) **Step 5**.
9. Locate the radial artery and puncture the skin with the bevel of the needle at a 45° angle. The syringe should rapidly fill with bright red blood **Step 6**. Do not dig or probe with the needle! If you have difficulty, slightly reposition the needle. If you continue to have difficulty, stop and restart the procedure.
10. Once the appropriate amount of blood has filled the syringe, rapidly remove the needle and hold pressure for approximately 2 to 5 minutes **Step 7**.
11. Safely remove the needle and expel any air contained within the syringe **Step 8**.
12. Place a rubber stopper on the syringe.
13. Label the syringe with appropriate patient identification **Step 9**.
14. Immediately perform the test or place the specimen on ice.
15. Reassess neurovascular status **Step 10**.

Blood Group Testing

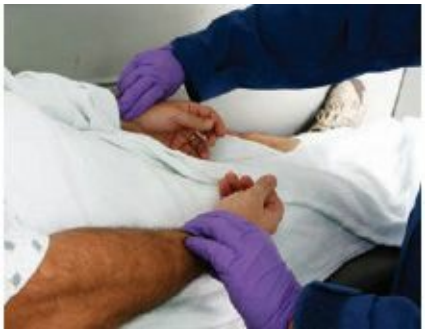
Blood typing is done to determine the blood group according to the ABO system and other classification systems. The ABO system is based on the antigen groups that cause the largest humoral response. There are four that result from two primary antigens, A and B, giving the blood types of A, B, AB, and O. Type A carries the A antigen, type B carries the B antigen, type AB carries both antigens, and type O carries neither antigen. People with type AB are known as universal recipients. They may receive blood of any type. People with type O are universal donors; their blood can be given to people with any type, but they can receive only type O blood. Nevertheless, caution is warranted because people with any blood type can develop an immune response if they receive large amounts of unmatched blood.

It is also important to know about the Rh antigens found in all blood types. The Rh antigen D, which is called the Rh factor, is important in immune responses. Rh positive means the person has the Rh

antigen; people who are Rh negative do not have the antigen. The majority of the population is Rh positive, but caution is needed if a patient is Rh negative. An Rh-negative patient who receives an Rh positive blood transfusion may have an immune response reaction, anaphylaxis, that could develop into anaphylactic shock. For an Rh-negative woman who receives Rh-positive blood, even in the absence of an immediate reaction, she will develop antigens that can cause significant problems with future pregnancies. Most patients needing blood products are given type O–negative blood until typing and crossmatching (which determines the compatibility between donor blood and recipient blood) can be done. Blood groups are discussed in more depth in Chapter 9.

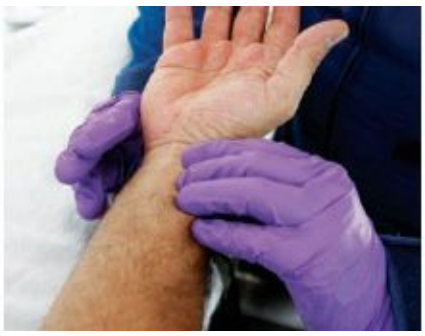
Skill Drill 8-1

Obtaining an Arterial Blood Sample



1 Determine the appropriate arm from which to take the specimen.

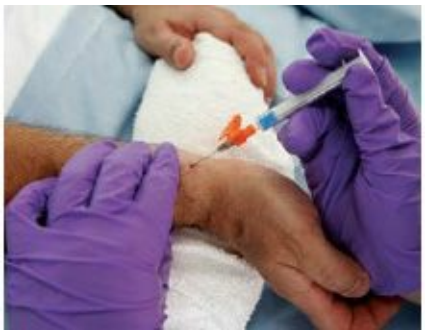
2 Have the patient make a fist. Compress the radial and ulnar arteries.



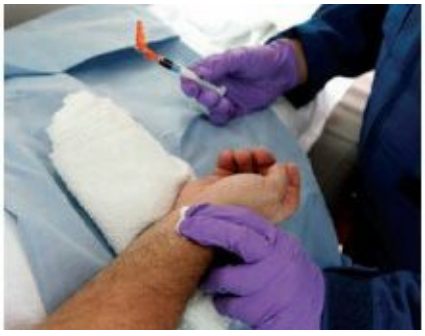
3 When the patient's hand turns white or pale, release compression of the ulnar artery and assess circulation. Repeat for radial artery. Ensure universal precautions.

4 Position the patient's arm with a small towel roll beneath the wrist to slightly extend the wrist.

5 Palpate the radial artery with your index and middle fingers.

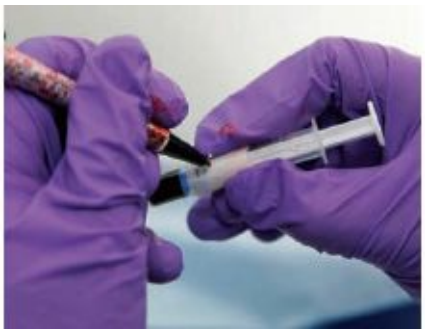


6 Locate the radial artery and puncture the skin with the bevel of the needle at a 45° angle.

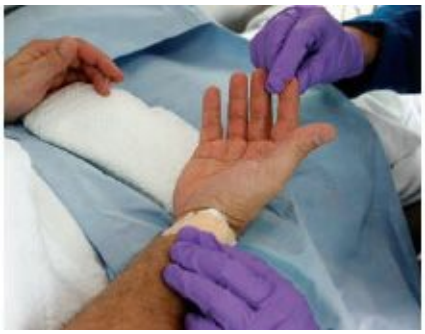


7 Rapidly remove the needle and hold pressure for approximately 2 to 5 minutes.

8 Safely remove the needle and expel any air contained within the syringe.



9 Label the syringe with appropriate patient identification.



10 Reassess neurovascular status.

Obtaining a Venous Blood Sample

Obtaining venous blood samples should initially be completed before administering any IV fluids or medications, such as during the baseline evaluation of the patient. For many medications, ongoing lab results are required to determine the continued course of therapy. Some antibiotics require that a peak and trough be monitored at certain times. Samples for serial levels of CK-MB and troponins and for PT and aPTT testing need to be drawn at specific times. Therefore, blood samples may need to be obtained during transport of the patient.

If the patient has an IV line and is receiving fluids or medications, if at all possible, the other extremity should be used. If the patient has IV lines in both extremities, the one without IV medication or without the medication that is being tested should be used. The flow of fluid or medication needs to be paused or stopped immediately prior to obtaining the blood sample or the sample may be contaminated with infusate.

Blood Tube Uses

Blood tubes are selected primarily for the preservatives or lack of preservatives that they contain. Most laboratories have specific requests regarding tube selection and the number of tubes needed. The desired tube selection is usually communicated by the laboratory **Table 8-2 Figure 8-7**.

TABLE 8-2 Blood Tube Uses

Blood Tube Color	Use
Lavender	Complete blood count
Red	Blood banking
Marbled	Serum chemistry tests
Blue	Coagulation studies
Green	Plasma studies
Yellow	Human leukocyte antigen typing
Navy	Trace metals
Gray	Lactate

Figure 8-7 The colors of blood tube tops relate to their intended use.

Urine Lab Values

Urinalysis (UA) involves performing various laboratory tests (some of the same performed on blood samples) on a patient's urine. Roughly one quarter of the heart's cardiac output enters the kidneys for filtration, and the resulting urine provides a way of investigating the patient's physiologic status. The tests performed on urine may range from simple (such as color) to complex (such as specific gravity). Many drugs can be detected in the urine for hours, days, or weeks after use of the drug. **Table 8-3** lists drugs of abuse that can be tested in urine, including how long the drug can be detected in the urine after the drug's use. **Table 8-4** lists toxic levels of selected substances.

Color

Color may be the most simplistic laboratory analysis performed. This assessment simply notes the urine color, which is often classified as yellow, pale, clear, and so forth. Urine's color is a function of concentration, with more concentrated urine being darker yellow. Various other particulates in the urine can affect the color as well. If the person's glomeruli or renal tubular system is damaged and blood is spilling into the urine, the urine may be reddish. Brown or tea-colored urine often reflects the presence of

large amounts of protein in the urine and is a hallmark sign of rhabdomyolysis.

TABLE 8-3 Drugs of Abuse That Can Be Tested in Urine

Drug	Length of Time Detected in Urine
Alcohol	7–12 h
Amphetamine	48 h
Barbiturate	24 h (short acting); 3 wk (long acting)
Benzodiazepines	3 d
Cocaine	6–8 h (metabolites, 2–4 d)
Heroin	36–72 h
Marijuana	3 d to 4 wk (depending on dose)
Methadone	3 d
Methaqualone	7 d
Morphine	48–72 h
Phencyclidine	8 d
Propoxyphene	6–48 h

TABLE 8-4 Toxic Levels of Selected Substances

Drug Serum Screen	Toxic Blood Level
Acetaminophen	> 250 µg/mL
Alcohol	
Intoxicated	0.1%–0.4%
Stuporous	0.4%–0.5%
Comatose	> 0.5%
Aspirin	> 300 µg/mL
Barbitals	
Sedatives	> 10 µg/mL
Antiepileptics	> 40 µg/mL
Carboxyhemoglobin	> 20%
Phenytoin (Dilantin)	> 20 mg/L
Lead (adult)	> 40 µg/dL
Lithium	> 2.0 mEq/L

Appearance

Similar to color, urine appearance is a rather coarse assessment. Appearance is categorized as clear

(similar to color) or **turbid**. Turbidity can be indicative of a bladder infection and should be examined further.

Specific Gravity

Specific gravity is the chemical property of a fluid that relates its density to the density of water. Distilled water is arbitrarily given a specific gravity of 1, so fluid that is denser than water has a specific gravity greater than 1. The more concentrated a urine sample, the denser the sample and the higher its specific gravity. Conversely, a low specific gravity indicates a more dilute urine sample. At the most simple level, athletes measure specific gravity to assess their hydration status. Higher-than-normal specific gravity would reflect volume depletion in an otherwise healthy athlete.

Numerous factors can affect the kidney's ability to concentrate the urine. For example, if a low level of antidiuretic hormone is secreted from the pituitary gland, diabetes insipidus may result. This metabolic disorder impairs the ability of the kidney to concentrate the urine (that is, the body reabsorbs free water), so the specific gravity of urine will be very low (there would be copious amounts of dilute urine). Other disorders, such as glomerulonephritis and pyelonephritis, may also impair the kidney's ability to concentrate urine. The normal range for urine specific gravity is 1.003 to 1.035; in the setting of impaired ability to concentrate urine, the specific gravity may approach 1.001.

pH

The H^+ concentration in the urine can be a useful marker for metabolic acidosis. Unlike serum pH, urine pH has a relatively wide normal range, 4.5 to 8. Patients with acidic urine often have large amounts of unbuffered acid (such as lactic acid or keto acid). Salt wasting may also occur as the body excretes a positive ion (such as Na^+) to counteract the negatively charged acid salt of lactate or keto acetate.

Certain renal abnormalities may induce the inability to excrete H^+ in the urine, even in the presence of overwhelming metabolic acidosis. This abnormality is called **renal tubule acidosis (RTA)**. Four types of RTA are distinguished, although the specifics of each type are not included here. Nevertheless, CCTPs should be able to identify the presence of RTA, defined as an arterial pH of less than 7.35 and a urine pH of greater than 6. Generally, the biggest concern is that the kidneys are not able to concentrate urine, which will cause wasting of potassium and magnesium.

Glucose

The presence of glucose in the urine (**glycosuria**) is almost always indicative of elevated serum glucose levels. In healthy people, the urine is free of glucose, so the normal value is 0. If glucose is present, its amount is graded on a scale of mild to severe, expressed as + 1, + 2, + 3, or + 4. The presence of glycosuria may not always signal ineffective insulin production or use (as in diabetes mellitus); however, it may result from the administration of a carbohydrate-containing intravenous fluid such as 5% dextrose in water. The context of the patient will dictate when a value outside the normal range is pathologic.

Ketone Bodies

If the body does not completely break down fat to carbon dioxide and water, ketone bodies are produced. Fat catabolism occurs when the need for glucose is not satisfied by the available supply. The term **ketone bodies** describes a number (typically three) of organic products: acetoacidic acid, acetone, and betahydroxybutyric acid. Acetoacidic acid and beta-hydroxybutyric acid are the keto acids that result from poorly controlled diabetes mellitus when inadequate insulin is present and cells are unable to use glucose as a source of energy. Although ketone bodies are not normally present in the urine, the presence

of ketone bodies can help identify inadequately controlled diabetes mellitus. Other causes of **ketonuria** (ketone bodies in the urine) include alcoholic ketoacidosis and starvation ketosis. Grading of the level of ketonuria uses the same system as grading of the amount of glucose in urine—that is, +1, +2, +3, or +4.

Protein

Although the body excretes 40 to 80 mg of protein (one third of which is albumin) in the urine daily, this amount is below the detectable threshold; thus, the normal reference value in a qualitative test for protein would be negative (or not detectable). Urinary protein is one of the most important assessments for kidney diseases. Detectable levels of urinary protein can result from renal causes (such as glomerulonephritis, polycystic kidneys, diabetic nephropathy, toxic nephropathy, nephrosclerosis, and nephritic syndrome) or extrarenal causes (such as preeclampsia, multiple myeloma, urinary tract disease, amyloid disease, systemic lupus erythematosus, congestive heart failure, and constrictive pericarditis).

Blood and Hemoglobin

The presence of blood in the urine is highly suggestive of kidney or urinary tract damage. Recall that blood is not able to cross the glomerular membrane and enter the filtrate. Thus, the presence of hemoglobin in the urine (**hemoglobinuria**) most often indicates traumatic passage of RBCs through the collecting ducts, urinary bladder, or urinary tract. Urinary hemoglobin may also result from filtering of serum hemoglobin during hemolytic transfusion reactions or certain blood disorders such as hemolytic anemia.

The presence of RBCs in the urine (**hematuria**) may indicate the presence of glomerular membrane or urinary tract disease. The cause may be infectious processes (commonly from urinary tract infections), neoplasms, ureterolithiasis, or trauma. Note that RBCs detected in a urinalysis performed soon after placement of an indwelling urinary catheter may merely reflect trauma from insertion of the catheter.

Finally, the presence of hemoglobin in the urine without the presence of RBCs can indicate rhabdomyolysis.

To get a better handle on how the body is eliminating certain electrolytes, a quantitative analysis is performed on the urine. Similar to a basic metabolic panel, this panel analyzes numerous components of the urine. Although their discussion is beyond the scope of this text, urine electrolytes can be useful in clinical management as well.

Cerebrospinal Fluid

Cerebrospinal fluid is obtained for testing by inserting a needle into the lumbar section of the back, between the vertebrae, a procedure that is called a lumbar puncture. Fluid collection and testing are done to identify many conditions, such as increased intracranial pressure, diabetic coma, diabetes, multiple sclerosis, Guillain-Barré syndrome, and meningitis (bacterial or fungal).

Normal results are as follows:

- Pressure: 50 to 180 mm H_2O
- Appearance: clear, colorless
- Total protein: 10 to 45 mg/100 mL
- Glucose: 50 to 80/100 mL
- Cell count: 0 to 5 WBCs, no RBCs
- Chloride: 110 to 125 mEq/L

Common Errors in Specimen Collection

Incorrect patient identification and the mislabeling of samples are the most common specimen collection errors. Two of the most common problems associated with any specimen collection in the field are improper technique or handling and improper labeling. CCTPs generally care for only one patient at a time.

Improper labeling can be resolved by placing the minimal required information on the label for the sample. This required information includes the patient's name and date of birth, date and time the specimen was collected, and the CCTP's name or initials should there be any questions. Ideally, a patient identification number or the last four digits of the patient's Social Security number will be included as well.

Improper technique or handling can be difficult to address, especially in the field setting. CCTPs may encounter extreme temperatures, which can affect optimal processing and results. Trauma to blood specimens may create a hemolyzed sample. Fluids or medication the patient is receiving may interfere with obtaining a "clean" sample. Particular laboratories may require samples to be supplied in specific tubes or containers.

Additional factors that lead to errors in lab results include hemolyzed samples, incorrect collection techniques, and insufficient quantities for accurate testing.

Diagnostic Imaging

Diagnostic imaging is typically performed by a technologist and is often interpreted by a physician on site or possibly sent off site for interpretation. Typically, copies are included with the patient's charts upon transfer.

The CCTP should be familiar with these studies and be able to perform basic interpretation for obvious abnormalities, such as long-bone fractures, masses, and catheter placements. It is important to have a system and use that system every time. Think of it like the steps of a primary exam. This will prevent the CCTP from overlooking findings. Extensive interpretation requires significant training and is beyond the scope of this book.

Standard Radiographs

One of the oldest and still frequently used diagnostic imaging modalities is the standard or plain radiograph. It can be rapidly obtained and used for quick diagnostics in patient care.

A radiograph or roentgenogram is radiation projected through the tissue and onto a photographic plate. Different densities of the body cause gradation changes in black and white onto the film. Dense objects such as bone or blood appear white, whereas less dense objects such as air appear black. Objects with a medium density such as tissues will appear as shades of gray.

Typically obtained in a lateral or AP (anteroposterior) view, a radiograph can provide confirmation of endotracheal tube placement, cervical fractures, chest interpretations, and most major bone fractures.

Computed Tomography

Computed tomography (CT), also called CAT scanning, is an imaging method in which cross-sectional images of the structures in the body plane are reconstructed by a computer program. Focused beams examine an area of interest and are reconstructed by computer to give a high-resolution view. The patient is typically on a table that feeds into the scanner device.

CT scans are commonly used when it is necessary to evaluate different characteristics of tissues, bones, and organs. Unlike conventional x-ray images, CT allows differences to be quantified, which

allows differentiation of tumors from soft tissue, old from new fractures, air from fluid, and blood clots from normal blood. The views or "slices" can pick up abnormalities not seen with traditional plain radiography. Interpreting a CT scan can reveal conditions such as aortic dissection, lung injuries, abdominal organ injuries, fractures, pulmonary emboli, pleural effusions, and acute respiratory distress syndrome.

The CT scan has seen major improvements with multiple slices (64 slices) and higher-speed capabilities. It is now a standard for diagnostics in multiple disciplines. It can be limited by morbidly obese patients who simply do not fit into the scanner or exceed the table operating limits. When transporting a critically ill patient who has had a recent CT scan, the CCTP should attempt to obtain a printed or electronic copy of the scan from the transferring facility to expedite care on arrival at the receiving center.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is primarily a diagnostic imaging technique most commonly used to visualize the internal structure and function of the body. MRI provides a much greater contrast of the different soft tissues of the body than a CT scan does. This makes it especially useful in brain, spinal cord, musculoskeletal, cardiovascular, and oncologic imaging. Another benefit is that the patient is not exposed to radiation.

MRI uses a large circular magnet and radio waves to align the water molecules and generate signals from atoms in the body. An image is obtained from this alignment and the speed at which molecules alter or release. The strength of the magnetic field precludes many types of patient monitoring and requires special ventilators for intubated patients. As such, MRIs are not often used to aid in the diagnosis of critically ill patients. When done, they are usually used to provide exquisitely detailed images of the brain, spine, skeletal system, heart, abdomen, or pelvis.

MRI may be limited by patient size and/or the patient's ability to tolerate an enclosed "tunnel" environment. Any preexisting metal in the patient's body may contraindicate a scan as well. Many advances have been made in the size and "openness" of the MRI, which allows for a more tolerable environment.

It is important to mention safety, because the powerful magnet will cause certain metal objects to become "missiles." Keeping your equipment and any other metals you may be wearing away from the control area is essential.

Ultrasonography

As with MRI, the ultrasound does not expose the patient to any radiation. It also has the advantage of being very portable.

Ultrasound uses sound, which is transmitted through an area of the body; the denser material reflects waves back to a transducer, which produces an image. This image may appear to be somewhat of a blur to the untrained eye, but an experienced technician can identify the area in question.

Ultrasound is typically used for obstetrics and gynecology as well as abdominal diagnostics. It has gained great momentum in blood-flow studies and cardiac diagnostics.

Summary

One of the core goals of CCTPs is to form as complete a clinical picture of the patient as possible. Each patient is unique in terms of his or her hemodynamic and ventilatory status, with various degrees of end-organ dysfunction. Laboratory analysis allows clinicians to assess or confirm the system or organ status.

The key lesson in laboratory interpretation is that no lab result must be analyzed in isolation. Not only are trends important, but also, derangements must be confirmed by other means. If that principle is retained throughout the patient's care, clinicians can be confident that their consideration of lab values is of the highest caliber.

Table 8-5 summarizes normal and abnormal lab values.

TABLE 8-5 Normal and Abnormal Lab Values for Adults*

Name	Abbreviation	Normal	Critically Low	Critically High
Electrolytes and Other Chemicals				
Sodium (mEq/L)	Na^+	136–142	< 125	> 145
Potassium (mEq/L)	K^+	3.5–5.0	< 3.0	> 5.0
Chloride (mEq/L)	Cl^-	96–106	Varies	Varies
Calcium, total (mg/dL)	Ca^{++}	8.2–10.2	< 6.5	> 13.5
Calcium, ionized (mmol/L)	Ca^{++}_i	1.15–1.27	Varies	Varies
Total co_2 (mEq/L)	co_2	22–28	< 15	> 40
Phosphate (mEq/L)	PO_4	2.3–4.7	< 1.2	> 9
Metabolites				
Blood urea nitrogen (mg/dL)	BUN	8–23	< 2	> 80
Creatinine (mg/dL)	Cr	0.6–1.2	< 0.4	> 2.8
Lactate/lactic acid (mmol/L)	N/A	Venous: 0.4–2.0 Arterial: 0.5–1.6	Varies	> 4.99
Proteins				
Protein, total (g/dL)	N/A	6.0–8.0	Varies	Varies
Albumin (g/dL)	N/A	3.5–5.0	Varies	Varies
Myoglobin (µg/L)	N/A	19–92	Varies	Varies
Troponin I (ng/mL)	cTnI	0–0.4 (negative)	Varies	Varies
Enzymes				
Lactate dehydrogenase (U/L)	LDH	100–200	Varies	Varies
Alanine aminotransferase (U/L)	ALT	10–40	N/A	Varies
Aspartate aminotransferase (U/L)	AST	10–30	N/A	Varies
Alkaline phosphatase (U/L)	Alk Phos	30–120	Varies	Varies
Amylase (U/L)	N/A	27–131	Varies	Varies
Lipase (U/L)	N/A	31–186	Varies	Varies
Creatine kinase (U/L)	CK	40–150	Varies	Varies
Coagulation				
Prothrombin time (s)	PT	10–13	N/A	Varies
Activated partial thromboplastin time (s)	aPTT	25–40	N/A	> 60
International normalized ratio	INR	2.0–3.0 (target for therapeutic anticoagulation therapy) Normal range for a patient who is not anticoagulated: 0.8–1.2	Varies	Varies
Serum				
Osmolality (mOsm/kg)	N/A	275–295	< 240	> 320
Blood Count	CBC			
Hematocrit (%)	Hct	41–50	< 20	> 60
Hemoglobin (g/dL)	Hgb	14.0–17.5	< 7	> 20
Erythrocytes ($\times 10^6$/µL)	RBC	3.9–5.5	Varies	Varies
Leukocytes (/µL)	WBC	4,500–11,000	< 2,000	> 30,000
Arterial Blood Gases				
Percentage of hydrogen ions	pH	7.35–7.45	< 7.2	> 7.6
Partial pressure of oxygen (mm Hg)	Pao_2	80–100	< 40	N/A
Partial pressure of carbon dioxide (mm Hg)	$Paco_2$	35–45	< 20	> 77
Bicarbonate (mEq/L)	Hco_3^-	21–28	< 10	> 40
Base excess (mEq/L)	BE	−2 to +3	< −5	> +5

*It is important to know the normal range for the laboratory where the specimen is tested.

Data sources:

Fischbach FT, Dunning MB III, eds. *Manual of Laboratory and Diagnostic Tests*. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.

Iverson C, Christiansen S, Flanagin A, et al. Table 2: Selected laboratory tests, with reference ranges and

conversion factors. *AMA Manual of Style: A Guide for Authors and Editors*. 10th ed. New York, NY: Oxford University Press; 2007. American Medical Association: 798–815.

Case Study

YOU HAVE BEEN DISPATCHED TO TRANSPORT , via helicopter, a 37-year-old man whose pretransport diagnosis is acute renal failure after renal transplantation.

You arrive at a rural hospital at the base of a mountainous region. The physician gives you the following history: The patient and his friend took a day hiking trip into the mountains and got lost. A search-and-rescue team was dispatched after 24 hours. The patient and friend were found 48 hours later. They were found severely dehydrated and confused. They only had "a little" water to sustain themselves. The patient underwent kidney transplantation approximately 1 year ago. Before today, he has not had any difficulties with the transplant. The patient has missed 2 days of his transplant antirejection drugs. The search-and-rescue team had inserted a large-bore IV needle and had provided fluid resuscitation to the patient with 2,000 mL of normal saline before getting him to the local hospital.

The staff gives you the following report: The patient is now alert and oriented with generalized weakness. He has had a total intake of 2,500 mL of normal saline. He has an indwelling urinary catheter in place with 100 mL of urine output since the catheter was placed. His current vital signs are as follows: temperature, 96.0°F (35.6°C); heart rate, 88 beats/min; respirations, 20 breaths/min; and oxygen saturation, 98% with 4 L/min of oxygen per nasal cannula. His blood pressure measures 150/90 mm Hg. He has two large-bore IV needles in place. His current ECG shows a normal sinus rhythm, with a rate of 88 beats/min. He has tall, peaked T waves in the precordial leads V_1 through V_6. His QRS is prolonged, measures 130 milliseconds, and shows an incomplete right bundle block pattern.

Lab analysis reveals the following results: sodium, 135 mEq/L; potassium, 7.3 mEq/L; chloride, 100 mEq/L; and total calcium, 8.8 mg/dL. His BUN is 70 mg/dL; and creatinine, 3.0 mg/dL. The physician says that the CBC and glucose levels were normal. He also says that the CK and myoglobin levels were elevated. He "does not remember the numbers" but will send hard copies along on the transport. The physician tells you he is transporting to tertiary care (45 minutes by air) for emergency dialysis and management of the patient's renal failure.

You give 1 g of calcium gluconate IV. You reassess the ECG and notice that the QRS is narrower at 90 milliseconds (within normal limits) and the T wave is less peaked. You elect to load and go with this patient, realizing that he still needs emergency treatment, but the rest of the medications can be given en route. En route, you administer 50 mL of 50% glucose in water solution ($D_{50}W$) IV followed by 10 U of regular insulin IV; sodium bicarbonate, 50 mEq IV; and 5 mg of albuterol in 3 mL of normal saline via nebulizer.

1. What are your priorities in care before transport?
2. What are your priorities in care during transport?
3. What reassessment parameters are important during transport?

Analysis

In evaluating the situation, you realize that this patient is in acute renal failure with hyperkalemia. You also realize the renal failure could be a result of his severe dehydration, transplant rejection, and/or rhabdomyolysis; however, your transport priority is the hyperkalemia. The markers for emergency treatment of hyperkalemia are profound muscle weakness and/or marked ECG changes. This particular patient has generalized weakness and an intraventricular conduction delay along with peaked T waves,

and he requires immediate treatment. The most important emergency intervention is the administration of calcium, which directly antagonizes the membrane actions of hyperkalemia. Calcium chloride or calcium gluconate may be administered. Calcium chloride contains three times the elemental calcium that calcium gluconate does and should be given via a central line placed at the hospital. You elected to give 1 g of calcium gluconate IV. You reassess the ECG and notice that the QRS is narrower at 90 milliseconds (within normal limits) and the T wave is less peaked. The effects of calcium are usually seen in 3 to 5 minutes and last approximately 30 to 60 minutes.

While en route, you also want to drive the potassium back into the cells by increasing the availability of insulin; therefore, you administer 10 U of regular insulin IV followed by 50 mL of $D_{50}W$ IV (the $D_{50}W$ prevents hypoglycemia). These effects are seen within 15 minutes, usually peak in 60 minutes, and can last for several hours.

Another medication that promotes the shift of potassium into the cells is sodium bicarbonate. Raising the systemic pH will also release hydrogen ions from the cells; potassium is then shifted back into the cell to maintain a neutral state. You gave sodium bicarbonate, 50 mEq IV. These effects are usually seen within 30 minutes and last a few hours.

Last, beta-2 adrenergic agonists (like insulin) drive potassium back into the cell by increasing the Na^+/K^+ ATPase activity needed for the sodium pump. To accomplish this effect, you elected to give 5 mg of albuterol in 3 mL of normal saline via nebulizer. The onset of action is about 30 minutes, and the effects last 2 to 3 hours.

During transport, it is imperative to continuously assess the ABCs. This patient's airway has remained patent, his respiratory rate has been 16 to 20 breaths/min, and oxygen saturation has been 98% with 4 L/min of oxygen. Continuous cardiac monitoring has shown a normal sinus rhythm with a heart rate in the 80s, a normal-appearing T wave, and a normal QRS at 90 milliseconds.

You have point-of-care laboratory testing available during transport. You check his blood glucose level and it is 130 mg/dL. You are also able to test basic electrolytes en route, and the results are as follows: sodium, 140 mEq/L; potassium, 4.4 mEq/L; chloride, 103 mEq/L; and total CO_2, 23 mEq/L. His extracellular potassium level is now within normal limits.

Last, you assess the patient's strength because he was previously weak. He has much stronger upper grip strength, which is equal, and has equal and strong dorsiflexion of his feet. His urine output en route is approximately 200 mL. The patient reports that he generally feels much better.

The patient was admitted directly to the intensive care unit, where you gave a report to the nursing and physician staff. The plan was to restart his antirejection medication and monitor serial lab values to see if the hyperkalemia and kidney function improved. If not, emergency dialysis would be performed.

Prep Kit

Ready for Review

- Laboratory tests—including laboratory examinations of the patient's blood, possibly urine, cerebrospinal fluid, or other body fluid—can be very useful in determining the seriousness of a patient's condition or aiding in anticipation of potential problems en route to the hospital.
- A CCTP must be comfortable with not only the normal ranges for each lab value, but also the test's associated physiologic meaning.
- The value of laboratory analysis is determined by the test's accuracy (a marker of whether the value measured by a test conforms to the true value), precision (a marker of tolerance or variation within

multiple measurements), sensitivity (the ability of a certain test to indicate whether a person does or does not have a certain condition), and specificity (the proportion of people without the target disorder who have a negative test result).

- A normal range for a test indicates the values that 95% of healthy individuals would have for the particular test. It may or may not be reflective of the patients who are being transported by ambulance.
- Although abnormal lab values cannot be ignored, it is essential that they be put into the context of the patient's complex clinical picture.
- Errors in specimen collection, identification, and labeling or errors in laboratory analysis procedures may result in erroneous values being reported.
- Different laboratories have different normal ranges for the same tests.
- A culture and sensitivity (C and S) report can be determined for any type of specimen. The culture results will reveal what is causing an infection; in bacterial infections, the sensitivity results will indicate which antibiotic will most effectively treat the organism causing the infection.
- The ionic components of plasma that are analyzed in the laboratory constitute the electrolytes, which include sodium (Na^+), potassium (K^+), chloride (Cl^-), calcium (Ca^{++}), and magnesium (Mg^{++}).
- Fluid characteristics assessed in the laboratory include osmotic pressure, which arises in a space divided by a semipermeable membrane owing to differences in concentrations of solutes (dissolved substances) found in the solutions on either side of the membrane. Osmotic pressure depends only on the number of particles in the fluid, not on the size of the particles.
- Concentration is the amount of a substance present in a given volume of fluid. In the physiologic system, most concentrations are incredibly small, leading to use of units such as millimoles (mmol), milliequivalents (mEq), and milliosmoles (mOsm) to quantify them.
- Thousands of different kinds of proteins are found in the human body, and specific laboratory tests examine the amount of the various types of protein present in a sample. For example, tests may assess levels of enzymes, which act as catalysts for biochemical reactions.
- In laboratory testing, groups of related tests can be performed as a single unit, called a panel (or profile).
- The basic metabolic panel examines the electrolytes and metabolites in the extracellular fluid and is one of the most basic and fundamental assessments done in the emergency department or intensive care unit.
- Serum sodium is a convenient marker for a patient's fluid status and is one of the key components of the serum osmolality calculation. An abnormal sodium level will not manifest with ECG changes.
- Elevated sodium levels are treated with diuretics and restricted fluid intake; the net effect of this treatment is aimed at lowering intracerebral pressure. Hyponatremia (an abnormally low level of sodium in the blood) is often encountered in patients with congestive heart failure, renal failure, or liver disease and in patients taking diuretics. Hyperkalemia (an abnormally high level of potassium in the blood) can lead to cardiac arrhythmias. Potassium levels of 5.5 to 6.5 mEq/L can result in the classic peaked T waves on an ECG, with flattened P waves occurring when the potassium level exceeds 7.0 mEq/L.
- Older patients (especially patients taking digitalis) are more likely to have arrhythmias and ECG changes with hypokalemia (an abnormally low level of potassium in the blood).
- For any patient with a cardiac history, altered renal function, liver disease, or GI disturbances, or a patient receiving insulin, a recent potassium level should be available for review before transport.

- Patients with hypochloremia (an abnormally low level of chloride in the blood) may have impending renal dysfunction. Care should be used with patients taking diuretics, because they may also have abnormally low chloride levels.
- Venous bicarbonate levels below the normal range could indicate metabolic acidosis or respiratory alkalosis, whereas elevated levels could indicate metabolic alkalosis or respiratory acidosis.
- Urea, which is considered a useful marker for adequate kidney function, is measured via the BUN test. The BUN level tends to increase with age as a consequence of gradually declining renal function.
- Creatinine levels can be used to assess kidney function, although some patients may have an increased creatinine level with adequate renal function. Geriatric patients with increased creatinine levels may have more kidney damage than might be suspected by testing the creatinine level.
- The glucose level is commonly assessed in the field with a point-of-care testing device, and prehospital care providers often measure the blood glucose level of unconscious or semiconscious patients.
- Total calcium is increased in elevated parathyroid hormone states such as hyperparathyroidism and parathyroid-secreting tumors. It is decreased in renal insufficiency, hypomagnesemia, and hyperphosphatemia and in patients who have undergone a massive blood transfusion or who have a decreased parathyroid hormone state.
- Low levels of ionized calcium can cause serious arrhythmias and are especially pronounced in prolonged cardiac arrest. Calcium administration may be warranted in patients with hyperkalemia, hypocalcemia, or toxic levels of calcium-channel blockers.
- A CBC includes measurements of hematocrit (the percentage of cells in a venous blood sample), hemoglobin (the protein responsible for carrying oxygen to the body's cells and, to a lesser extent, carbon dioxide back to the lungs), red blood cells (erythrocytes), white blood cells (leukocytes), and platelets (useful for assessing coagulation status).
- Protein-based tests include those for total protein and albumin. With albumin, abnormally low levels indicate liver disease, whereas abnormally high levels lead to acute respiratory distress syndrome.
- Elevated liver function test results may indicate the presence of liver damage. They measure enzymes that normally appear in liver cells (such as aspartate aminotransferase [AST], alanine aminotransferase [ALT], and bilirubin).
- Tests of pancreatic function include measurements of amylase (an enzyme used by the body to metabolize carbohydrates) and lipase (more specific than amylase for identifying pancreatic disease).
- Cardiovascular lab tests include those for lactate dehydrogenase (useful in diagnosing cellular damage), creatine kinase (elevated levels indicate muscle damage), and troponin (useful in detecting myocardial infarction and predictive of adverse outcomes in patients with severe unstable angina).
- Lab tests assessing the coagulation system focus on the prothrombin time (the time from creation of thrombin from prothrombin by the enzyme prothrombin activator), activated partial thromboplastin time (used to assess for various diseases), and the international normalized ratio.
- Lactate levels are a global indicator of perfusion and oxygenation; they do not indicate which tissues are inadequately perfused or oxygenated.
- Levels of ethanol (ethyl alcohol) can be quite high when patients consume alcohol. Owing to the legal implications of ethanol measurements, precise protocols often govern the technique for obtaining the blood sample and storing and analyzing it.
- The AST:ALT ratio is useful for determining the cause of liver dysfunction—for example,

distinguishing between acute (viral) and cirrhotic liver disease.

- An increased BUN:creatinine ratio may indicate dehydration, GI bleeding, or increased catabolism. A decreased ratio may be present in patients with acute tubular necrosis, in those with a low protein intake, or following hemodialysis.
- An excessively large osmolality gap (the difference between measured osmolality and calculated osmolality) often indicates the presence of a low-molecular-weight toxin, usually ethanol.
- Disturbances in the overall electrical balance of the serum can indicate disease; the anion gap is a measure of this balance.
- Urinalysis involves performing laboratory tests—ranging from the simple (such as color or appearance) to the complex (such as specific gravity)—on a patient's urine. Many drugs of abuse can be detected in the urine for a considerable amount of time after the drug's use.
- Other tests performed on urine include pH, glucose, ketone bodies (presence can help identify inadequately controlled diabetes mellitus), protein (one of the most important assessments for kidney diseases), and blood and hemoglobin (which are not normally found in urine).
- A typical arterial blood gas (ABG) panel assesses a patient's acid-base status and oxygen status.
- The acid-base balance (pH)—which is assessed based on carbon dioxide tension ($PaCO_2$), bicarbonate level (HCO_3^-), and base excess (BE)—indicates how well the body's respiratory and metabolic systems are functioning. Abnormalities result in acidosis or alkalosis.
- Oxygenation status is measured by assessing the partial pressure of oxygen, arterial (PaO_2) and oxygen saturation (SaO_2) levels.
- Blood groups are identified by using the ABO classification system, which is based on the antigen groups that cause the largest humoral response. The two primary antigens, A and B, yield the four blood types: A, B, AB, and O.
- Rh antigens are found in all blood types. The Rh antigen D (Rh factor) is important in immune responses.
- The majority of the population is Rh positive—so CCTPs need to be extremely aware and alert if they have an Rh-negative patient. A patient who is Rh negative and receives an Rh-positive blood transfusion may develop anaphylaxis, which could develop into anaphylactic shock.
- When possible, initial blood samples should be obtained before administering IV fluids or medications.
- If the patient has an IV line and is receiving fluids or medications, the other extremity should be used for taking a blood sample, if possible. If the patient has lines in both extremities, the one without IV medication or without the medication that is being tested should be used.
- Two of the most common problems associated with any specimen collection in the field are improper technique for handling the specimen and improper labeling of the specimen.
- The CCTP should be familiar with diagnostic imaging studies and be able to perform basic interpretation for obvious abnormalities, such as long-bone fractures, masses, and catheter placements.
- Radiographic studies can provide confirmation of endotracheal tube placements, cervical fractures, chest interpretations, and most major bone fractures.
- Computed tomography is an imaging method in which cross-sectional images of a body plane are reconstructed by a computer. A CT scan can pick up abnormalities not seen with traditional plain radiography. The CCTP should obtain a copy of the scan from the transferring facility to expedite care

on arrival at the receiving center.

- Magnetic resonance imaging provides a much greater contrast of the different soft tissues of the body than a CT scan does, which makes it especially useful in brain, spinal cord, musculoskeletal, cardiovascular, and oncologic imaging.
- Ultrasonography does not expose the patient to any radiation and has the advantage of being a very portable imaging technology.

Vital Vocabulary

accuracy A measure of the likelihood that an average of a set of test values will be the same as individual values in the set.

activated partial thromboplastin time (aPTT) A value that represents the intrinsic coagulation pathway's clotting ability; also known as partial thromboplastin time (PTT).

alanine aminotransferase (ALT) An intracellular enzyme that is found in large amounts in the liver and in the kidney, skeletal muscle, and heart; formerly known as SGPT.

albumin The most common protein in the body. It acts as a transport protein, is a free radical scavenger, and serves as the main source of protein-generated oncotic pressure.

alkaline phosphatase An enzyme that is essential for proper digestion and absorption through the mucous membrane in the gastrointestinal tract; clinically useful for testing liver function and for diagnosing a common bile duct obstruction.

amylase A key enzyme used by the body to metabolize carbohydrates; it is produced primarily by the salivary glands and the pancreas.

anion A negatively charged ion.

anion gap (AG) A summary of the relationship among the three major contributors to the overall electrical charge (Na^+, Cl^-, and HCO_3^-); abnormal AG values may signal disturbances in the overall electrical and acid-base balance of the serum and presence of disease.

arterial blood gas (ABG) Analysis of the following characteristics of blood: pH, partial pressure of carbon dioxide (in arterial blood), partial pressure of oxygen (in arterial blood), concentration of bicarbonate ion, base excess (indicating whether the patient is acidotic or alkalotic), and oxygen saturation of the hemoglobin molecule.

aspartate aminotransferase (AST) An intracellular enzyme that is found in large amounts in the liver and in skeletal muscles, the brain, red blood cells, and the heart; formerly known as SGOT.

AST:ALT A calculated index that is useful for determining the cause of liver dysfunction.

base excess (BE) A measure of metabolic derangement that is part of the arterial blood gas panel; also known as base deficit (BD) as the value can be either positive (excess) or negative (deficit).

bicarbonate (HCO_3^-) An ion (HCO_3^-) that is present in the blood; measurement represents the metabolic component of the arterial blood gas panel.

blood urea nitrogen (BUN) A test used to measure urea, which is a biomarker for adequate kidney function.

B-type natriuretic peptide (BNP) A polypeptide whose value that is indicative of abnormal ventricular function and congestive heart failure.

BUN:Cr A calculated index that is used to determine the cause of increased levels of blood urea nitrogen and creatinine.

carboxyhemoglobin (COHb) A measure of the amount of hemoglobin–carbon monoxide complexes in the blood.

cation A positively charged ion.

computed tomography (CT) An imaging study in which focused beams examine an area that is then reconstructed by computer to give a high-resolution view; multiple "slices" or views are created; also called CAT scanning.

concentration The amount of a substance present in a given volume of fluid.

C-reactive protein (CRP) An acute phase protein synthesized in the liver; an indicator of inflammation.

creatine A major storehouse of intramuscular high-energy phosphate.

creatine kinase (CK) An enzyme that cleaves the high-energy phosphate from creatine in muscle tissues and transfers it to adenosine diphosphate to yield adenosine triphosphate; measurement is used in the assessment for a myocardial infarction.

creatinine (Cr) A breakdown product of creatine that results from muscle metabolism.

D-dimer test A test of hypercoagulability that detects a fragment from the fibrinolysis process; the test can be used to help diagnose and monitor diseases and conditions related to inappropriate clotting; for example, it can be used to test for deep venous thrombosis.

direct bilirubin Conjugated bilirubin; the result of bilirubin's conjugation in the liver, which is ultimately excreted in the bile.

enzymes Proteins that act as catalysts for biochemical reactions within the body.

glycosuria The presence of glucose in the urine.

hematocrit The percentage of formed elements (that is, cells) in a venous blood sample.

hematuria The presence of red blood cells in the urine.

hemoconcentration Decreased fluid in the blood, which means that concentrations of other blood components increase.

hemoglobin (Hgb) The protein responsible for carrying oxygen to the body's cells and, to a lesser extent, carbon dioxide back to the lungs.

hemoglobinuria The presence of hemoglobin in the urine.

hemolysis Destruction of red blood cells, sometimes following massive blood transfusions or blood transfusion reactions.

hyperchloremia An abnormally high level of chloride in the blood.

hyperkalemia An abnormally high level of potassium in the blood.

hypoalbuminemia An abnormally low level of albumin in the blood.

hypochloremia An abnormally low level of chloride in the blood.

hypokalemia An abnormally low level of potassium in the blood.

hyponatremia An abnormally low level of sodium in the blood.

indirect bilirubin A by-product of the metabolism of red blood cells that is unconjugated and, therefore, not water soluble.

international normalized ratio (INR) A comparative rating of a patient's prothrombin time to help standardize the prothrombin time when planning treatment.

ionic bond A type of chemical bond formed between oppositely charged ions.

ionized calcium Calcium that is not bound or chelated (also called free calcium); its value is useful in assessing for renal failure, nephrotic syndrome, acid-base derangements, and decreases or elevations in chelating compounds.

ketone bodies Organic products of fat catabolism—specifically, acetoacidic acid, acetone, and beta-hydroxybutyric acid.

ketonuria The presence of ketone bodies in the urine.

lactate The form of lactic acid that is physiologically present in the body.

lactate dehydrogenase (LDH) An enzyme that catalyzes the metabolism of pyruvate (the end product of glycolysis) to lactate in the absence of a functioning citric acid cycle.

leukocytosis An abnormally high number of white blood cells.

lipase A pancreatic hormone that metabolizes lipids.

liver function test (LFT) A test for liver damage that measures enzymes that normally appear in liver cells but may spill out into the vasculature with parenchymal damage.

magnetic resonance imaging (MRI) A diagnostic imaging technique used to visualize the internal structure and function of the body, which works by using a powerful magnet to align water molecules present in body compartments; an image is obtained from this alignment and the speed at which molecules alter or release.

myoglobin An oxygen-carrying heme protein present in high concentrations in the cytoplasm of cardiac and skeletal muscles.

normal range A range of values encompassing the results that 95% of healthy people would have for the particular test.

osmolality The amount of dissolved substance in 1 kg of water.

osmolarity The amount of dissolved substance in 1 L of water.

osmosis The diffusion of water across membranes.

osmotic pressure The pressure created in a space divided by a semipermeable membrane owing to differences in concentrations of solutes found in the solutions on either side of the membrane.

panel Groups of related tests that are performed as a single unit; also called profile.

partial pressure of oxygen (Po_2) A measurement of the amount of oxygen dissolved in the blood.

platelet (Plt) count A measurement of the number of platelets in the blood, which is useful for assessing a patient's coagulation status.

precision A measure of how a value is likely to be the same every time a test is performed.

prothrombin time (PT) A value that represents the extrinsic coagulation pathway's clotting ability by taking into account various clotting factors, fibrinogen, the prothrombin ratio, and the international normalized ratio.

red blood cell (RBC) count A measure of the total number of erythrocytes in the blood.

renal tubule acidosis (RTA) An inability to excrete H^+ in the urine, even in the presence of

overwhelming metabolic acidosis.

sensitivity The ability of a certain test to indicate whether a person has a certain disease.

specific gravity The chemical property of a fluid that relates its density to the density of water.

specificity The ability of a certain test to indicate whether a person does not have a certain disease.

thrombocytopenia An abnormally low level of platelets.

total protein The total quantity of protein in a blood sample.

troponin A key protein involved in muscle contraction that is present in the serum only after cellular necrosis releases the cellular contents of cardiac muscle (such as after myocardial infarction).

turbid Cloudy or opaque.

ultrasound An imaging technique that uses sound, which is transmitted through an area of the body, then denser material reflects waves back to a transducer that produces an image; typically used for obstetrics, gynecology, and abdominal diagnostics.

urinalysis (UA) Laboratory tests performed on a patient's urine.

white blood cell (WBC) count A measure of the total number of leukocytes in the blood.

Bibliography

Abelow B. *Understanding Acid-Base*. Baltimore, MD: Williams and Wilkins; 1998.

Allon M, Shanklin N. Effect of albuterol treatment on subsequent dialytic potassium removal. *Am J Kidney Dis*. 1995; 26:607–613.

Clausen T, Everts ME. Regulation of the Na,K-pump in skeletal muscle. *Kidney Int*. 1989;35:1–13.

Czarnecki M. *Critical Care Field Guide*. Sudbury, MA: Jones and Bartlett Publishers; 2001.

Evans KJ, Greenberg A. Hyperkalemia: a review. *J Intensive Care Med*. 2005;20:272–290.

Fischbach FT, Dunning MB III, eds. *Manual of Laboratory and Diagnostic Tests*, 8th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2009.

Garrett R, Grisham C. *Biochemistry*, 2nd ed. Orlando, FL: Harcourt Brace College Publishers; 1999.

Garth D. Hyperkalemia. Available at: http://emedicine.medscape.com/article/766479-print. Published February 20, 2007. Accessed March 1, 2008.

Halperin M, Goldstein M. *Fluid, Electrolyte, and Acid-Base Physiology*, 3rd ed. Philadelphia, PA: WB Saunders Co; 1999.

Iverson C, Christiansen S, Flanagin A, et al. Table 2: selected laboratory tests, with reference ranges and conversion factors. In: *AMA Manual of Style: A Guide for Authors and Editors*, 10th ed. New York, NY: Oxford University Press; 2007. 798–815.

Marieb E. *Human Anatomy and Physiology*. 3rd ed. Redwood City, CA: Benjamin/Cummings Publishing Co; 1995.

Markovchick V, Pons P. *Emergency Medicine Secrets*, 2nd ed. Philadelphia, PA: Hanley & Belfus; 1993.

Masterson W, Hurley C. *Chemistry: Principles and Reactions*, 2nd ed. Orlando, FL: Harcourt Brace College Publishers; 1993.

Parsons P, Wiener-Kronish J. *Critical Care Secrets*, 2nd ed. Philadelphia, PA: Hanley and Belfus; 1998.

Program for Appropriate Technology in Health (PATH). *RBP-EIA: Collecting, Processing, and Handling Venous, Capillary, and Blood Spot Samples*. Available at: http://www.path.org/files/TS_rbp-eia_blood_collct.pdf. Published June 2005. Accessed April 20, 2009.

Skobe C. The basics of specimen collection and handling of urine testing. *LabNotes*. 2004;14:1. Available at: http://www.bd.com/vacutainer/labnotes/Volume14number2. Accessed April 20, 2009.

Smith B. Preanalytical errors in the emergency department. *LabNotes*. 2007;17:1. Available at: http://www.bd.com/vacutainer/labnotes/volume17number1. Accessed April 20, 2009.

Tintinalli J, Ruiz E, Krome R. *Emergency Medicine: A Comprehensive Study Guide*, 4th ed. New York, NY: McGraw-Hill; 1996.

Treseler KM. *Clinical Laboratory and Diagnostic Tests: Significance and Nursing Implications*, 3rd ed. Englewood Cliffs, NJ: Prentice-Hall; 1995.

Worthley L. *Handbook of Emergency Laboratory Tests*. New York, NY: Churchill Livingstone; 1996.

Shock, Sepsis, and Multiple Organ Dysfunction Syndrome

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Objectives

1. Discuss oxygen transport and utilization of oxygen by the cell (p 265–267).
2. Discuss cellular respiration (p 264–265).
3. Discuss the pathophysiology of shock and its stages: initial, compensatory, progressive, and refractory (p 268–271).
4. Describe the clinical manifestations associated with the various shock states (p 268–271).
5. Discuss the classification of shock: hypovolemic, cardiogenic, distributive (neurogenic), anaphylactic, and septic (p 272–280).
6. Identify from a patient presentation what type and stage of shock the patient is experiencing (p 268–271).
7. Define the following terms: infection, sepsis, sepsis syndrome, systemic inflammatory response syndrome (SIRS), severe sepsis, septic shock, and multiple organ dysfunction syndrome (MODS) (p 271, 272, 280).
8. Describe the etiology and pathophysiology of shock, sepsis, SIRS, and MODS (p 280–282).
9. Describe the history and incidence of sepsis relative to mortality (p 280).
10. Describe the risk factors considered important in the development of sepsis (p 281).
11. Describe the new theory in the development of SIRS, including the four response stages (p 280).
12. Explain the pathophysiology of SIRS (p 282).
13. Describe the mediators involved in the development of sepsis (p 280).
14. Recognize the individual organ manifestations related to the development of SIRS (p 280).
15. Identify the signs and symptoms that describe the patient in shock, sepsis, SIRS, or MODS (p 273–276, 279, 281, 286).
16. Discuss lab values of the septic or MODS patient (p 281).
17. Discuss assessment and management of the shock/MODS patient (p 284–289).
18. List the parameters necessary to monitor during the transport of a critical patient experiencing shock, sepsis, SIRS, or MODS (p 274, 275, 278, 279, 286).
19. Select from a list those interventions necessary during transport of the critically ill patient experiencing shock, sepsis, SIRS, or MODS (286–294).
20. Discuss pharmacologic agents used in the treatment of the shock/MODS patient (p 288).
21. Discuss blood administration, including the ABO blood system, various blood products, and the

procedure for administering blood (p 289–294).

Introduction

Shock, sepsis, and multiorgan dysfunction syndrome (MODS) are complex disorders that affect the body in multifaceted dimensions. As unique as each of these are, they share a common denominator—a breakdown in oxygen supply and demand. When oxygen delivery to the cells is compromised, cells are damaged. A cascade of unfortunate events begins with localized tissue damage and may eventually progress to organ failure and death. Before jumping into the pathophysiology of shock, sepsis, and MODS, it is imperative to grasp the basic physiology of how the cell and the microcirculation function. After all, the cell and the microcirculation are the first to experience the insult of inadequate oxygen supply.

Cellular Respiration

All cells require a continuous supply of oxygen, glucose, and other nutrients for normal metabolic function and homeostasis. However, there is another essential piece: energy. Energy is required to maintain cellular metabolic processes, much like electricity is needed to power manufacturing plants. It is the responsibility of the mitochondria to take the nutrients supplied and manufacture them into energy in the form of adenosine triphosphate (ATP), the primary energy-carrying molecule in the body.

ATP is manufactured through a complex process known as cellular respiration. Essentially, cellular respiration involves three parts: glycolysis **Figure 9-1**, the citric acid cycle **Figure 9-2**, and the electron transport chain, otherwise referred to as oxidative phosphorylation. These three components work together in an aerobic environment to produce large amounts of ATP.

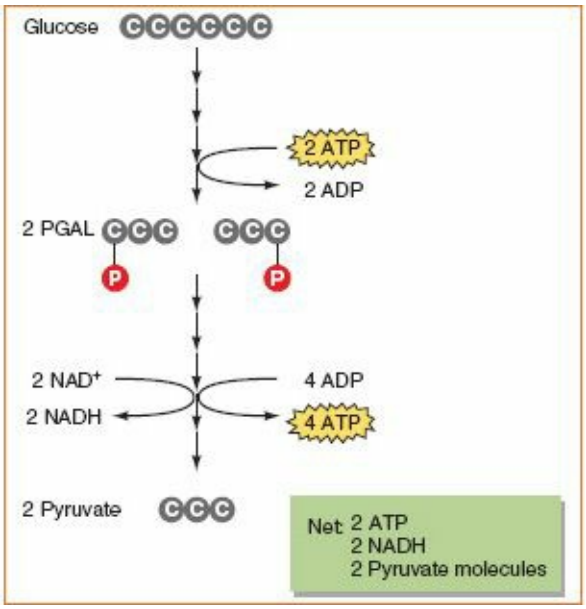

Figure 9-1 During glycolysis, glucose is broken down into two molecules of glyceraldehyde-3-phosphate (PGAL), which are converted to pyruvate. The cell nets two ATP and two NADH (reduced form of nicotinamide adenine dinucleotide) molecules.

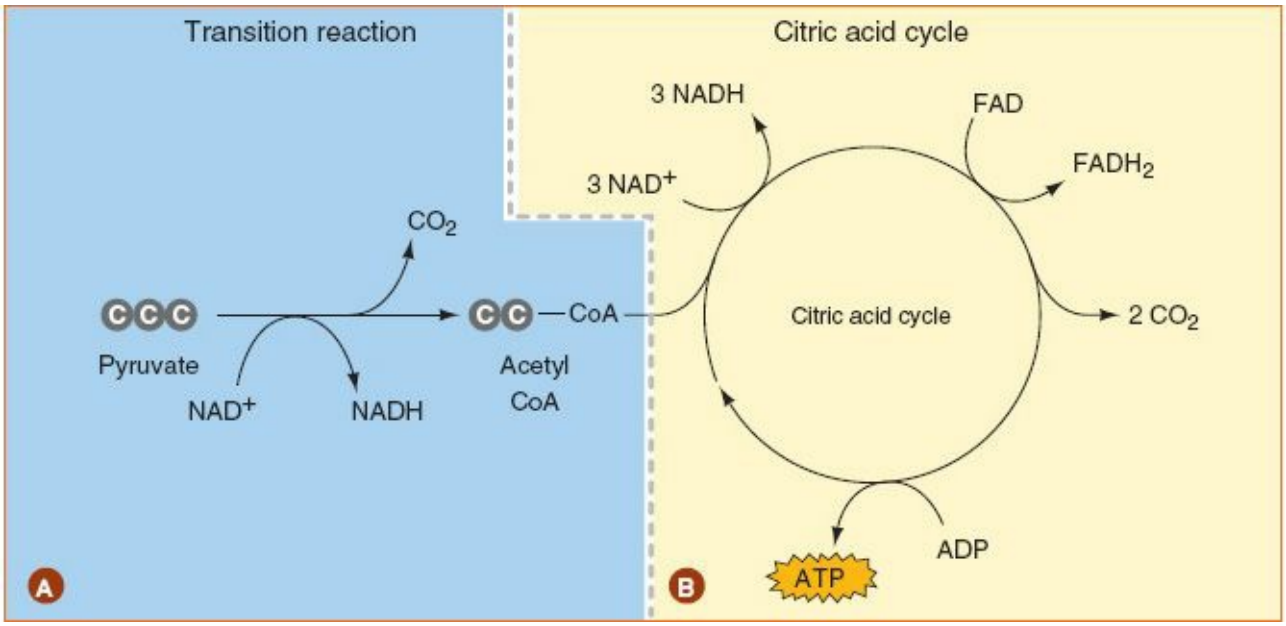

Figure 9-2 The citric acid cycle. **A.** Coenzyme A is created. **B.** In the mitochondrion, the citric acid cycle liberates two carbon dioxide molecules and produces one ATP molecule per pyruvate molecule. Its main products, however, are NADH (reduced form of nicotinamide adenine dinucleotide) and $FADH_2$ (a reduced form of flavine adenine dinucleotide), bearing high-energy electrons that are transferred to the electron transport system.

The citric acid cycle and the electron transport chain can only work in the presence of oxygen to break down glucose. However, glycolysis, the breakdown of glucose for energy, can occur in an aerobic or anaerobic environment. When there is not enough oxygen for aerobic metabolism of glucose, glycolysis must stand alone to produce ATP. This solo attempt to support cellular respiration causes a reduction in ATP and an increased production of lactic acid. As a result, cells are damaged and tissue function is impaired, leading to a cascade of injurious effects.

In the ATP molecule, the critical component for energy production is triphosphate. These phosphorus groups are connected to each other by oxygen molecules. Under normal conditions, the oxygen molecules have a negative charge and, thus, repel each other. It is this property that produces the potential energy of ATP. Removing just one of the phosphate groups results in conversion to adenosine diphosphate (ADP) and a release of energy. This conversion from ATP to ADP is crucial for supplying the energy required for biologic functions.

In **aerobic metabolism**, the ultimate acceptor of electrons is oxygen. The mitochondria utilize glucose, amino acids, and fatty acids combined with oxygen and ADP to produce ATP, carbon dioxide, water, and heat. The energy is stored as ATP, and the resultant carbon dioxide is eliminated by way of the respiratory tract.

In **anaerobic metabolism**, an alternative pathway converts glucose to pyruvic acid with the simultaneous production of ATP; however, significantly less ATP results from this process. This process also results in the production of lactate, which is released into the extracellular fluid and decreases the pH of body fluids. This process is less efficient than aerobic metabolism.

The Microcirculation and the Cell

Understanding the imbalance between oxygen supply and demand is critical to understanding the pathophysiology of shock, sepsis, and MODS. To a large extent, the pathophysiology can be compared with mechanisms of injury in trauma. When EMTs and paramedics respond to a vehicular trauma scene,

injuries can be identified and correlated to the impact the patient endured. This is all part of a good scene size up. This same principle is carried over to understanding the cell and the **microcirculation**—the nexus between the arterioles and venules consisting of the capillaries that course between cells of the various organs. The size of the conduits is large enough to allow passage of a single red blood cell. In cases of shock, sepsis, and MODS, the crash site is hidden deep within the body, at the cellular level.

Oxygen Transport and Utilization

For cells to function properly, oxygen is required. Oxygen is a key nutrient in maintaining homeostatic balance within the body. Put another way, homeostasis can only be maintained when the supply of oxygen is sufficient to meet the cellular demands of the body. Several requirements are necessary to achieve this balance. First, each of the body's systems must be intact and functioning normally. Next, the respiratory system must allow oxygen and carbon dioxide exchange across the alveolar-capillary membrane.

Once this transfer occurs, arterial blood transports oxygen to the tissues. Oxygen is dissolved in plasma (3%) or bound to hemoglobin (97%). Since the majority of oxygen is bound to hemoglobin, it is important to have an ample supply of hemoglobin. Simple formulas exist to determine the oxygen-carrying capacity of hemoglobin. As a standard, 1 g of hemoglobin carries approximately 1.39 mL of oxygen. (Note: Some texts say that 1 g of hemoglobin carries only 1.34 to 1.36 mL of oxygen, based on the theory that a certain percentage will be bound to methemoglobin and not hemoglobin.) Oxygen-carrying capacity is determined by using the following formula:

$$\text{Hemoglobin (Hgb)} \times 1.39 = \text{Oxygen-Carrying Capacity}$$

If the hemoglobin level is low, blood should be administered to optimize oxygen-carrying capacity. It may also be necessary to determine the total amount of oxygen in the arterial blood. The **arterial oxygen content (CaO_2)** formula is used to calculate this value. The normal arterial oxygen content is 17 to 20 mL/100 mL. The formula for determining arterial oxygen content is as follows:

$$Cao_2 = (Hgb \times 1.39 \times Sao_2) + (0.0031 \times Pao_2)$$

Use of the arterial oxygen content formula helps verify the importance of hemoglobin, oxygen saturation (SaO_2), and partial pressure of oxygen, arterial (PaO_2). However, hemoglobin and oxygen saturation have a greater impact on arterial oxygen content than does PaO_2. For this reason, some clinicians use an abbreviated formula for measuring arterial oxygen content:

$$Cao_2 = Hgb \times 1.39 \times Sao_2$$

Table 9-1 explains the components of the arterial oxygen content formula.

The cardiovascular system has the responsibility of delivering oxygen-rich blood to the tissues. There are various factors that can affect transport, including blood volume, viscosity, and arterial elasticity. However, the primary determinants of transport are blood pressure and cardiac output. Systolic blood pressure depends primarily on **cardiac output (CO)**, the force and volume of blood ejected from the ventricles during systole. Cardiac output is dependent on heart rate and the components of stroke volume—preload, afterload, and contractility. Diastolic blood pressure depends on peripheral resistance, which is determined by arteriolar vasoconstriction. Blood pressure is necessary in calculating the **mean arterial pressure (MAP)**. The MAP can provide information on how well organs are being perfused. Overall, an adequate cardiac output and a stable blood pressure are essential for proper perfusion and general performance of the body. The amount of oxygen delivered to the tissues each minute is reflected in

oxygen delivery (Do_2). Oxygen delivery is a calculated formula that combines arterial oxygen content with cardiac output. **Table 9-2** discusses the components of the formula shown here (CO represents cardiac output, and CaO_2 represents arterial oxygen content):

TABLE 9-1 Components of the Arterial Oxygen Content Formula		
Arterial Oxygen Content Formula: $Cao_2 = (Hgb \times 1.39 \times Sao_2) + (0.0031 \times Pao_2)$		
Hgb	97% of oxygen bound with Hgb	Hgb × 1.39 = Oxygen-Carrying Capacity
1.34 to 1.39 (varies in literature)	Each gram of Hgb capable of carrying 1.39 mL of oxygen	
Sao_2	The amount of oxygen carried on Hgb	Oxygen-Carrying Capacity × Sao_2
0.0031	0.0031 mL O_2/100 mL plasma/mm Hg Also known as the solubility coefficient for oxygen	0.0031 × Pao_2 = Amount of Oxygen Dissolved in Plasma
Pao_2	Partial pressure of oxygen dissolved in plasma (normal is 0.8-1.0 mm Hg)	

TABLE 9-2 Oxygen Transport and Utilization: Formula Description		
Step	**Formula Component**	**Normal Values**
Arterial oxygen content (Cao_2):		
1. Transfer of oxygen across the alveolar-capillary membrane 2. Adequate Hgb to carry the oxygen	$(Hgb \times 1.39 \times Sao_2) + (0.0031 \times Pao_2)$	17-20 mL/100 mL arterial blood
Oxygen delivery (Do_2):		
3. Adequate cardiac output to deliver the oxygen-rich blood to the tissues	$CO \times Cao_2$	640-1,400 mL/min
Oxygen consumption ($\dot{V}o_2$):		
4. Appropriate release of oxygen from Hgb	$CO \times Hgb \times 13.9 \times (Sao_2 - Svo_2)$	180-280 mL/min
5. Adequate utilization of oxygen by cells	Oxygen extraction ratio (ERO_2) $\frac{\dot{V}o_2}{Do_2}$	25%

$$Do_2 = CO \times Cao_2$$

If the arterial oxygen content is normal and oxygen delivery is down, measures are taken to enhance CO. These measures include fluid challenges and administration of an inotropic agent. Once all measures for CO enhancement have been attempted, pressor support is used.

When the oxygen-bound hemoglobin reaches the capillaries, oxygen is released from the hemoglobin to the cells in exchange for cellular waste. The amount of oxygen used by the cells and tissues is reflected in **oxygen consumption ($\dot{V}O_2$)**. The formula for $\dot{V}O_2$ is as follows:

$$\dot{V}O_2 = CO \times Hgb \times 13.9 \times (Sao_2 - Svo_2)$$

This formula is unique because it subtracts the percentage of oxygen saturation in the venous bed ($S_{\dot{V}}O_2$) from the oxygen saturation in the arterial bed (SaO_2). Mixed venous oxygen saturation (SvO_2) is measured much like an arterial blood gas (ABG), except that in measuring the venous saturation, blood is drawn from the distal port of the pulmonary artery (PA) catheter. This value reflects how well the body's tissues are able to extract and utilize the oxygen attached to hemoglobin. A sample from the distal port of the PA catheter reflects only a global perspective of how well the tissues are utilizing oxygen. The problem with this test is that different tissues extract more or less oxygen than others. For example, pulmonary and cardiac tissues extract approximately 40% of the oxygen from hemoglobin. As a result, the SvO_2 of venous blood drawn directly from the cardiovascular or pulmonary circulation will normally be about 60%. In contrast, the skin typically only extracts about 5% of the oxygen from hemoglobin. Measurement of the SvO_2 by using the tip of the PA catheter provides an average of the oxygen that was extracted from hemoglobin throughout the body. The normal SvO_2 level measured in the pulmonary artery is approximately 60% to 80%. When this value is abnormal, there is no way to specify which tissue bed or organ is responsible for the dysfunction. There are some specialty catheters that allow direct SvO_2 measurements.

Under certain circumstances, oxygen will more readily be extracted from hemoglobin or bound to hemoglobin, sometimes referred to as hemoglobin's affinity for oxygen. This affinity for oxygen is dependent on the PaO_2, the pH of the blood, the level of carbon dioxide in the blood, the ambient temperature, and the effects of BPG (2,3-biphosphoglycerate—a chemical that binds to deoxygenated hemoglobin, which helps red blood cells release oxygen). The effects of PaO_2 on the oxyhemoglobin saturation of red blood cells are reflected in the oxyhemoglobin dissociation curve (shown in Chapter 6). As the PaO_2 decreases, the amount of oxygen bound to hemoglobin in the arterial blood decreases, and less oxygen will be available for utilization by the tissues. A rightward shift of the dissociation curve causes decreased affinity for oxygen by hemoglobin. Conditions that cause a rightward shift are increased carbon dioxide level, BPG level, and temperature and a decreased pH. A leftward shift of the dissociation curve causes an increased affinity for oxygen by hemoglobin. In other words, oxygen is more readily bound to hemoglobin, and the body can maintain oxygen saturation with a lower PaO_2. However, release of the bound oxygen to tissues becomes more difficult. Conditions that cause a leftward shift are the opposite of those that cause a rightward shift. They are decreased carbon dioxide level, BPG level, and temperature and increased pH.

Finally, it is the responsibility of the cells to utilize the oxygen. The $\dot{V}O_2$ is dependent on the DO_2. The relationship between these two components is reflected in the **oxygen extraction ratio** (ERO_2), which is as follows:

$$ERO_2 = \frac{\dot{V}O_2}{DO_2}$$

Under normal conditions, only 25% of the oxygen delivered to the tissues is actually extracted. Therefore, the normal SvO_2 when measured in the pulmonary artery is approximately 75%. This allows the body a buffer zone for periods of low oxygen delivery when the cells must extract more oxygen. For

example, if the DO_2 drops to 600 mL/min, for $\dot{V}O_2$ to remain within normal limits (180 to 280 mL/min), the ERO_2 will need to increase 33%. If the $\dot{V}O_2$ drops, cells are forced to work under anaerobic conditions. Mortality rates increase as $\dot{V}O_2$ is compromised. Therefore, treatment is geared toward maximizing $\dot{V}O_2$, which is done partially by maintaining adequate oxygen supply, providing hemoglobin through administration of blood products, and optimizing cardiac output. If there is a breakdown in one or more of these components, homeostasis is lost.

Figure 9-3 summarizes the cycle of oxygen transport and utilization.

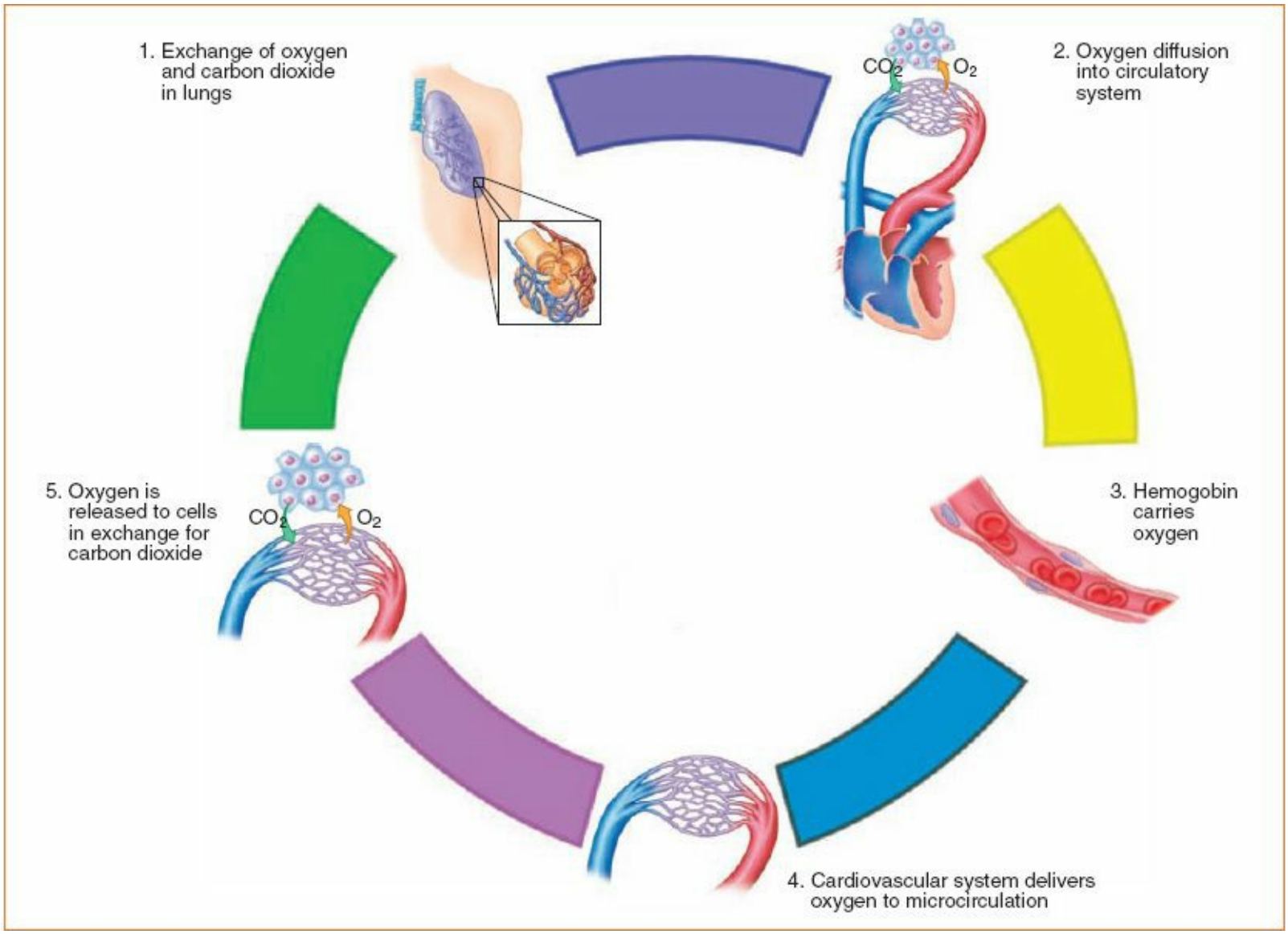

Figure 9-3 Oxygen transport and utilization.

Shock

Shock is a whole-body response to an inadequate supply of oxygen within cells, tissues, and organs from one or multiple causes. The beginning stages of shock occur at the cellular level. When oxygen is unable to be supplied to the microcirculation, cells are forced to function in an anaerobic environment, leading to widespread cellular hypoxia. Shock is a progressive condition that is commonly classified in four stages: initial, also referred to as early shock; compensatory; decompensatory or progressive shock; and, finally, irreversible or refractory shock.

Initial Stage

As a compensatory mechanism to maintain blood flow to the heart, brain, and adrenal glands, the microcirculatory blood flow to other tissue beds is severely restricted. The initial stage of shock begins as blood flow into the microcirculatory beds decreases to a point that oxygen delivery to the cells falls below levels required to maintain normal aerobic cellular function. Initially, the tissues respond to the restricted oxygen delivery by consuming as much available oxygen as possible, thereby increasing oxygen consumption. Eventually, oxygen oversupply is used up and cells are unable to extract enough oxygen. The mitochondria cannot function aerobically to produce ATP, and hypoxia develops. This anaerobic environment leads to increased lactate and carbon dioxide levels. As lactic acid levels rise, ATP production is severely reduced and cells are unable to maintain homeostasis, and, more specifically, microcirculatory flow to the heart, brain, and adrenal glands: failure of these organs will result in rapid death. In the early stages of shock, there are only very subtle clinical signs and symptoms of hypoperfusion or no signs at all. Vital signs may change temporarily. For example, heart rate and respiratory rate may increase, and the MAP may fall somewhat, but compensatory mechanisms are able to return these values to baseline. Research in this area has demonstrated that lactic acidosis begins to increase in the initial stage of shock. When clinical signs are normal, the serum lactate level is already on the rise.

Compensatory Stage

The compensatory stage of shock begins as the body uses its own physiologic mechanisms in an attempt to maintain cellular homeostasis. Compensatory mechanisms can be divided into neural, hormonal, and chemical mechanisms. The sympathetic nervous system controls these three mechanisms. The neural mechanism is typically thought of first because it can be reflected in the vital signs and physical assessment as increased heart rate, increased contractility, and vasoconstriction, which help shunt blood to vital organs. In reality, these compensatory mechanisms occur simultaneously to restore circulating volume and tissue oxygenation. Common signs and symptoms of compensatory shock are as subtle as mild tachypnea and tachycardia. The MAP may decrease 10 to 15 mm Hg from baseline. Cardiac output may also drop slightly. In compensatory shock, the body is still doing a great job of using its neural, hormonal, and chemical mechanisms in attempt to correct the problem, making it sometimes difficult to recognize the patient in shock **Figure 9-4**, **Figure 9-5**, and **Figure 9-6**.

In compensatory shock, oxygen delivery to the tissues is already severely reduced. To compensate for this imbalance, cells try to increase oxygen consumption by pulling more oxygen from the capillary bed. However, the cells are already working in an oxygen-deprived environment. Trying to work harder in this anaerobic environment produces more lactic acid and carbon dioxide. Chemoreceptors, strategically located in the carotid arteries and aortic arch, recognize an increase in carbon dioxide levels and relay this information to the brain, which, in turn, stimulates an increase in the respiratory rate. A patient in compensatory shock becomes tachypneic in an attempt to take in more oxygen while concurrently exhaling excess carbon dioxide. Blowing off carbon dioxide is a compensatory mechanism to raise the acidic pH caused by lactic acidosis and hypercarbia.

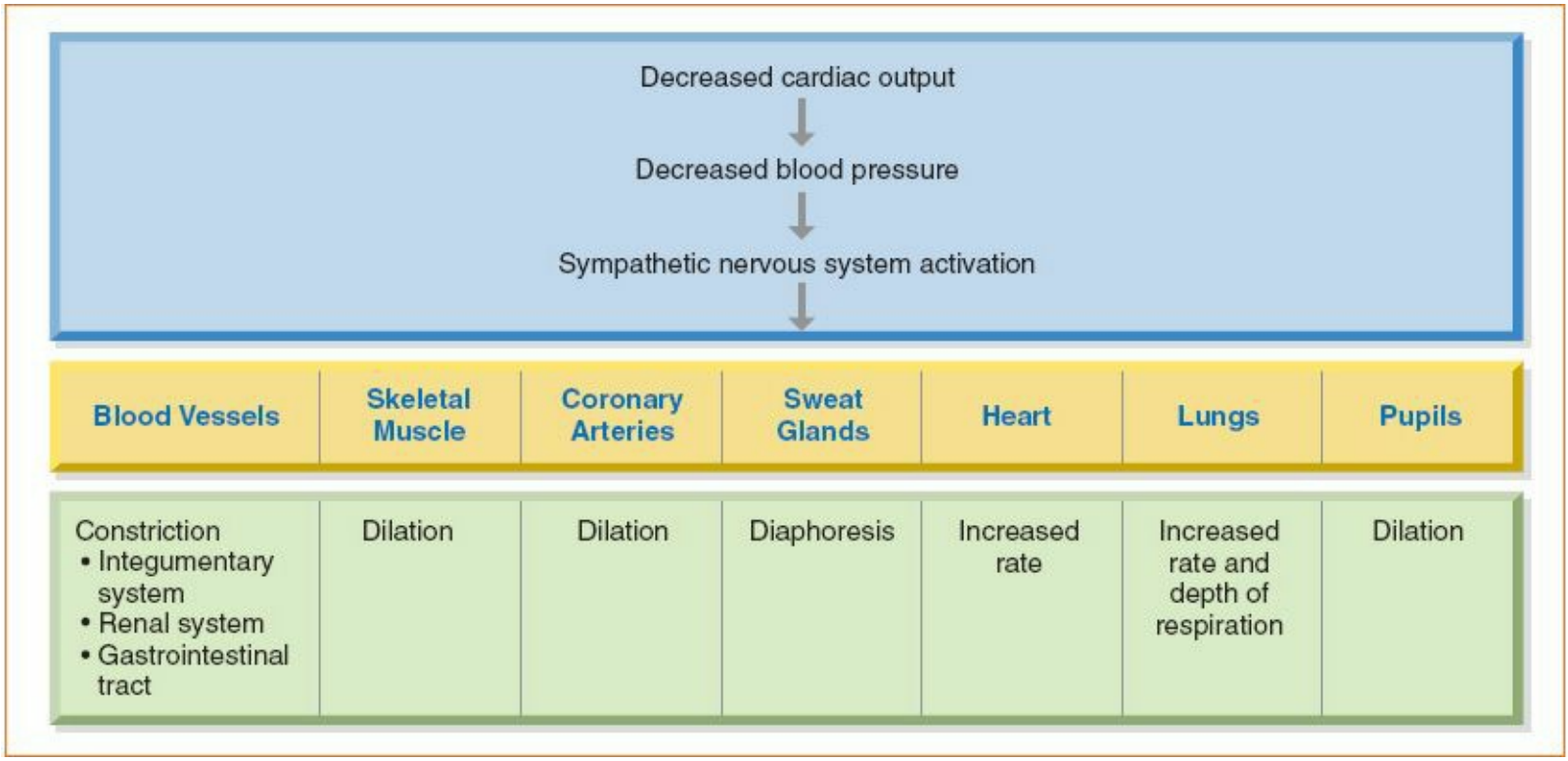

Figure 9-4 Neural compensation.

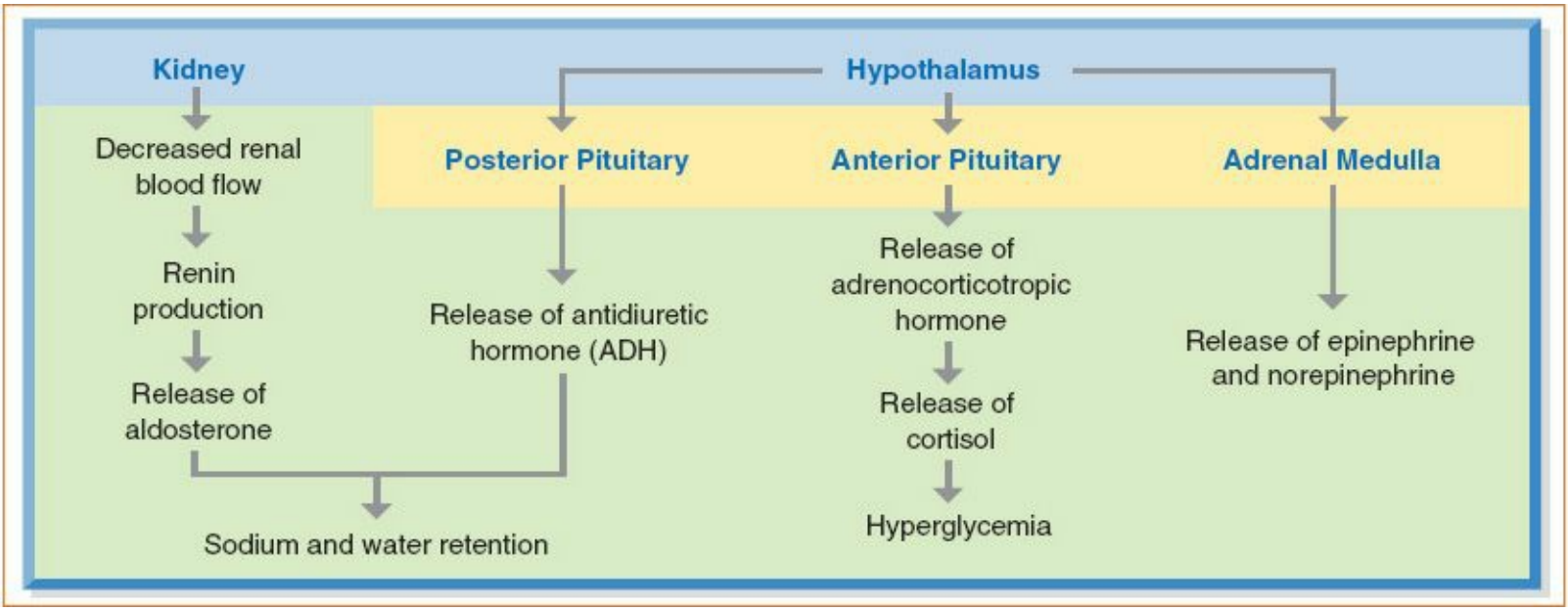

Figure 9-5 Hormonal compensation.

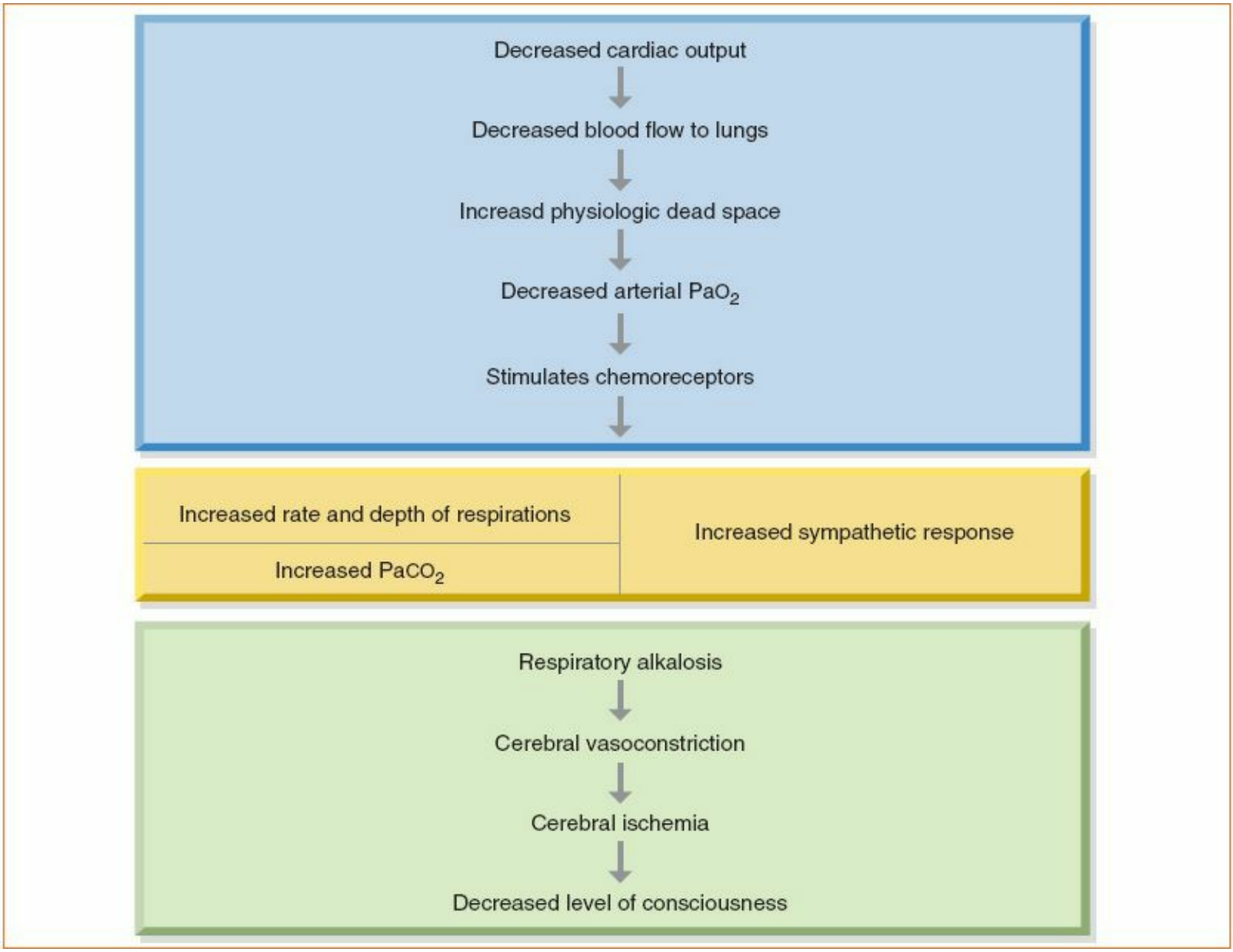

Figure 9-6 Chemical compensation.

While the respiratory system is working to balance pH and pull in more oxygen while exhaling waste, the heart and circulatory system are simultaneously working to maintain cardiac output. When the stroke volume and MAP begin to fall, baroreceptors in the carotid sinus and aortic arch recognize the change in pressure and send a signal to the vasomotor receptors in the medulla. The autonomic nervous system is then activated, stimulating the sympathetic nervous system and the anterior pituitary to release epinephrine and norepinephrine from the adrenal glands. These hormones work together to increase MAP and CO through increased heart rate, contractility, and peripheral vasoconstriction.

The renin-angiotensin-aldosterone system is a hormonal compensatory mechanism that is activated in response to low arterial pressures. Decreased blood pressures stimulate the release of renin, which stimulates the conversion of angiotensinogen to angiotensin I and, finally, angiotensin II. Angiotensin II increases arterial blood pressure through three mechanisms: vasoconstriction, stimulation of the thirst receptors in the hypothalamus, and stimulation of the release of aldosterone from the adrenal glands. Aldosterone increases circulatory volume by stimulating the reabsorption of sodium and water in the renal tubules.

Low arterial pressures also trigger the posterior pituitary gland to release antidiuretic hormone. This hormone increases water reabsorption within the kidneys and assists the adrenal hormones in

vasoconstriction to nonvital areas of the body. When vasoconstriction limits the supply of oxygen and blood flow to peripheral areas, capillary fluid shifts result. Capillary pressure is dependent on an adequate perfusion pressure (MAP). When capillary pressure drops, the pressure balance within the capillary bed is lost. The physiology of the capillary pressures reveals that hydrostatic pressure pushes fluid out and osmotic pressure pulls fluid back in. Normally, the hydrostatic pressure at the arterial end of the capillary is higher than the osmotic pressure, causing interstitial fluid to filter through the capillary endothelium into the interstitial space. At the venous end of the capillary, the hydrostatic pressure is lower than the osmotic pressure, causing fluid to diffuse back into the capillary. When this normal pressure balance is lost, edema may result. Edema results from any disturbance that alters the regulation of fluid transfer in the capillary and interstitial space. These disturbances include inflammation (more porous capillary), decreased colloid osmotic pressure, increased capillary hydrostatic pressure, and lymphatic channel obstruction.

The ability of the body to compensate for shock often masks what is occurring at the cellular level. Compensatory mechanisms can cover up a low circulatory volume, poor stroke volume, and inadequate CO before any noticeable changes in the blood pressure are identified, especially in children and healthy young adults. Laboratory studies may illustrate a clearer picture of what is going on inside the body. The ABGs show a low pH indicative of acidosis. The lactate level continues to climb, and hyperkalemia develops.

The transport treatment of compensated shock includes administering high-flow oxygen, performing good ventilator management, and, possibly, providing fluid replacement, depending on the cause.

Progressive or Decompensatory Stage

Shock will progress to an uncompensated state if the underlying cause remains untreated. At this stage, compensatory mechanisms fail to supply the much-needed oxygen to the cells. Patients have more pronounced signs of shock, including a drop in the MAP of more than 20 mm Hg from baseline, altered mental status, and tachycardia. Laboratory analysis continues to show a progression of acidosis, hyperkalemia, and a climbing lactate level.

This clinical picture develops for many reasons. Hypotension becomes more prominent as the microcirculation loses its ability to autoregulate, leading to increased capillary permeability. Blood starts to pool in the capillary bed, increasing hydrostatic pressure. The marked change in hydrostatic pressure within the microcirculation leads to edema and third spacing, decreasing venous return to the heart, which can be noted in a patient in shock by the presence of mottled skin.

Hemodynamic effects of this decreased preload are reflected in the blood pressure as hypotension and in the pulmonary artery (Swan-Ganz) catheter readings as decreased central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP). The CVP is a direct measurement of right atrial pressure and an indirect measurement of the preload of the right ventricle (right ventricular end-diastolic pressure). The PCWP reflects the left atrial pressure. At end diastole, pressure equalizes between the left atrium and the left ventricle; therefore, the PCWP can be used as an indirect measurement of the left ventricular pressure. The CVP and PCWP are discussed in more depth in Chapter 14.

Altered blood flow to the capillary bed causes microemboli to develop, which increases ischemia in the tissues and organs. Furthermore, the microclots stimulate the consumption of clotting factors, which may lead to **disseminated intravascular coagulation (DIC)**, a complex coagulopathy that can develop in critically ill patients for a variety of reasons. The patient's underlying condition triggers the coagulation cascade, resulting in microclots. These clots obstruct blood flow through the capillaries of organs and tissues, and microcirculatory thrombosis leads to tissue and organ ischemia, infarction, and organ dysfunction. Simultaneous fibrinolysis occurs in an attempt to reopen the microcirculation. The result is

bleeding *and* thrombosis. During the process, stimulation of the clotting cascade (discussed later in this chapter) rapidly depletes platelets and other coagulation factors, using them faster than the body can replace them. This situation leaves the body vulnerable to bleeding, or hemorrhage.

Hypotension is also a by-product of increased anaerobic metabolism. Anaerobic metabolism is self-limiting, and the cells cannot live in this type of environment for long. Anaerobic metabolism leads to a more acidic environment. Eventually, this environment leads to rupture of the lysosomes, which then spill their content into the cellular cytoplasm, leading to an even more acidic environment. The sodium-potassium pump then begins to fail. Sodium ions begin to accumulate within the cell. When this occurs, fluid is drawn into the cell, causing cellular swelling and, eventually, destruction of the cellular membrane. This is an important consideration in patient management because a number of devastating effects, such as cardiac arrhythmias, can follow. As cellular damage becomes more pronounced, cells start to die and release toxins into the microcirculation. For example, lysosomes, which normally defend cells from invaders, rupture and unleash their digestive enzymes, causing further damage within the microcirculation. Cell mediators attempt to counterattack this release and initiate the inflammatory response. Shock is a widespread condition, and the inflammatory response is not isolated to a specific area but is found throughout the body. When various complex compensatory components fail, vasodilation, smooth muscle contraction, and increased tissue permeability result in hypotension. Unfortunately, this cascade of events only gets worse as clotting mechanisms are activated in response to cell breakdown. The inflammatory response is discussed in more detail in the section on sepsis. These multifaceted systemic responses to cell breakdown can be summarized in a process known as SIRS, discussed in detail later in this chapter.

The progressive stage of shock is a life-threatening emergency requiring immediate treatment. Patients have a chance for recovery, even though the mortality rate increases with each stage of shock. Treatment is targeted at the underlying cause of shock. However, the effects of shock can be seen in nearly every organ **Table 9-3**. This is why fluid resuscitation and, if needed, vasopressor support are necessary early.

Refractory or Irreversible Stage

When a patient reaches the **refractory stage** of shock, compensatory mechanisms have failed. Anaerobic metabolism progresses to permanent organ dysfunction as cells die. Treatment options fail to reverse the mass effects of this stage in shock. Multiple organ failure ensues, and the mortality rate is at its highest.

A patient in the refractory or irreversible stage is unresponsive to verbal stimuli, blood pressure will be inadequate at less than 90 mm Hg systolic, heart rate will be increased, respiratory rate will be increased, and respirations will be shallow. The skin will be cold, cyanotic, and/or mottled, and peripheral pulses will be weak and thready to absent. Urinary output will decrease to less than 20 mL/h, and bowel sounds will be absent.

TABLE 9-3 Effects of Shock on the Body

Organ or System Affected	Progressive Result of Ischemia
Heart	Arrhythmias Cardiac failure
Lungs	Pulmonary hypertension Pulmonary edema Acute respiratory distress syndrome
Brain	Ischemia or infarction

Kidney	Decreased urine output Acute tubular necrosis Renal failure
Gastrointestinal	Stress ulcer Ischemic bowel with release of toxins
Pancreas	Ischemia Myocardial depressant factor released
Liver	Hypoglycemia Toxicity
Hematologic and immune	Initial leukocytosis followed by leukopenia Systemic inflammatory response DIC
Adrenal glands	Adrenal insufficiency, leading to hypotension, and hypoglycemia

Multiple Organ Dysfunction Syndrome

Progressive cell death will result in tissue death and eventual organ failure. When two or more organs stop functioning, **multiple organ dysfunction syndrome (MODS)** is diagnosed. MODS can be classified as primary or secondary. **Primary MODS** results from a direct insult such as trauma. **Secondary MODS** is the more common cause of organ failure and is a slower, more progressive insult to organs. It frequently results from the sepsis cascade. Mortality rates are high in patients with MODS. Patients with two failing organs have a 22% to 33% chance of recovery. When four or more organs are involved, the mortality rate is nearly 100%.

The organs affected early in MODS are the brain, kidneys, liver, adrenal glands, and heart. The kidneys are dependent on having adequate perfusion pressure. Once the MAP drops, the kidneys stop functioning. Oliguria occurs early in shock. Diagnostic studies reflective of renal function show elevated blood urea nitrogen (BUN) and creatinine levels. Many patients need continuous bedside dialysis when renal failure develops. Renal failure by itself increases the risk of death by 30%.

The liver is a complex organ with a lead role in detoxifying and excreting wastes and toxins. It is essential for the synthesis of blood proteins and coagulation proteins, along with the storage of glycogen, iron, and vitamins. When the liver stops working, the body has lost a key manager of the homeostatic process. Laboratory studies in patients with MODS reflect elevated levels of liver enzymes: aspartate aminotransferase, alanine aminotransferase, and total bilirubin. Unfortunately, there are no definitive treatments for liver failure. All treatments are focused on minimizing the effects of the liver damage.

Nearly every course in health care recognizes that a patient's level of consciousness deteriorates quickly in hypoxic states. This is taken to the extreme in MODS. The brain is also considered a peripheral organ and will gladly sacrifice itself in the hope of maintaining adequate blood pressure. As hypoxia ensues, the brain experiences ischemia, and many patients have permanent deficits from anoxic brain injury.

The heart potentially suffers more than any other organ. As the heart struggles to maintain arterial perfusion, it, too, undergoes hypoxia. Hypotension is unable to be managed despite fluids and vasopressors. Compensatory tachycardia cannot exist without oxygen, and bradycardic arrhythmias occur or arrhythmias such as ventricular tachycardia and ventricular fibrillation develop. Relentlessly, the shy pancreas also adds to the progression of cardiac failure. As the pancreas becomes more ischemic, it releases myocardial depressant factor, further altering the ability of the myocardium to contract.

Table 9-4 summarizes common organs affected by hypoperfusion and associated signs.

TABLE 9-4 Common Organs Affected by Hypoperfusion and Associated Signs

System or Organ	Signs of Dysfunction
Neurologic	Altered level of consciousness Confusion
Pulmonary	Tachypnea Hypoxemia • PaO_2 < 70 mm Hg • SaO_2 < 90% • PaO_2/FIO_2 ratio < 300 Respiratory alkalosis
Cardiovascular	Tachycardia Hypotension Altered hemodynamics • Decreased systemic vascular resistance • Decreased cardiac output • Decreased CVP Arteriovenous shunting • Cellular hypoxia • Lactic acidosis
Renal	Oliguria Anuria Elevated creatinine level
Hepatic	Jaundice Elevated enzyme levels Decreased albumin level Prolonged PT
Hematologic	Decreased platelet count Prolonged PT/activated partial thromboplastin time Decreased protein C level Elevated D-dimer level
Adrenal glands	Hypotension Hypoglycemia

Abbreviations: CVP, central venous pressure; PT, prothrombin time.

Classification of Shock

Shock is traditionally classified by cause. The three primary classifications of shock coincide with the conditions that cause them: cardiogenic, hypovolemic, and distributive. Distributive shock, which is

characterized as a relative hypovolemia, can be further broken down into chemical and neural causes. More than one type of shock may be present at the same time. For example, many trauma patients present with hypovolemic shock from sustained injuries. However, these patients may also have cardiogenic or neurogenic shock.

Cardiogenic Shock

Cardiogenic shock is failure of the heart to pump blood effectively. The definition of cardiogenic shock includes the following:

- A sustained systolic blood pressure of 80 to 90 mm Hg for greater than 30 minutes
- An MAP 30 mm Hg below baseline or less than 65 mm Hg over time
- A severe reduction in the **cardiac index** (< 1.8 L/min/m^2 without support or < 2.0 to 2.2 L/min/m^2 with support)
- Elevated ventricular filling pressure (ie, left ventricular filling pressure of > 18 mm Hg or right ventricular filling pressure of > 10 to 15 mm Hg)

The diagnosis of cardiogenic shock is usually made using a PA catheter, but some institutions make the diagnosis with echocardiography, which is an acceptable alternative.

Cardiogenic shock can be the result of a variety of causes. The most common cause of cardiogenic shock is left ventricular failure as a result of a large myocardial infarction (MI). Other causes of cardiogenic shock may be right ventricular failure, valvular disorders, cardiomyopathies, septal defects, papillary muscle rupture, and sustained arrhythmias. The causes may be classified as intrinsic or extrinsic, with the aforementioned examples being intrinsic causes. These conditions manifest with poor contractility, decreased cardiac output, or impaired ventricular filling. Extrinsic causes of cardiogenic shock include pericardial tamponade, effusion, pulmonary emboli, and tension pneumothorax. Some experts, including the American Heart Association, classify extrinsic causes as cardiogenic shock because there is impaired filling or emptying of the ventricles or a combination of the two. The most common cause of cardiogenic shock is an acute MI (AMI) accompanied by 40% dysfunction of the left ventricle. Noncoronary causes include myocardial contusion, pericardial tamponade, ventricular rupture, and pulmonary embolus. Cardiogenic shock may occur in approximately 5% to 8% of patients admitted to the hospital following an MI. Populations at the greatest risk of developing cardiogenic shock are elderly patients, patients with a history of diabetes mellitus, and patients with a history of MI with an **ejection fraction** of less than 35%. Risk stratification of patients in cardiogenic shock formerly was very high, with a mortality rate as high as 80%. Such a high mortality rate led to a very grim prognosis with little chance of survivability. Newer treatment modalities have greatly improved the risk stratification, and the prognosis is no longer grim. Patients with cardiogenic shock now have an excellent chance for long-term survival. Recent data suggest that the average mortality rate for cardiogenic shock is now approximately 50%. The mortality rate varies based on risk factors and demographics. Modern treatment modalities include early percutaneous coronary interventions, early revascularization, hemodynamic management with PA catheters, mechanical support with the intra-aortic balloon pump, and total circulatory support through the use of left-ventricular assist devices, biventricular assist devices, and extracorporeal life support, all of which are discussed in Chapter 15.

Cardiogenic shock results when the ventricles are unable to pump blood forward. Because the ventricles are unable to contract normally, blood remains in the ventricles. As a result, stroke volume is reduced, ultimately decreasing CO. The five major determinants of myocardial oxygen consumption are contractility, preload, wall tension, afterload, and heart rate. With inadequate oxygen supply to the periphery, cells are forced to function in anaerobic conditions, enabling the cascade of events leading to

SIRS and MODS.

Manifestations of cardiogenic shock vary depending on the underlying cause **Table 9-5**. Initially, the patient exhibits signs and symptoms associated with poor CO and low blood pressure. Both right and left heart failure result in hypotension, reflected as a systolic blood pressure of less than 90 mm Hg for a sustained period or an MAP 30 mm Hg below baseline. Altered mental status; cool, pale, diaphoretic skin; and a drop in urine output develop. The first compensatory mechanism to present itself is tachycardia. However, the heart is not able to sustain or improve CO, and the pulse becomes weak and thready. Heart sounds may be distant, or abnormal heart sounds may appear as S_3 or S_4. These extra heart sounds develop from unsynchronized closure of the valves. Preload is elevated from the inability of blood to push forward. This lack of forward movement may cause fluid accumulation in the lungs (acute pulmonary edema), the periphery (jugular venous distention), or both, depending on the ventricle(s) affected. Hypotension becomes more pronounced as compensatory mechanisms create systemic vasoconstriction. This increases afterload, which increases the pressure the already struggling myocardium must pump against.

Controversies

The popular notion that patients with heart failure have rales has recently been challenged. In fact, most patients with chronic heart failure do not have rales. The most reliable sign of volume overload is jugular venous distention. When rales are present in conjunction with other signs of congestion, heart failure is likely. Rales reflect the rapidity of onset of heart failure. Fully 50% of patients with chronic heart failure experience periods of clinically significant volume overload without peripheral edema or rales.

In addition to a systolic blood pressure of less than 90 mm Hg or an MAP of 30 mm Hg below the patient's baseline, other hemodynamic patterns in cardiogenic shock include a narrow pulse pressure, a cardiac index of less than 1.8 L/min/m^2 without support or 2.0 to 2.2 L/min/m^2 with support, and PCWP as measured by a pulmonary artery (Swan-Ganz) catheter of greater than 18 mm Hg. If the right ventricle is affected, the right atrial pressure (RAP) and CVP will also be elevated. Analysis of ABGs may reflect hypoxemia with a low Pa_{O_2} level and a corresponding metabolic acidosis with compensatory respiratory alkalosis. Acidosis develops as a result of poor perfusion due to cardiac dysfunction and the inability of the heart to pump oxygen to the peripheral tissues. Respiratory alkalosis is the compensatory mechanism that develops as the patient becomes tachypneic in attempts to blow off the excess carbon dioxide from increased anaerobic metabolism. Eventually, this compensatory mechanism will fail and the ABGs will reflect metabolic and respiratory acidosis.

TABLE 9-5 Findings in Cardiogenic Shock

	Left Ventricular Heart Failure	Right Ventricular Heart Failure	Biventricular Heart Failure
Heart rate	Increased	Increased	Increased or decreased
Blood pressure	WNL in the initial stage, then decreases as patient decompensates	WNL in the initial stage, then decreases as patient decompensates	Decreased
Pulse pressure	Narrow	Narrow	Narrow
CVP/right atrial pressure	WNL	Increased	Increased
PCWP	Increased	Decreased	Increased
Cardiac output/cardiac index	Decreased	Decreased	Decreased
SVR	Increased	Increased	Increased
Svo_2	Decreased	Decreased	Decreased
Urinary output	Decreased	Decreased	Decreased
Jugular vein distention	Absent	Present	Present
Heart sounds	S_3	Normal	S_3 or S_4
Edema	Pulmonary	Peripheral	Systemic

Abbreviations: CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; SVR, systemic vascular resistance; WNL, within normal limits.

Signs and Symptoms

Cardiogenic Shock

- Poor CO
- Low blood pressure (systolic blood pressure of < 90 mm Hg or mean arterial pressure 30 mm Hg below baseline)
- Altered mental status
- Cool, pale, diaphoretic skin
- Decreased urine output
- Weak and thready pulse
- Distant or abnormal S_3 or S_4 heart sounds
- Fluid accumulation in the lungs or limbs (for example, jugular vein distention or pedal edema)
- Tachypnea

Differential Diagnosis

Cardiogenic Shock

- Aortic dissection
- Coronary artery disease
- MI
- Pulmonary embolus
- Acute myocarditis

Transport Management

Cardiogenic Shock

- Perform simple procedures to relieve the cause, including the following:
  - For cardiac tamponade, perform pericardiocentesis if allowed per protocols.
  - For tension pneumothorax, also perform needle decompression and chest tube therapy.
- Provide high-flow oxygen.
- Eliminate chest pain.
- Reduce anxiety.
- Perform hemodynamic and cardiac monitoring.
- Provide antiarrhythmics if arrhythmias are present.
- Provide judicious fluid therapy.
- Consider administering vasopressors to physician-ordered parameters.
- Consider administering inotropic agents to physician-ordered parameters.
  - Use with caution. Research shows that high doses of inotropic support lead to a higher incidence of mortality.
- Consider administering diuretics.
- Consider using agents to decrease afterload.
- Insert an indwelling urinary catheter to monitor urinary output.

Primary management of cardiogenic shock is focused on enhancing CO while decreasing left ventricular workload. If the underlying cause is known, simple procedures to relieve the cause of cardiogenic shock should be taken first. This may require pericardiocentesis for cardiac tamponade (if protocols allow), along with needle decompression, and chest tube therapy for tension pneumothorax. If the cause is an AMI, revascularization should be initiated early (although this is an in-hospital procedure and will not be performed during transport).

Figure 9-7 Administer IV fluids to the critical care shock patient judiciously.

High-flow oxygen therapy should be provided to any patient exhibiting signs of shock. One of the manifestations of cardiogenic shock is pulmonary edema, and when this occurs, mechanical ventilation should be considered as guided by ABG analysis. Supportive therapy is also necessary to eliminate chest pain and reduce anxiety. Arrhythmias are common with cardiogenic shock. Therefore, antiarrhythmics may be necessary to optimize cardiac function.

Fluid therapy is never wrong when done judiciously **Figure 9-7**. Isotonic crystalloids in small 200- to 250-mL (5- to 10-mL/kg) boluses may improve CO. In suspected cardiogenic shock, vital signs, lung

sounds, and heart sounds should be frequently reassessed after fluid boluses to measure adequacy of therapy. Excessive crystalloids may cause pulmonary edema or worsen existing pulmonary edema.

Inotropic support may be needed to improve contractility. Milrinone and dobutamine are two inotropic agents used to increase CO. These agents assist the heart by increasing the cardiac contractile force, thus improving blood pressure. Milrinone has inotropic effects and minimal chronotropic effects but is not a vasoconstrictive agent. Milrinone also causes pulmonary vasodilation and peripheral arterial vasodilation. However, owing to excessive hypotension with a loading dose, loading with milrinone should be avoided in acute heart failure. Dopamine is also used, but more as a supportive agent in conjunction with dobutamine or milrinone. However, dopamine, like all vasopressors, needs to be used with caution. In general, all vasopressors increase afterload through their vasoconstrictive properties and increase oxygen consumption by elevating heart rate and increasing force of contractility.

Preload and afterload reductions are primary concerns in cardiogenic shock. Diuretics are frequently administered to reduce CVP and PCWP. Once the blood pressure is stabilized, vasodilators are started. Venous vasodilators, such as nitroglycerin, reduce preload; however, this agent must be used with caution in patients with a systolic blood pressure of less than 100 mm Hg. Decreasing left ventricular workload with afterload reducers such as nitroprusside (Nipride) or milrinone is also important in improving CO.

When pharmacologic methods are contraindicated or fail to support the failing heart, other methods of afterload reduction are necessary. The intra-aortic balloon pump and ventricular assist device are two mechanical treatments for cardiogenic shock and are discussed in Chapter 15. The intra-aortic balloon pump is a large balloon catheter that lies in the thoracic aorta and inflates during diastole, displacing blood in the aorta. Balloon inflation forces blood in two directions, back toward the aortic arch and the coronary arteries and downward through the descending aorta, forcing blood to the peripheral circulation, including the renal and mesenteric arteries. The balloon deflates during systolic contraction, allowing the heart to pump against very low systemic pressures. If all of these methods fail, the alternatives to support the heart are limited. Extracorporeal membrane oxygenation and ventricular assist device therapies may be needed to provide temporary pressure and oxygenative support to a patient in cardiogenic shock.

Hypovolemic Shock

Hypovolemic shock is present when there is too little circulating blood volume within the vascular system, resulting in hypotension. Hypovolemia may be present when blood or fluid is lost or displaced internally or externally. Examples of conditions associated with hypovolemia are fever, vomiting and diarrhea, hemorrhage, burns, and excessive third spacing.

Manifestations of hypovolemic shock are evident early in shock. The initial stage of hypovolemic shock is characterized by low circulating volume with minimal signs of hypoperfusion. However, as the body begins to compensate for low venous return, decreased stroke volume, and low CO, patients begin to have tachycardia, hypotension, and signs of poor tissue perfusion, including pallor and delayed capillary refill. Patients become more confused and anxious as oxygen supply to the tissues is compromised and cellular metabolism is altered. Compensatory mechanisms continue to increase systemic vascular resistance (SVR) in an attempt to improve hemodynamics. As SVR increases and CO drops, patients have cold, mottled, and pulseless extremities with worsening mental status. Left untreated, the shock will eventually progress to decompensated shock that is refractory to any therapy **Table 9-6**.

TABLE 9-6 Findings in Hypovolemic Shock

Heart rate	Increased
Blood pressure	WNL in the early stages, then decreases as patient

	decompensates
CVP/RAP	Decreased
PCWP	Decreased
Cardiac output/cardiac index	Decreased
SVR/systemic vascular resistance index (SVRI)	Increased
Svo_2	Decreased
Urinary output	Decreased
Jugular vein distention	Flat
Hematocrit (percentage of whole blood components versus plasma)	Decreased (with hemorrhage) Increased (with dehydration)

Abbreviations: CVP, central venous pressure; RAP, renal artery pressure; SVR, systemic vascular resistance; WNL, within normal limits.

The hematocrit value decreases with hemorrhage. A fall in the hematocrit value after blood loss will happen after one to several hours with no fluids (as interstitial and cellular fluid shifts occur). The hematocrit value decreases faster with exogenous fluid challenges.

Hypovolemic shock caused by hemorrhagic trauma has been classified by the American College of Surgeons Committee on Trauma into four classes, each with their own specific characteristics and treatments **Table 9-7**.

Treatment of hypovolemic shock revolves around treating the underlying condition, administering oxygen, and initiating volume replacement. Isotonic crystalloids of normal saline or lactated Ringer's are the preferred solutions used in 250- to 500-mL increments to maintain blood pressure in low normal ranges. Many trauma surgeons prefer lactated Ringer's over normal saline because lactated Ringer's may help decrease the acidosis in patients with severe hemorrhagic hypovolemia. However, this belief remains controversial and either solution will benefit the patient. The use of hypertonic saline, 5 mL/kg of 7.5% sodium chloride, in 250-mL increments may also be effective in treating hypovolemic shock caused by hemorrhage. Studies have shown that hypertonic solutions may optimize blood pressure, CO, intracranial pressure, and microvascular flow without increasing bleeding or volume overloading patients. This is achieved by increasing the osmolarity within the vasculature, causing a shift of interstitial fluid into the vasculature. However, there has been no change in survival rates compared with standard fluid resuscitation. More research in this area may change the face of fluid therapy, but for now, most disciplines in the United States continue to use isotonic crystalloid therapy.

Signs and Symptoms

Hypovolemic Shock

- Tachycardia
- Hypotension
- Signs of poor tissue perfusion (pallor and delayed capillary refill)

- Confusion
- Anxiety
- Cold, mottled, and pulseless extremities
- Decreased mentation

Differential Diagnosis

Hypovolemic Shock

- Fever
- Dehydration
- Hemorrhage
- Burns
- Excessive third spacing

Transport Management

Hypovolemic Shock

- Treat the underlying condition.
- Administer oxygen.
- Maintain the airway and ensure optimal ventilation.
- Initiate volume replacement (normal saline or lactated Ringer's preferred, in 250- to 500-mL increments).
- Administer vasopressors if hypotension is refractory to fluid resuscitation.
- Administer blood products early if hemorrhage is suspected.

TABLE 9-7 Estimated Fluid and Blood Loss for a 70-kg Male

	Class I	Class II	Class III	Class IV
Blood loss (mL)	< 750	750-1,500	1,500-2,000	> 2,000
% Blood loss	< 15	15-30	30-40	> 40
Heart rate (beats/min)	< 100	> 100	> 120	> 140
Blood pressure	Within normal limits	Normal	Low	Low
Pulse pressure	Within normal limits	Narrow	Narrow	Very narrow
Capillary refill	Within normal limits	Delayed	Delayed	Absent
Respiratory rate (breaths/min)	14-20	20-30	30-40	> 35
Central nervous system/mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Skin condition	Cool, pink	Cool, pale	Cold, pale, moist	Cold, cyanotic
Urine output (mL/h)	> 30	20-30	5-15	Minimal or none
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Modified from: American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support for Doctors: ATLS Student Course Manual*. 8th ed. Chicago, IL: American College of Surgeons; 2009.

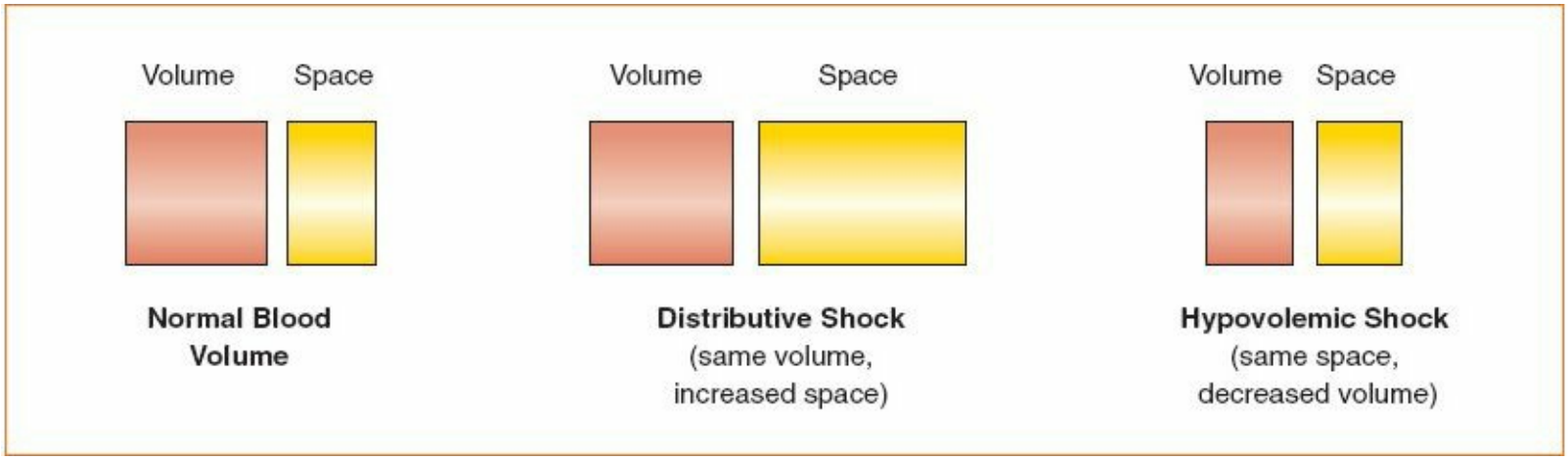

Figure 9-8 Conceptual illustration of distributive vs hypovolemic shock.

Blood products should be started early if hemorrhage is suspected. The American College of Surgeons recommends starting blood products in class III and IV hemorrhage after an initial 2 L of isotonic solution is administered. Colloids have not shown to be of any more benefit than crystalloids in improving patient outcome and, therefore, are not typically recommended.

The next section discusses distributive shock. **Figure 9-8** shows a conceptual illustration of distributive vs hypovolemic shock.

Distributive Shock

Distributive shock is a catch-all term used to describe several types of shock involving loss of vasomotor tone or increased vascular permeability; it includes neurogenic, anaphylactic, and septic shock. The human body has approximately 60,000 miles of blood vessels and 5 to 6 L of blood. Homeostasis involves constant regulation of vessel diameter and flow through capillary beds. When a disruption occurs in the vascular compartment causing widespread vasodilation and relative hypovolemia, distributive shock is present.

Neurogenic Shock

Neurogenic shock is one form of distributive shock caused by loss of sympathetic tone. When sympathetic tone is lost, there is substantial peripheral vasodilation, baroreceptor inhibition, and altered thermoregulation. This type of shock results from conditions that impede the ability of the sympathetic nervous system to control the constriction and dilation of vessel walls. Trauma to the brain or spinal cord is a common condition associated with disruption of sympathetic tone. Spinal cord injuries above the thoracolumbar spinal cord T6 level are the most common spinal cord injuries associated with neurogenic shock. Guly et al found that neurogenic shock was present in 19% of patients with cervical cord injuries and 7% of patients with thoracic and lumbar cord injuries. In the thoracolumbar spinal cord T6 level, the spinal space is narrower, and almost all the space is occupied by nervous tissue. The bony protection is more fragile and the head freely mobile. Neurogenic shock is not limited to trauma. Many pharmacologic agents, such as general and spinal anesthetics, opiates, and barbiturates; insulin shock; various toxins; and extreme parasympathetic stimulation, may also be responsible for neurogenic shock.

Table 9-8 offers a useful reference to vital signs associated with spinal trauma.

Neurogenic shock is characterized by relative hypovolemia as the result of vasodilation. The amount of fluid in the vasculature remains constant, but the container size is increased. As a result, there is a significant drop in the SVR and blood pressure. The signs and symptoms associated with neurogenic shock can be very different from those found in hypovolemic shock **Table 9-9**. One profound difference is

the presence of bradycardia in the face of hypotension. This is caused by the combined loss of sympathetic tone and baroreceptor response. However, the presence of tachycardia with hypotension does not exclude neurogenic shock. It is vital to piece together all possible causes of hypotension to determine the best treatment.

Treatment for neurogenic shock is focused on maintaining an MAP greater than 70 mm Hg or a systolic blood pressure at greater than 90 mm Hg. Fluid therapy with isotonic crystalloids should be started. If large volumes of crystalloid solutions are required to maintain the blood pressure, colloids should be considered. Colloids stay in the vascular system longer than crystalloids and, because of their high colloid osmotic pressure, are able to pull fluid from the cells and interstitial spaces back into the vascular system. Inotropic agents and vasopressors will need to be started if traditional fluid resuscitation is insufficient to maintain MAP. Dopamine, norepinephrine (Levophed), and phenylephrine (Neo-Synephrine) are common vasopressor agents used in neurogenic shock. Norepinephrine and phenylephrine raise the blood pressure by directly stimulating the alpha-adrenergic receptors in the smooth muscle of blood vessels, causing vasoconstriction. Dopamine at moderate doses stimulates the beta 1–adrenergic receptors, increasing the force of contraction and, therefore, increasing CO. Higher dopamine doses stimulate the alpha-adrenergic receptors, mimicking the effects of norepinephrine.

Source: Reprinted from *Resuscitation*, H. R. Guly, et al., The incidence of neurogenic shock in patients with isolated spinal cord injury in the emergency department, pp. 57-62, Copyright (2007), with permission from Elsevier. [http://www.sciencedirect.com/science/journal/03009572]

TABLE 9-9 Hemodynamic Findings in Distributive Shock Compared With Hypovolemic Shock

Hemodynamic Findings in Distributive Shock			Comparison With Hypovolemic Shock
	Neurogenic Shock	Anaphylactic Shock	
Heart rate	Decreased	Increased	Increased
Blood pressure	Decreased	Decreased	Within normal limits in the initial stage, then decreases as patient decompensates
Pulse pressure	Normal	Narrow	Narrow
CVP/RAP	Decreased	Decreased	Decreased
PCWP	Decreased	Decreased	Decreased
Cardiac output/cardiac index	Decreased	Decreased	Decreased
SVR/SVRI	Decreased	Decreased	Increased
Svo_2	Decreased	Decreased	Decreased
Urinary output	Decreased	Decreased	Decreased
Skin	Pink, warm, dry	Flushed, warm to hot, pruritis, hives	Cool, pale, moist

Abbreviations: CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; RAP, renal artery pressure; SVR, systemic vascular resistance; SVRI, systemic vascular resistance index.

Signs and Symptoms

Neurogenic Shock

- Relative hypovolemia as the result of vasodilation
- Bradycardia

- Significant drop in blood pressure
- Normal pulse pressure
- Decreased CVP/RAP
- Decreased PCWP
- Decreased CO/cardiac index
- Significant drop in the SVR/SVRI
- Decreased Svo_2
- Decreased urinary output
- Pink, warm, dry skin

Differential Diagnosis

Neurogenic Shock

- Loss of sympathetic tone
- Trauma to the brain or spinal cord
- Pharmacologic agents such as general and spinal anesthetics, opiates, and barbiturates; insulin shock; various toxins; and extreme parasympathetic stimulation

Transport Management

Neurogenic Shock

- Maintain MAP at greater than 70 mm Hg and/or systolic blood pressure at greater than 90 mm Hg.
- Consider administering vasopressors (dopamine, norepinephrine, phenylephrine). Administer inotropic agents and vasopressors if traditional fluid resuscitation is insufficient to maintain MAP.
- Initiate fluid therapy with isotonic crystalloids.
- Consider colloids if large volumes of crystalloid solutions are required to maintain blood pressure.
- Maintain the heart rate (increase heart rate via pacemaker therapy or atropine administration).

The control of heart rate is also important in neurogenic shock. Loss of sympathetic tone may result in profound bradycardia. Methods for increasing heart rate involve pacemaker therapy or atropine. Atropine works by blocking acetylcholine (muscarinic) receptors, creating a temporary increase in heart rate.

Transport considerations for neurogenic shock center on ensuring a patent airway, adequate ventilation, and oxygenation because, depending on the level of involvement, the patient may be unable to breathe without assistance or may have impaired ventilatory capabilities. Care includes cardiac monitoring, administration of IV fluids and vasopressor medications as ordered, and maintenance of spinal immobilization. The patient also needs to have an indwelling urinary catheter.

Anaphylactic Shock

Anaphylactic shock is a severe, life-threatening allergic reaction producing systemic vasodilation in response to histamine release. The list of possible antigens in humans is extensive and includes diagnostic agents, foods, bites, stings, chemical agents, and several classes of medications, such as antibiotics,

immunologic agents, anesthetics, anti-inflammatory agents, narcotics, and hormones. There are two types of anaphylactic shock: true and anaphylactoid. **True anaphylaxis** results when the allergen binds to IgE on the cell membranes of basophils and mast cells, stimulating the release of histamine from the cell. An **anaphylactoid reaction** is a non–IgE-mediated response that causes the rupture of mast cells and basophils, which then release histamine and other defense mediators. Non–IgE-mediated responses can give the same reactions as true shock without prior exposure. Either reaction can be massive and can happen in minutes after exposure, or the response may be delayed for hours or even days and is then called a delayed hypersensitivity response. Histamine, a cell mediator, is responsible for relative hypovolemia caused by widespread vasodilation, increased capillary permeability, and smooth muscle constriction. This smooth muscle constriction is responsible for the bronchoconstriction, laryngeal edema, and angioedema seen with anaphylaxis. As a result, the airway may become quickly compromised.

Rapid treatment is necessary to limit the effects of anaphylaxis. Standard oxygen therapy may not be enough. Patients may need to be intubated, or they may require an emergency cricothyrotomy or tracheostomy to maintain an adequate airway.

Treatment is focused on removing the allergen that caused the severe reaction, including discontinuing the medication causing the reaction or simply removing a stinger after a bee sting. Drug therapy for anaphylaxis requires supporting the cardiovascular system and preventing further mediator release. Epinephrine is the drug of choice for anaphylactic shock because it acts as a physiologic antagonist to the **leukotrienes** and other cell mediators released in anaphylaxis. Epinephrine increases blood pressure through stimulation of alpha-1 receptors, leading to vasoconstriction. It also has bronchodilation effects by activation of the beta-2 receptors. The dose of epinephrine is 0.3 to 0.5 mg (0.3 to 0.5 mL) of a 1:1,000 solution subcutaneously or 0.3 to 0.5 mg (0.3 to 0.5 mL) of a 1:10,000 solution IV. If hypotension persists, an epinephrine drip may need to be started. Epinephrine may not work on patients taking beta-blockers. In these cases, glucagon may be given IV at 5 to 15 μg/min. Glucagon is effective in treating anaphylaxis by increasing heart rate and contractility.

Owing to the increased container size, fluid therapy needs to be started early to maintain an MAP of greater than 70 mm Hg (or a systolic blood pressure of greater than 90 mm Hg). Normal saline should be used to sustain adequate preload volumes. Vasopressor agents may be started if crystalloid therapy is inadequate for maintaining CO.

Other pharmacologic agents are beneficial for treating anaphylactic shock. Antihistamines are routinely used in the treatment of allergic reactions. Diphenhydramine (Benadryl) is used to block histamine release from the H_1 receptors on smooth muscles, gland cells, and some nerve endings. The dose of diphenhydramine is 1 to 2 mg/kg IV, repeated every 4 to 8 hours, but not exceeding 50 mg. Ranitidine (Zantac), an H_2 blocker, is sometimes used to treat severe allergic reactions because of its effects on the heart, decreasing stimulation and decreasing acid secretion within the stomach. Bronchodilators are administered to relieve the shortness of breath associated with anaphylaxis. Albuterol is a beta 2–selective bronchodilator that works as a physiologic antagonist toward leukotrienes and other inflammatory mediators, thereby relaxing smooth muscle. Corticosteroids are given to stabilize the capillary membrane, thus offsetting the inflammatory response and preventing a delayed reaction. Corticosteroids decrease swelling and inflammation in the airway and also decrease IgE-mediated responses that may occur hours after the initial event. Approximately 20% of anaphylactic reactions follow a biphasic course, with an asymptomatic period for 1 to 8 hours after the initial event. After this 1- to 8-hour period, there can be a recurrence of the reaction. Recurrence has even been reported as late as 24 to 38 hours after treatment for the initial episode. (It is possible for a patient to experience a recurrence later than the 1- to 8-hour period following initial onset.) About one third of the second-phase reactions are more severe than the initial event. Another third are equally severe as the initial event, and

the remaining third are less severe. Corticosteroid administration can prevent or minimize these second-phase reactions.

Signs and Symptoms

Anaphylactic Shock

- Increased heart rate
- Decreased blood pressure
- Narrow pulse pressure
- Decreased CVP/RAP
- Decreased PCWP
- Decreased CO/cardiac index
- Decreased SVR/SVRI
- Decreased Svo_2
- Decreased urinary output
- Flushed skin that is warm to hot, with pruritis and hives
- Bronchoconstriction
- Laryngeal edema
- Angioedema
- Airway compromise

Differential Diagnosis

Anaphylactic Shock

- Allergic reaction (from a triggering substance such as a diagnostic agent, specific food, bite, sting, chemical agent, or medication [antibiotics, immunologic agents, anesthetics, anti-inflammatory agents, narcotics, and hormones]
- Massive anaphylactic reaction that occurs in minutes, or lesser reaction that occurs over hours or days

Transport Management

Anaphylactic Shock

- Administer oxygen.
- Maintain an adequate airway.
- Remove the allergen that caused the reaction.
- Administer epinephrine.
- If patient is taking beta-blockers, administer glucagon.
- Initiate fluid therapy early with normal saline to maintain an MAP of greater than 70 mm Hg or a systolic blood pressure of greater than 90 mm Hg.
- Administer vasopressor agents if crystalloid therapy is inadequate for maintaining CO.

- Consider other pharmacologic treatments: antihistamines, ranitidine (Zantac, an H_2 blocker), bronchodilators (such as albuterol, a beta 2–selective bronchodilator), and corticosteroids are other options.
- Perform cardiac monitoring.
- Observe for a possible second-phase reaction.

SIRS, Sepsis, and Septic Shock

The third form of distributive shock is called septic shock. Septic shock is a progressive disease that typically results from an infection. Septic shock usually starts with a localized infection and then develops into a widespread inflammatory process, more commonly referred to as systemic inflammatory response syndrome, or SIRS. When hypotension develops, septic shock is present. Infection is the leading cause of SIRS and septic shock. However, any shock state can cause the beginning of SIRS and lead to sepsis before reaching multiple organ failure. Sepsis is the leading cause of MODS. It is for this reason that more time is spent discussing the sepsis process.

Sepsis

The terminology associated with sepsis continues to evolve. In 1992, the American College of Chest Physicians and the Society of Critical Care Medicine published definitions for **systemic inflammatory response syndrome (SIRS)** and sepsis as a result of a consensus conference on sepsis and organ failure. This meeting established basic criteria for early recognition and diagnosis of SIRS. Another meeting in 2001 established new criteria to better diagnose and classify patients with sepsis-associated conditions in the hospital. Sepsis is defined as having SIRS with a known or highly suspected source of infection.

SIRS is defined as the widespread inflammatory process associated with infectious and noninfectious causes. Acute respiratory distress syndrome (ARDS), burns, pancreatitis, intestinal endotoxin, and major trauma are the most commonly identified triggers for the development of SIRS when there is no known microorganism involved. In 1992, a basic tool was created to identify minimum criteria for early recognition of SIRS **Table 9-10**. Four criteria are included: temperature, heart rate, respiratory rate, and white blood cell (WBC) count. To classify a patient with SIRS, a minimum of two markers need to be met. This basic tool has been used to identify this complex disease for more than a decade and continues to serve as a guideline for research in sepsis.

Sepsis is a mild systemic response to an infection or a suspected infection. The common language used to describe sepsis typically implies a bacterial infection. However, this is not necessarily true because 25% of patients with sepsis have no identifiable source of infection.

Severe sepsis is defined as sepsis with a confirmed or highly suspected diagnosis of infection and organ dysfunction of at least one organ. In severe sepsis, organ dysfunction is caused by hypoperfusion. Any organ may be involved; however, the lungs and the kidneys are typically affected first, with an incidence rate near 20%. At the 2001 International Sepsis Definition Conference, new terminology was created to better define and identify severe sepsis. Experts established the diagnostic criteria in **Table 9-11** incorporating the 1992 minimal diagnostic criteria for severe sepsis.

TABLE 9-10 Systemic Inflammatory Response Syndrome Criteria From the American College of Chest Physicians, 1992

Criteria	Marker

Temperature	> 38°C (100.4°F) < 36°C (96.8°F)
Respiratory rate	> 20 breaths/min $PaCO_2$ < 32 mm Hg
Pulse rate	> 90 beats/min
WBC count	> 12,000/μL < 4,000/μL > 10% immature band forms

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Septic shock incorporates hemodynamic instability with SIRS and a confirmed or highly suspected source of infection that is unresponsive to fluid resuscitation. Hypotension is recognized as a systolic blood pressure (SBP) of less than 90 mm Hg, a drop in SBP by 40 mm Hg from baseline, or an MAP of less than 70 mm Hg. Hypotension associated with septic shock is unresponsive to standard fluid challenges of 20 mL/kg.

Sepsis may progress through septic shock and result in organ failure. When two or more organs fail, MODS develops. Once organs begin to shut down, there is a profound increase in mortality.

Epidemiology

The prevalence of sepsis-related infections is increasing. Martin et al identified a steady 13.7% annual increase in the number of sepsis cases between 1979 and 2000. However, the decade up to 2000 reported a 66.3% increase in the number of sepsis-related illnesses. By the year 2020, the Centers for Disease Control and Prevention estimates there will be 1.1 million new cases of sepsis in the United States, and numerous research articles confirm these predictions. These predictions mean that CCTPs will likely be involved with more cases of sepsis.

Sepsis affects all populations and age groups. Martin et al found sepsis to be most prevalent in nonwhite men between the ages of 57 and 70 years. A number of coexisting medical conditions have been associated with sepsis. These are listed in order of their prevalence: diabetes, hypertension, congestive heart failure, chronic obstructive pulmonary disease (COPD), cirrhosis, human immunodeficiency virus (HIV) infection, cancer, and pregnancy.

The mortality rate for sepsis alone is near 15%. However, the mortality rate increases significantly as the severity of sepsis increases. Severe sepsis, defined as infection with failure of at least one organ, is the leading cause of death in non–coronary intensive care units and the 10th leading cause of death in the United States.

TABLE 9-11 Diagnostic Criteria for Severe Sepsis

Variables	Criteria	Marker
General	Fever	Temperature > 38.3°C or 101°F
	Hypothermia	Temperature < 36°C or 96.8°F
	Pulse rate	> 90 beats/min or > 2 SDs above normal for age
	Tachypnea	Respiratory rate > 20 breaths/min
	Altered mental status	Confused, delirious, or changed from baseline mental status
	Significant edema	> 20 mL/kg of fluid in 24 h
	Hyperglycemia	Glucose > 120 mg/dL or 7.7 mmol/L
Inflammatory	Leukocytosis	WBC count > 12,000/μL
	Leukopenia	WBC count < 4,000/μL
	Normal WBC count with immature forms	> 10% immature forms
	Plasma C-reactive protein level	> 2 SDs above normal value
	Plasma procalcitonin level	> 2 SDs above normal value
Hemodynamic	Arterial hypotension	SBP < 90 mm Hg
		MAP < 70 mm Hg
		SBP decrease of > 40 mm Hg in adults or > 2 SDs below the normal range for age
	Svo_2	> 70%
	Cardiac index	> 3.5 L/min/m²
Organ dysfunction	Arterial hypoxemia	Pao_2/Fio_2 < 300
	Acute oliguria	Urinary output < 0.5 mL/kg/h × 2 h
	Creatinine level increase	> 0.5 mg/dL
	Coagulation abnormalities	INR > 1.5
		Activated partial thromboplastin time > 60 s
	Ileus	Absent bowel sounds
	Thrombocytopenia	Platelet count < 100 × 10^3/μL
	Hyperbilirubinemia	Total bilirubin > 4 mg/dL or 70 mmol/L
Tissue	Hyperlactatemia	> 2 mmol/L
	Delayed capillary refill	Mottling

Abbreviations: INR, international normalized ratio; MAP, mean arterial pressure; SBP, systolic blood pressure; SDs, standard deviations; WBC, white blood cell.

There are an estimated 751,000 cases of severe sepsis annually in the United States, with the mortality rate reported at 28.6%. This mortality rate translates to approximately 575 patient deaths daily as a result of severe sepsis in the United States alone. Statistics also indicate that with population adjustments, there will be an annualized increase in the incidence of 8%.

Despite the development of sophisticated technologies and advancements in medical therapies, sepsis remains a challenge to the medical community. Health care professionals must be aware of the many risk factors associated with severe sepsis to keep mortality rates low. The conditions that place people at greatest risk for developing sepsis are listed in **Table 9-12**. Also, prolonged hospitalization is a risk factor for sepsis for any person. The longer a patient is in the intensive care unit (ICU) (especially) or the hospital, the greater the risk for sepsis. Invasive procedures, comorbidities, and use of immunosuppressants increase this risk to even greater proportions.

TABLE 9-12 Risk Factors for Sepsis

Persons with a compromised immune system	Extremes of age: infants and elderly
	Cancer patients
	Transplant recipients
	HIV/AIDS patients
	Immunosuppressed patients

	Alcoholic patients Pregnancy
Chronic and coexisting diseases	Diabetes COPD Cirrhosis Heart failure Renal failure
Infections and exposures	Meningitis Community-acquired pneumonia Cellulitis Urinary tract infections Organisms with antimicrobial resistance
Critically ill patients	Increased use of invasive catheters and prosthetic devices Postoperative abdominal surgery

Pathophysiology

To understand the pathophysiology of sepsis, it is necessary to review some basic anatomy and physiology of the immune system. The immune system is frequently compared with a well-trained military force because it defends the body against attacks. It is one of the most complex and multifaceted systems in the body. Activation of the immune system elicits a cellmediated response through the production of T cells in the thymus gland and a humoral response through B lymphocytes in the plasma. These immune responses are typically classified into innate and adaptive. The **innate immune system** puts up the first line of defense. It poses a generalized, fast-acting response to **antigen** recognition. The **adaptive immune system** offers a precise response to specific antigens but requires more time to mobilize its defense if the pathogen is unknown. This subdivision is called adaptive because it relies on previous exposure to pathogens and lymphocyte memory cells. Memory cells remember specific antigens they have come in contact with, so when an antigen tries to invade again, the adaptive immune system can elicit a faster response. Each of these subdivisions has its own soldiers performing specific tasks. However, many of these soldiers work in the innate *and* adaptive subdivisions of the immune system. This dual function allows the attacks on pathogens to come from multiple directions while simultaneously enhancing the communication within the immune system.

Pathogens

The microorganisms responsible for activating the immune system are referred to as **pathogens**. Severe sepsis can occur as a result of infection at any body site, including the lungs, abdomen, skin, soft tissue, and urinary tract, and as a result of a primary bloodstream infection, such as in patients with meningococcemia. Bacteria are the pathogens most commonly associated with the development of sepsis, although fungi, viruses, and parasites can also cause sepsis. Each pathogen has identifiers on it called antigens. These antigens elicit an immune response within the body. A pathogen is recognized in the body when its antigen binds to special cells within the immune system. It is the responsibility of this system to classify the pathogen as a “self” antigen or a “nonself” antigen. Self antigens are sometimes referred to as normal flora. The body allows self antigens to reside in areas of the body where they will do little harm. These microorganisms live on the skin, in the nasal passages and respiratory tract, and in the digestive system, including the mouth and intestines. However, if the microorganisms start to colonize, they become

pathogens, and the body steps into action to destroy them.

Infectious agents may produce endotoxins and exotoxins that alter the living cells and ultimately destroy them. This is the primary way bacteria affect the immune system stimulus response and cause death. Endotoxins are proteins that are released by gram-negative bacteria when they die; exotoxins are proteins that are secreted by some bacteria and fungi to aid in the death and digestion of other cells.

When a pathogen is identified as nonself, the immune system is activated and a cascade of events begins. Gram-negative and gram-positive bacteria are the two most common organisms responsible for sepsis. The 2000 data from Martin et al identified that 52.1% of sepsis cases were from gram-positive organisms and 37.6% were from gram-negative bacteria. The remaining 10% are caused by other microorganisms, including anaerobes, viruses, and protozoa, and involve polymicrobial and fungal infections.

Infection

Infection is simply an invasion of a host by a pathogen or multiple pathogens. The body's first lines of defense against pathogens are its natural barriers. Typically, when an infection ensues, it is because a pathogen was able to penetrate the natural barriers of the human body. The skin, mucous membranes, and gastrointestinal tract are examples of natural barriers. They mitigate infection by inhibiting the entry of nonself pathogens into the body and by keeping a careful watch of the self antigens.

Isolated infections are confined to a small area, and the invading organisms are destroyed by cells within that location. The first response to a pathogen invasion requires the innate system to stimulate the inflammatory process. Julius Cohnheim, in the 19th century, observed three characteristic changes in the microcirculation (arterioles, capillaries, and venules) near the site of injury secondary to inflammation.

1. Blood vessels dilated, increasing blood flow to the area.
2. Vascular permeability increased, facilitating an influx of fluid and plasma cells (exudate) into the area.
3. Certain WBCs (**neutrophils**) adhered to the inner walls of vessels and then immigrated through vessel walls to the site of injury. When destroying the pathogen, these phagocytic cells cause the release of lytic enzymes, which are potent enough to damage healthy cells near the area of injury.

Inflammation occurs when chemical mediators, also called **cytokines**, are released from injured cells (the endothelium of the microcirculation) in response to tissue insult. Some of these cytokines include interleukins, **tumor necrosis factor (TNF)**, histamine, prostaglandins, and bradykinins. These chemicals cause vasodilation, smooth muscle contraction, increased tissue permeability, and pain **Table 9-13**. Inflammation is an important process because it helps contain the injury, allowing phagocytes and the complement system to clean up the area. Cytokine communicators also send out distress signals informing the body of injury. This signal alerts phagocytic troops, drawing neutrophils and macrophages to the front lines. When neutrophils arrive on the scene, they roll across the endothelium until they come in contact with selectin. This glycoprotein molecule is released from leukocytes (B and T cells) at the area of insult and assists the neutrophils in adhering to the endothelium. After adhesion, neutrophils begin immigrating through the endothelial wall to the site of injury, where they begin their phagocytic role.

TABLE 9-13 The Body's Response to Inflammatory Chemical Mediators

Inflammatory Chemical Mediator Action	Body Response
Vasodilation	Redness and heat production
Smooth muscle contraction	Difficulty breathing

Increased capillary permeability	Edema and swelling
Stimulation of pain receptors	Pain

B lymphocytes initiate an acute response when memory cells recognize the pathogen. If microorganisms are new to the body, helper T cells activate B cells to form antibodies and begin establishing a memory for the specific antigens. Recognition of antigens allows immunoglobulins (antibodies) to release more cytokines and activate the complement system. It is the responsibility of the complement system to assist the phagocytes by neutralizing the pathogen.

Vasodilation and increased capillary permeability activate the clotting pathways. Tissue factor is released, leading to the production of thrombin, a proinflammatory substance that initiates fibrin clot formation in the microvasculature. Fibrin is the final product of the coagulation cascade and is responsible for preventing the spread of infection by building a fibrin clot barrier wall.

Systemic Infection

Systemic infections activate a widespread inflammatory response throughout the body, completely overwhelming and theoretically confusing the immune system. SIRS, sepsis, severe sepsis, and septic shock are part of a dynamic hypermetabolic, hyperinflammatory response to an insult that grossly affects the endothelium. Three identifiable physiologic processes are involved with the development of microvascular abnormalities associated with severe sepsis:

1. Proinflammation
2. Procoagulation
3. Decreased fibrinolysis

Normal inflammatory modulators are disrupted in SIRS and sepsis. Tumor necrosis factors, interleukins 1 and 6, and other cytokines are key contributors to this disruption. Interleukins and TNF, which manifest themselves as fever, edema, and tissue hypoperfusion, are produced rapidly after insult or injury and continue to proliferate as the inflammatory process persists. These chemical mediators lead to activation of neutrophils while simultaneously inhibiting selectin, the protein that adheres neutrophils to the endothelium. As a result of selectin inhibition, neutrophils are neither able to adhere to nor cross through the endothelial wall to the affected areas. This creates neutrophil rolling, which disrupts the finely tuned immune response and leads to a systemic inflammatory response **Figure 9-9**.

The inflammatory process, in association with the complement cascade, damages the endothelium, causing small clots to form in the capillaries. These microclots occur in the tissues and organs, inhibiting cellular respiration and causing more cell damage. The body uses up its clotting factors and fibrinogen faster than the liver is able to produce them. When the imbalance between coagulation and fibrinolysis occurs, DIC results. As mentioned earlier, this is largely because of the coagulation and fibrinolysis processes that occur simultaneously in sepsis states. Stated more technically, in sepsis there is an imbalance between fibrin clot formation and fibrin clot breakdown or fibrinolysis. Clot formation starts as inflammatory cytokines (TNF-α and interleukin 6) stimulate tissue factor, the first step in the extrinsic pathway of coagulation. Tissue factor turns on the coagulation cascade that leads to the production of thrombin, a proinflammatory substance that promotes fibrin clot formation. Once activated, the coagulation system becomes self-sustaining without the need for tissue factor to stimulate clot formation. Normally, this protective mechanism would help wall off an infected area and promote healing. But in severe sepsis, it results in thrombosis and dangerous deposition of fibrin clots in small blood vessels throughout the body. In severe sepsis, microvascular thrombi impair blood flow to the distal tissues, inducing further tissue hypoperfusion and increasing hypoxia. On the opposite end of the spectrum, while thrombin is developing microclots, antithrombin is working to inactivate thrombin. Antithrombin is a

naturally occurring protein found in plasma that binds to thrombin, inhibiting clot formation. Thrombomodulin, found in the vascular endothelium, also works to bind thrombin. When thrombin is bound to thrombomodulin, protein C is activated. Activated protein C (APC) inactivates specific components of the clotting cascade, factors Va and VIIIa, and neutralizes plasminogen activator inhibitor-1 (PAI-1) **Figure 9-10**. However, during the sepsis process, there is a decrease in the amount of available antithrombin and functional APC. Proinflammatory cytokines, TNF, and interleukins shear thrombomodulin from the endothelial surface, thus preventing the activation of protein C. Studies show that adequate circulating levels of APC can directly inhibit the production of TNF and interleukins and reduce mortality rates from about 30% to almost 25%. Without APC and antithrombin to aid anticoagulation, the procoagulant processes go uninhibited. Therefore, coagulation continues to outweigh anticoagulation.

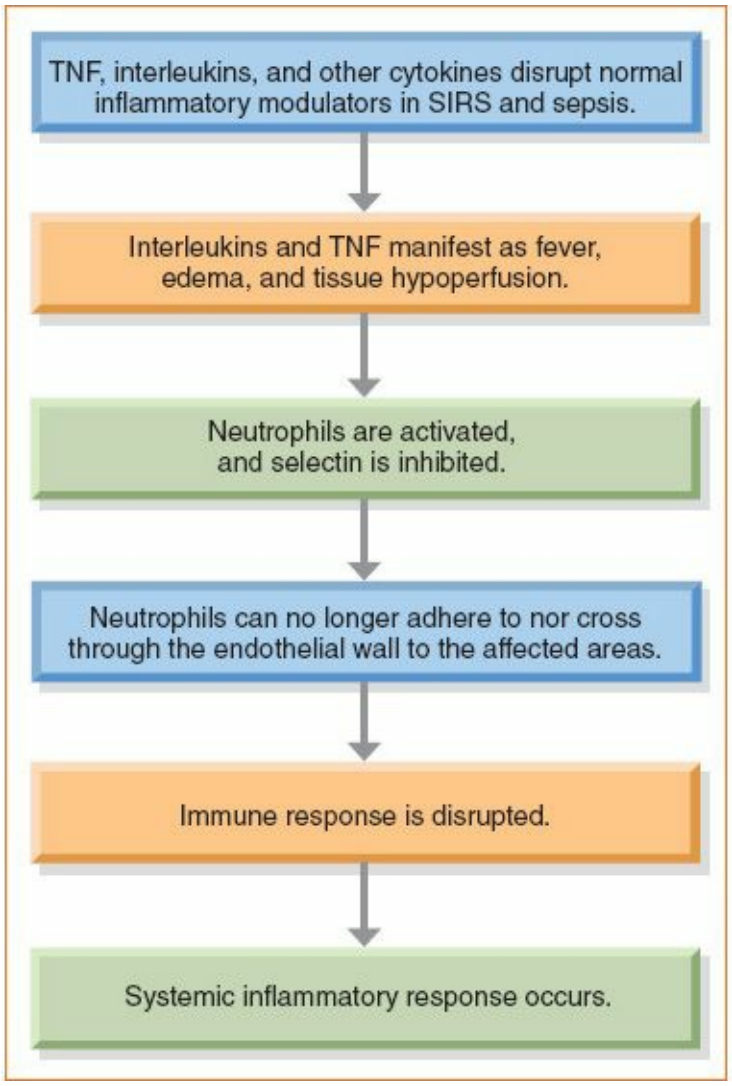


Figure 9-9 Summary of the process leading to a systemic inflammatory response.

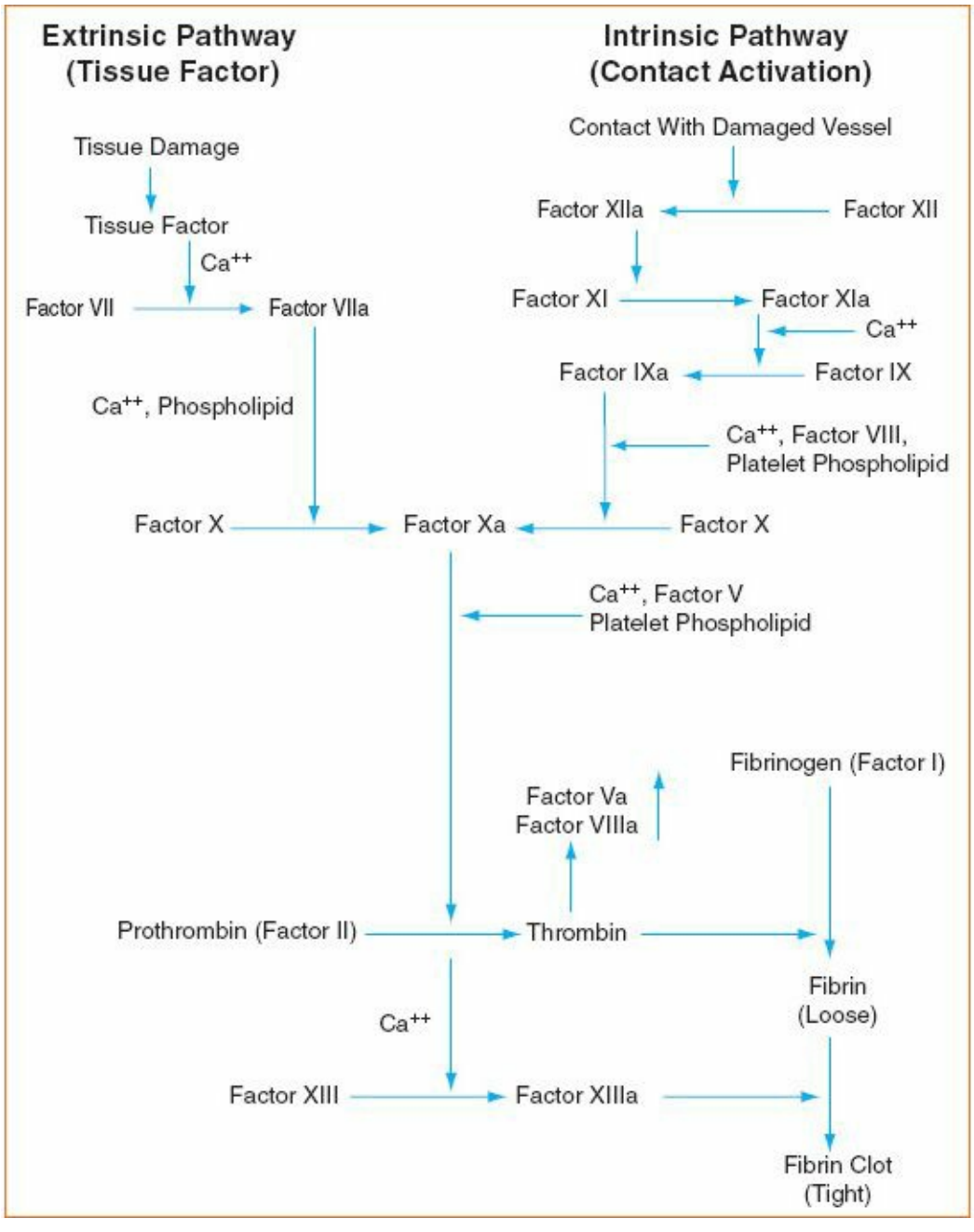


Figure 9-10 Coagulation cascade. Ca^{++} indicates the calcium ion.

In sepsis, there is also decreased fibrinolysis, the process of clot dissolution or lysis. Plasmin is the agent responsible for the breakdown in fibrin clots. Septic states alter the fibrinolytic process in two ways: First, plasmin is inactivated by antiplasmins. Second, PAI-1 is able to stop the conversion of plasminogen to plasmin in the extrinsic and intrinsic pathways. Elevated serum levels of PAI-1 have been correlated with high mortality rates in septic patients.

In summary, microvascular disruption and injury are responsible for hypoperfusion and the shunting of oxygen, resulting in decreased oxygen delivery to the tissues. The profound imbalance between oxygen delivery and consumption causes hypoxia within the cell and tissue. This complex cascade of events is the primary cause of severe sepsis and its progression to organ dysfunction and failure.

Figure 9-11 shows the coagulation imbalance during sepsis.

Assessment

Distributive shock caused by sepsis differs from other shock states in that it has two phases. Phase one is the **hyperdynamic state**, commonly referred to as warm shock. Phase two is the **hypodynamic state**,

commonly referred to as cold shock. The hyper-dynamic (warm) phase is characterized as a high-cardiac-output state that may last for hours or days. This phase may progress to hypodynamic (cold) shock when the patient's condition rapidly deteriorates and there is a sudden drop in cardiac output. The prognosis is much better in hyperdynamic shock than in hypodynamic shock.

The first priority in recognizing sepsis is to maintain a high index of suspicion for the disorder. A thorough patient history correlated with patient presentation can ensure early identification and intervention to break the progression of the sepsis process. Also, infection does not need to be present for sepsis to be present. In fact, in half the documented severe sepsis (SIRS) cases, an infectious cause was not identified. Specimen cultures are obtained from the blood, urine, sputum, and any drainage site to assist in identifying the causative organism. If the pathogen is bacterial, the WBC count is typically elevated and associated with immature neutrophils or bands. Once the pathogen is identified, more direct antimicrobial therapy can be initiated.

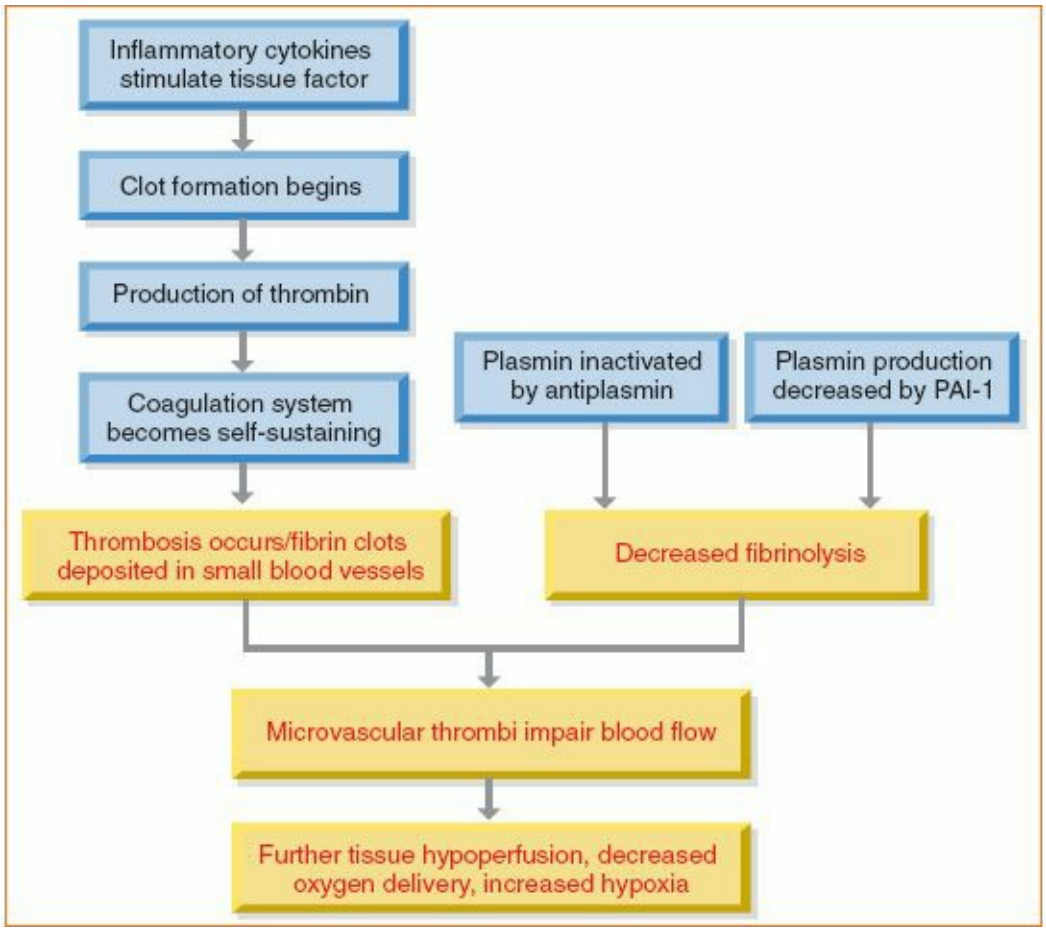


Figure 9-11 The imbalance between coagulation and fibrinolysis in sepsis results in hypoperfusion, decreased oxygen delivery, and increased hypoxia. Abnormal steps and processes are shown in red.

Many health care providers associate infection with fever. Fever is one clinical sign of infection, but it may not be present in geriatric, newborn, and infant populations; patients with chronic renal failure; and patients receiving steroids or other anti-inflammatory drugs. Hypothermia is present in about 10% of SIRS cases and is associated with a poor prognosis.

The cardiovascular and respiratory organs are the hardest hit in severe sepsis and septic shock. Cardiovascular changes result from the inflammatory process of sepsis. The massive vasodilation significantly lowers the SVR. In return, the heart increases CO. Cardiac output is extremely elevated in sepsis states. To produce this high-output state, the heart rate becomes tachycardic, and the stroke volume increases. Initially, normal to low-normal blood pressure is maintained. Skin color and mucous membranes appear normal because of the vasodilation with associated high CO. However, the skin

typically feels warm to the touch as the result of fever. Once hypotension ensues, the patient is in septic shock. Septic shock is refractory to fluid challenges and, therefore, vasoactive agents must be initiated. This is also the progression from hyperdynamic shock into hypodynamic shock. Hypodynamic shock gets its name from the low-cardiac-output state. Patients are hypovolemic in this phase, and their condition rapidly deteriorates as evidenced by low blood pressure, decreased pulse pressure, and low CO. Skin color and temperature change in hypodynamic shock. The patient becomes cold and clammy with pallor or cyanosis. In patients with DIC, petechiae may appear, and blood may ooze from mucous membranes, including the nose and mouth, and from any place where invasive procedures were necessary, including IV sites, indwelling urinary catheters, and chest tubes. Diagnostic patterns in hypodynamic septic shock reveal low levels of hemoglobin, fibrinogen, and platelets and a low hematocrit value. Thrombocytopenia (low platelet count) is an early indicator of progressing DIC and the development of MODS. Prolonged prothrombin time and an elevated D-dimer level are present in more than 90% of severe sepsis cases and, therefore, are a beneficial diagnostic tool. The D-dimer level is monitored throughout the course of sepsis to evaluate the effectiveness of treatment. Levels returning to normal indicate that treatment is working.

The respiratory system also manifests changes. In hyperdynamic septic shock, the respiratory rate increases along with the depth of respiration, leading to respiratory alkalosis. As shock progresses to the hypodynamic phase, ARDS develops, which is a complication of SIRS. Oxygen free radicals are small unpaired electrons that lie in the outer shell of the oxygen molecule and result from the breakdown of the covalent bond between two molecules. When this occurs, each molecule has an unpaired electron within its outer shell that becomes highly reactive. Owing to their reactiveness, the oxygen free radicals damage lung tissue. This damage results from a combined effort of increased oxygen free radicals released in SIRS and from the high percentage of oxygen that must be delivered to enhance oxygen delivery to the cells. Mechanical ventilatory support is needed in sepsis states associated with ARDS. When mechanical ventilatory therapy is initiated, it is important to maintain low tidal volumes at 6 mL/kg, while keeping end-inspiratory plateau pressures lower than 30 cm H_2O. End-inspiratory plateau pressures should also be monitored closely. Studies also show that the positive end-expiratory pressure (PEEP) should be maintained below 14 cm H_2O because higher settings do not change outcomes. Mild **hypercapnia** is allowed in patients with sepsis who have acute lung injury (ALI) or ARDS as long as low tidal volumes and pressure limits are controlled. The FIO_2 will need to be titrated, usually to less than 60%. Therapy goals should be an SpO_2 of greater than 92%. A CCTP may also see higher-than-average end-tidal carbon dioxide readings in patients with severe sepsis.

ALI and ARDS are defined as the following:

- Acute onset of respiratory failure
- Diffuse bilateral infiltrates evident on chest radiography
- Absence of left atrial hypertension (PCWP < 18 mm Hg) or no clinical evidence of left atrial hypertension
- Hypoxemia, defined as PaO_2/SaO_2 of 300 or less for ALI or PaO_2/SaO_2 of 200 or less for ARDS

Hyperlactatemia (> 36 mg/dL) is common in patients with severe sepsis or septic shock. Research has shown that serum lactate levels rise early as a result of anaerobic metabolism, even before hypotension develops. This happens because endotoxins cause the abdominal tissue to shunt blood away from the splanchnic (spleen) tissue while initially not affecting the muscular microvasculature.

Table 9-14 lists sepsis lab abnormalities.

TABLE 9-14 Sepsis Lab Abnormalities

Specimen cultures obtained from the blood, urine, sputum, and any drainage site to identify the causative organism
Elevated WBC count (if the pathogen is bacterial) associated with immature neutrophils or bands
Low hemoglobin level and hematocrit value
Low fibrinogen levels
Thrombocytopenia
Prolonged prothrombin time
Elevated D-dimer level
Hyperlactatemia (> 36 mg/dL)

Signs and Symptoms

Sepsis

- Distributive shock (beginning with hyperdynamic [warm] shock, progressing to hypodynamic [cold] shock)
- Infection confirmed or highly suspicious
- Fever may or may not be present
- Hypothermia may be present in later stages
- Cardiovascular changes
- Significantly lowered SVR
- Tachycardia
- Normal to low-normal blood pressure initially; hypotension once in septic shock
- Normal skin color, normal mucous membrane color, but warm skin initially
- Cold, clammy skin with pallor or cyanosis when sepsis is more progressed
- Petechiae
- Blood oozing from mucous membranes and procedure sites
- Decreased pulse pressure
- Increased respiratory rate
- Increased respiratory depth
- Respiratory alkalosis
- ARDS

Transport Management

Sepsis

- Maintain the following:
  - MAP greater than 70 mm Hg or systolic blood pressure greater than 90 mm Hg;
  - SvO_2 greater than 70%;
  - CVP of 8 to 12 mm Hg;

  - Urine output greater than 0.5 mL/kg/h;
  - SaO_2 greater than 93%; and
  - Cardiac index of 2.5 to 4.0 L/min/m^2.
- Administer oxygen.
- Provide fluid resuscitation (isotonic crystalloids in 20-mL/kg [500–1,000 mL of fluid] increments to stabilize filling pressures).
- Consider colloid therapy.
- Administer vasoactive agents if two 20-mL/kg fluid challenges are inadequate to maintain blood pressure.
- Monitor D-dimer level.
- Provide mechanical ventilatory support if patient has ARDS.
  - Maintain low tidal volumes at 6 mL/kg.
  - Provide high PEEP at ranges of 5 to 14 cm H_2O. The PEEP is increased as the need arises for higher oxygen saturation.
  - Make FIO_2 adjustments for oxygen saturation as needed.
  - Maintain PaO_2 between 55 and 80 mm Hg or SaO_2 between 88% and 95%.
  - Keep end-inspiratory plateau pressures lower than 30 cm H_2O.
  - Monitor $PaCO_2$ closely.
  - The oxygen saturation goal is greater than 92%.
  - Sedation, if needed, must be monitored closely.
- Give antibiotic therapy as ordered.
- Remove potential sources of infection: wound debridement, abscess drainage, and device removal.
- Consider administration of a corticosteroid or recombinant human APC.

Management

Early recognition and early management are vital for a positive patient outcome. Early goal-directed therapy has shown a significant reduction in mortality rates. The primary goal of sepsis treatment is to maintain organ perfusion while enhancing tissue oxygenation. Achieving this requires balancing preload, afterload, and contractility to achieve an optimal level of homeostasis. Target criteria for maintenance levels have been established as follows:

1. MAP greater than 70 mm Hg or systolic blood pressure greater than 90 mm Hg
2. SvO_2 greater than 70%
3. CVP of 8 to 12 mm Hg
4. Urine output greater than 0.5 mL/kg/h
5. SaO_2 greater than 93%
6. Cardiac index of 2.5 to 4.0 L/min/m^2

Figure 9-12 shows an algorithm for the treatment of sepsis.

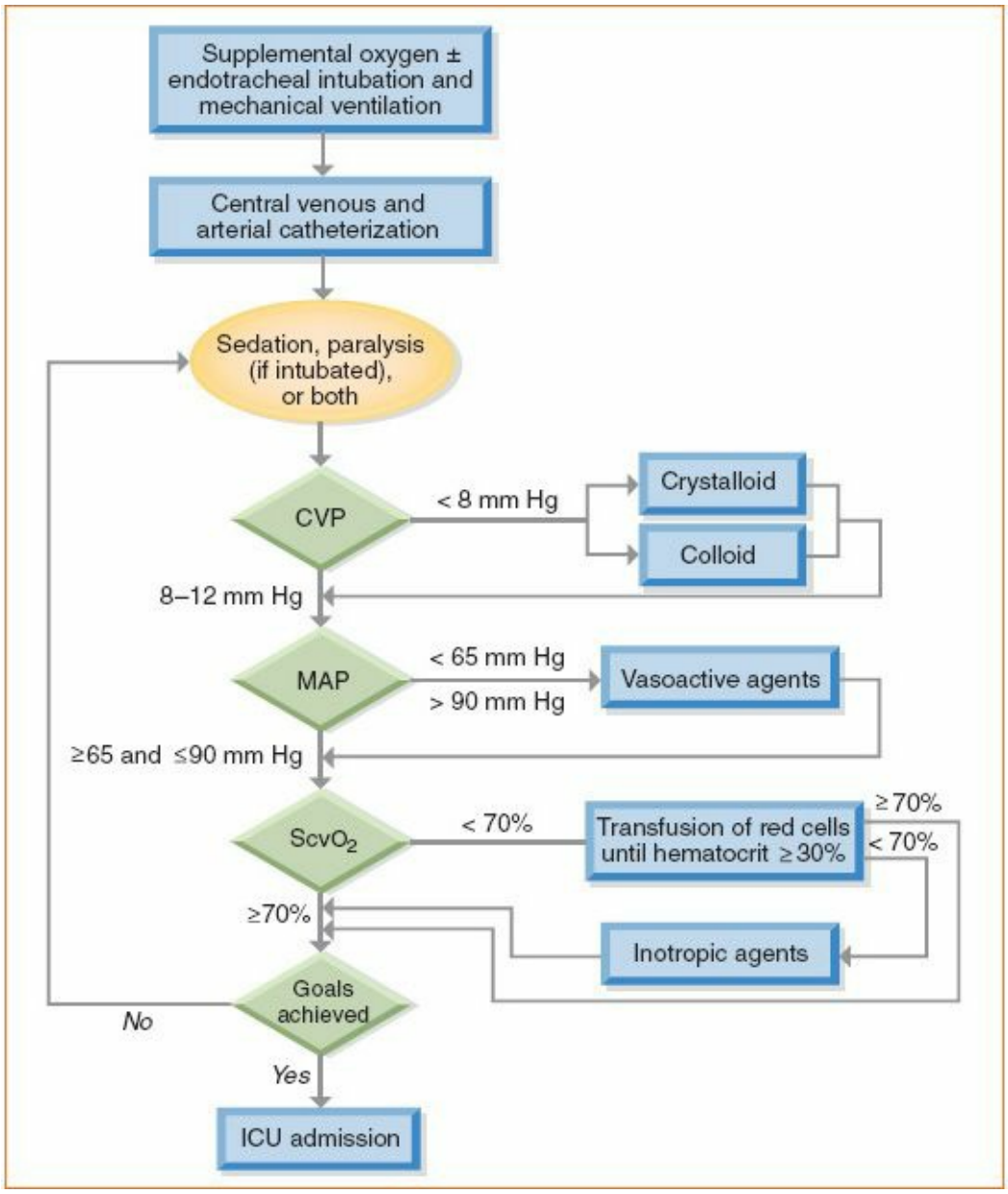


Figure 9-12 Protocol for early goal-directed therapy for sepsis. $ScvO_2$ denotes central venous oxygen saturation. *Source:* Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345(19):1371.

Fluid Resuscitation

The components of CO are preload, afterload, and contractility. Initial resuscitation includes volume expansion to optimize CO. Isotonic crystalloids in 20-mL/kg (500-1,000 mL of fluid) increments are used to stabilize filling pressures. When hemodynamic monitoring is performed, this translates to a CVP of 8 to 12 mm Hg with an MAP of greater than 65 mm Hg. Large volumes of crystalloids may be necessary to maintain blood pressure and CVP. If signs of fluid overload develop, colloid therapy may be initiated along with IV fluid. However, the use of colloids shows no significant improvement in outcome when compared with the use of crystalloids. The appeal for colloid use is the reduction in volume required to maintain hemodynamic stability: 1 L of normal saline (crystalloid) adds 275 mL to the vascular volume, whereas 1 L of 5% albumin (colloid) yields a 500-mL increase. The infusion of packed red blood cells should be considered if the hemoglobin level drops below 7.0 g/dL.

Vasopressor and Inotropic Support

Fluid therapy is not always sufficient to maintain blood pressure. Vasopressors should be started if two 20-mL/kg fluid challenges are inadequate for maintaining blood pressure. **Table 9-15** lists vasoactive agents and their doses, actions, and hemodynamic and adverse effects.

Norepinephrine and dopamine are the vasopressors of choice in hypotensive patients with sepsis. Dopamine may stimulate dopaminergic receptors, or beta 1– or alpha 1–adrenergic receptors, depending on the dose. Sepsis cases require stimulation of alpha 1–and beta 1–adrenergic mechanisms to improve MAP. This stimulation requires IV infusion rates between 5 and 30 µg/kg/min. The beta-adrenergic properties of dopamine will increase contractility but will simultaneously increase heart rate. The increase in heart rate is not a desired effect in sepsis because it increases metabolic demands on the heart. Low-dose dopamine to stimulate renal output has been ineffective in patients with severe sepsis and, therefore, is not used in treatment.

Norepinephrine, a sympathomimetic, acts directly on alpha-adrenergic receptors in the smooth muscle of blood vessels to rapidly increase blood pressure. Norepinephrine also stimulates beta-adrenergic receptors that work to increase cardiac output through increased contractility. Another way norepinephrine has proven itself as a treatment for sepsis is its minimal increase of the heart rate when compared with dopamine. Norepinephrine is typically started at 2 µg/kg/min and titrated to a desired MAP of greater than 65 mm Hg. In some cases, the initial dose may be as high as 10 µg/kg/min until the blood pressure stabilizes. Maintenance doses are typically between 2 and 4 µg/kg/min.

Dobutamine is an inotropic agent that has been very beneficial in treating cardiac depression associated with sepsis. This inotropic agent increases cardiac contractility through its selective beta 1–adrenergic properties. Dobutamine is especially useful when used in conjunction with norepinephrine. The heart rate is usually not significantly affected. An IV infusion typically starts at 2.5 µg/kg/min and may be titrated to as high as 40 µg/kg/min. When titrating dobutamine, it is important to monitor MAP, SvO_2, and the CO or cardiac index to establish adequate treatment therapies.

Source: Reprinted from *Ann. Emerg. Med.*, H. B. Nguyen, et al., Severe Sepsis and Septic Shock: Review of the Literature and Emergency Department Management Guidelines, pp. 28–55, Copyright (2006), with permission from Elsevier. [http://www.sciencedirect.com/science/journal/01960644]

Other vasopressors that may be used in the treatment of severe sepsis include phenylephrine, epinephrine, and vasopressin. The pure alpha-adrenergic properties of phenylephrine increase blood pressure significantly without affecting the heart rate. However, vasoconstriction within the visceral tissue and mesentery may increase lactic acidosis, furthering the hypoxic state in sepsis. Phenylephrine may be a good choice when tachyarrhythmias limit therapy with other vasopressors.

Epinephrine is a nonselective adrenergic activator, meaning it stimulates alpha-1, beta-1, and beta-2 receptors. Epinephrine increases MAP by increasing the cardiac index, stroke volume, SVR, and heart rate. Like phenylephrine, epinephrine must be used cautiously in sepsis because of its vasoconstrictive effects in the abdominal compartment. Administration of this agent is associated with an increase in systemic and regional lactate concentrations. Other undesirable effects include the potential to produce myocardial ischemia and the development of arrhythmias. The use of epinephrine as a single agent is not recommended for patients with septic shock. The use of epinephrine is recommended only for patients who are unresponsive to traditional agents.

Vasopressin may be administered if hypotension is refractory to other therapy. The mechanism of action in vasopressin differs from that of other pressor agents. Vasopressin works as a vasoconstrictor but

has no inotropic or chronotropic effects. Therefore, CO may decrease, resulting in decreased blood to the spleen. For this reason, vasopressin is never used alone or is used when the patient has a cardiac index of less than 2 L/min/m^2.

Septic patients with an elevated blood pressure may require afterload reduction. Nitroglycerin infusions at 5 to 60 μg/min can be used to reduce afterload and preload on the stressed heart.

Infection Control

Antibiotic therapy is initiated with broad-spectrum antibiotics. When specific microorganisms are identified by culture, antibiotic therapy will become more specific to the pathogen. Antibiotic therapy should begin within the first hour of suspecting sepsis to minimize mortality.

Infection control also requires therapy to remove potential sources, such as special interventions for wound debridement, abscess drainage, or device removal. Identifying and removing the potential source of infection is key to treating and lowering mortality in sepsis.

Immune-Specific Therapy

Corticosteroids

Glucocorticoid steroids are used in septic shock when hypotension requires the use of vasopressive agents. In severe sepsis, corticosteroid production drops and relative adrenocortical insufficiency develops. Studies indicate that 56% to 77% of mechanically ventilated patients with septic shock requiring vasopressors have this disorder. Low-dose glucocorticoid or mineralocorticoid therapy is used in selected patients based on serum measurements of endogenous cortisol levels or adrenocorticotropic hormone stimulation tests. Baseline data for cortisol levels or the adrenocorticotropic hormone stimulation test must be obtained before treatment begins. The goal of replacement therapy is to achieve the same physiologic level of glucocorticoids in the blood that would be present under normal conditions. Research studies on the effectiveness of corticosteroids continue. To date, treatment with hydrocortisone, 50 mg IV every 6 hours, and fludrocortisone, 50 μg orally once daily for 7 days, has been effective in treating septic shock.

Recombinant Human Activated Protein C

Drotrecogin alfa (activated) is a relatively new pharmacologic agent used in the treatment of severe sepsis. Tertiary and research centers are more likely to administer this medication than are primary facilities. For this reason, interfacility transport personnel are less likely to see this agent than others. However, this medicine is worth mentioning because research is showing that drotrecogin alfa (activated) is very beneficial for reducing mortality in patients with severe sepsis and corresponding organ failure. The pharmacokinetics of drotrecogin alfa (activated) include anti-inflammatory, anticoagulant, and profibrinolytic properties. There have been multiple studies on early use of recombinant human APC. The Surviving Sepsis Campaign Guidelines of 2008 state that it may have a probable reduction in mortality for patients with the following:

- Multiple organ failure
- Clinical assessment of high risk of death (APACHE [Acute Physiology and Chronic Health Evaluation] score of > 25)

The APACHE score is an in-hospital disease scoring system for adults in the ICU that is based on 12 physiologic measurements during the first 24 hours after ICU admission and other health information.

Blood Administration

Blood administration can be a part of managing shock, as discussed in the previous section. The decision

to give a transfusion to a patient during a critical care transport is more complex than the administration of blood in the hospital setting. The urgency of the transfusion, the "out-of-hospital" time, the availability of the blood products, **type-and-crossmatch** information, and the appropriate transport and care of the blood product must be considered. The risk-benefit analysis of the "right treatment for the right patient at the right time" must be determined.

Blood administration may be required to restore circulating blood volume, improve oxygen-carrying needs, or correct specific coagulation components. It can be in the form of whole blood, red blood cells, white blood cells, platelets, cryoprecipitate, or other blood products. It is uncommon to transfuse whole blood currently owing to the expense, uncommon availability, and the possibility of volume overload during whole blood administration. The need to consider blood administration includes hemorrhagic blood loss as a result of trauma, internal hemorrhage, perioperative and postoperative complications, specific disease entities such as leukemia or other cancers, anemia due to illness, or coagulation disorders.

Many CCT programs carry type O blood for field pickups or will take matched blood with the patient from outlying hospitals; however, the availability of blood products from rural health care settings may be vital in the decision about blood administration. Rural hospitals may have a limited supply of banked blood products, especially of certain blood types and specific blood products required by a critically ill patient. Such limitations alter the desired treatment of the patient.

Time becomes a factor in the transport decisions surrounding a critically ill or injured patient. Awaiting the availability of certain blood products may lengthen the time to definitive care at a tertiary care center; it may be more important to transport the patient urgently than to perform the transfusion. The decision must consider the out-of-hospital time for the patient. For example, if it would take 15 minutes to obtain the needed blood products, but the total transport time would be 20 minutes, it would likely be prudent to transport the patient and forgo the transfusion until the patient is at the receiving facility. If the patient can tolerate the transport without the en-route transfusion, the decision to defer blood administration until arrival at the receiving hospital may be in the best interest of the patient.

Blood administration can result in undesirable complications such as transfusion reactions. Complications of this nature are best handled in the controlled environment of a hospital setting and may be difficult to address adequately during transport. Management of transfusion reactions is covered later in this section.

The transfusion of blood or blood products is a form of human tissue transplantation and, therefore, some states regulate who may initiate blood transfusions. Many state or institution blood bank regulations require two licensed personnel to check blood for proper match of the blood unit to the patient. Each unit is treated as a new transfusion, not the continuation of the previous unit.

Care of the blood products must be considered if the transfusion is not initiated before transport. The specific container and ice provided by the sending facility's blood bank must be used to ensure that the quality of the blood product is maintained. Care must be taken to ensure the blood products stay cold throughout transport. Bags of ice are placed in an insulated ice box with ice beneath and on top of the blood products when transporting whole blood, packed red blood cells, or platelets. Care must be taken to prevent the ice from melting. This includes placing the ice box in a cool area and away from direct sunlight during transport. Fresh-frozen plasma and cryoprecipitate are thawed by the blood bank under specific guidelines. After thawing, fresh-frozen plasma must be transfused within a 24-hour period and cryoprecipitate must be transfused within a 6-hour period. Blood products are best stored in a refrigerator specially designed to store blood, with strict temperature regulations and consistently monitored temperature recording systems with required alarm systems in case the refrigerator fails to maintain the temperature settings. The extensive requirements for proper storage of banked blood products allow for little tolerance of deviation when blood administration occurs outside the hospital setting. Consequently,

the inability to adhere to the stringent care, storage, and temperature monitoring of blood products during transport results in refusal and waste of transferred blood products by the receiving facility's blood bank. If blood products may be needed during transport, but it is not certain whether they will be administered, the decision to transport the blood should be made with caution. In this circumstance, the receiving facility's blood bank will most likely destroy blood products that have been removed from a blood bank and transferred to another blood bank for acceptance. Policies in many institutions forbid the acceptance of transferred blood products from outside blood banks. The possible wasting of a scarce resource, such as blood products, may be important in the decision to transport blood products that may not be absolutely necessary for a patient during transport.

The final consideration is that blood administration requires a skilled health care provider with knowledge and experience in blood product administrations. Therefore, the expertise of the CCTP, medical direction, and prevailing medical protocol may further influence the decision to transfuse blood products during transport.

Once the decision to administer blood products has been made, the steps to proper blood administration must be followed. These include appropriate typing and crossmatching through laboratory analysis. An exception to the required crossmatching of the patient before transfusion may occur during emergency resuscitation for hemorrhagic shock when type O, Rh negative, known as the universal donor type, may be administered owing to the urgency of blood replacement surpassing the time necessary for typing and crossmatching the patient before transfusion. The appropriate IV access, blood tubing administration sets, priming of the tubing with normal saline, validation of the blood product's match with the specific patient before administration, and continued supervision by a skilled health care provider are required **Figure 9-13**.

ABO System

Blood transfusions became possible in the early 1900s after discovery of the A, B, O, and AB blood types. The first blood bank was founded in the United States in 1937. The use of whole blood and plasma was prevalent during World War II. The introduction of plastic blood storage containers and apheresis instruments allowed for the creation of blood component therapy options. The use of blood component therapy gained acceptance and surpassed the use of whole blood by the 1970s and remains the most common transfusion option today.

Blood contains a variety of antigens, with more than 400 red blood cell antigens identified. These antigens influence the compatibility of blood between individuals. Three types of blood antigens are considered, including the ABO blood group, Rh factor, and the human leukocyte antigen blood group. The crossmatch laboratory testing considers these antigen group characteristics. Identification and compatibility of ABO grouping is the most important step in the type and crossmatch process. Incompatibility at this step may cause the most severe transfusion reaction, known as acute **hemolysis**.

The ABO method of blood typing identifies two antigens on a red blood cell, known as A and B. If a person has only an A antigen identified, that person is type A. A person who has only a B antigen identified is type B. If both A and B antigens are identified, the person is type AB. When neither an A nor B antigen is identified, the blood type is O. The most common blood types in the United States are A and O, with 85% of the population falling within these two blood types. Type O is the most common blood type. Type B is found in 10% of the US population, with type AB noted in only 5% of the population.

Figure 9-13 Proper procedures for blood administration include appropriate IV access, appropriate blood tubing administration sets, priming of the tubing with normal saline, validation of the blood product's match with the specific patient before administration, and continued supervision by a skilled health care provider.

A blood transfusion reaction may occur when a patient possesses antigens to a blood type and receives that blood type. **ABO-incompatible transfusion reactions** contribute to approximately 25 deaths per year as a result of the misidentification of crossmatch samples or recipients. A patient with A antigens in his or her blood also has anti-B antibodies in his or her plasma. A patient with B antigens in his or her blood also has anti-A antibodies in his or her plasma. Transfusion reactions may occur if patients receive a blood type for which they have antibodies.

People who have type AB blood do not have antibodies and are known as **universal recipients**. They can receive blood types A, B, AB, and O without the threat of an ABO reaction. People who have type O blood are considered **universal donors** because their blood can be transfused into patients with types A, B, and AB blood. However, people with type O blood have antibodies to blood types A and B and can, therefore, only receive type O blood.

Patients should receive transfusions of their own blood type to minimize possible transfusion reactions. In an emergency situation in which the time necessary for crossmatching of blood cannot be taken, type O, Rh-negative blood, or plasma can be transfused until crossmatched blood becomes available.

Rh factors are antigens found on the cell membrane of red blood cells. These antigens are present in approximately 85% of the US population and are considered Rh positive. People lacking the Rh antigen are termed Rh negative. There are no natural Rh antibodies. An Rh-negative person may develop an Rh antibody if exposed to Rh-positive blood. This first exposure to Rh-positive blood allows a person to become sensitized. A second Rh-positive exposure could result in a fatal hemolytic reaction. This type of

reaction may occur during a transfusion or pregnancy.

Human leukocyte antigen (HLA) is present on the cell membrane surface of circulating platelets, white blood cells, and most tissue cells. Patients receiving platelets from multiple donors may experience febrile transfusion reactions as a result of HLA. This type of reaction causes an antigen-antibody reaction that destroys the platelets, reducing the benefit of the transfusion. HLA-matched platelet transfusions dramatically diminish this antigen-antibody reaction and are optimal for patients receiving multiple transfusions.

Blood Products

Blood component therapy has become common in the United States, with more than 22 million transfusions each year. A great number of these transfusions occur in surgical and obstetric patients. These transfusions are considered beneficial for increased tissue oxygenation, decreased blood loss, and improved clinical outcomes. The risks and costs of transfusions must always be considered. Transfusion reactions and the transmission of multiple infectious diseases must be factored into any decision to transfuse blood products. The seven blood components are discussed next.

Blood Components and Derivatives

Whole Blood

Whole blood contains all blood components, including red blood cells, white blood cells, platelets, plasma, and electrolytes. The usual volume is approximately 450 to 500 mL/U. Transfusion involving the use of whole blood is uncommon and indicated only in acute massive blood loss for blood volume expansion and increased oxygen-carrying capacity. Whole-blood transfusions should only be considered when patients have lost at least 25% of their total blood volume. Whenever possible, blood loss should be treated with options other than whole blood and should include blood components, crystalloids, or colloid solutions. Administration of whole blood may cause fluid volume overload in patients with cardiac compromise. Also, whole blood begins to degrade after 24 hours of storage. This cell breakdown causes elevated potassium levels, the formation of microaggregates, damaged platelets and granulocytes, and decreased clotting factors. Whole-blood transfusions must be ABO matched with the recipient as a result of the presence of antibodies. Hemoglobin levels should rise 1 g/dL per infusion of each unit of whole blood. Whole blood is stored at a temperature between 1°C and 6°C (34°F and 43°F). The expiration depends on the anticoagulant preservative. Administration should be delivered as rapidly as possible while maintaining hemodynamic stability. If necessary, multiple large-bore (14- or 16-gauge) catheters can be used to administer multiple units of blood simultaneously. The use of pressure bags or rapid infusion devices may be applied to enhance the flow of the transfusion under emergent resuscitation conditions. Whole blood may be administered through an IV catheter as small as 20 gauge, with normal saline solution, and within a 4-hour period. Close monitoring of the patient during infusion for possible fluid volume overload and transfusion reactions is essential.

Special Populations

Pediatric patients have proportionately higher blood and plasma volumes compared to adults. Transfusion of packed red blood cells is calculated by weight. Children younger than 4 months are initially given 10 mL/kg of PRBCs, whereas children 4 months of age or older can be transfused with 10 to 20 mL/kg depending on lab values and clinical condition. Increased attention to volume overload during blood product administration is particularly important in the pediatric population.

Packed Red Blood Cells

Packed red blood cells (PRBCs) retain all of the characteristics of whole blood with the exception of the extraction of approximately 250 mL of platelet-rich plasma from each unit of whole blood. This constitutes the removal of nearly 90% of the fluid around the RBCs. An anticoagulant preservative is then added. Each unit of PRBCs retains the same concentration of RBCs as a unit of whole blood. One unit of PRBCs is approximately 250 mL and is indicated in the restoration and maintenance of the oxygen-carrying quality of the blood. Anemia and blood loss are possible indications for PRBC administration. A patient's hemoglobin level is expected to raise 1 g/dL per unit of administered PRBCs. PRBCs have nearly 70% of the leukocytes removed from each unit, thus reducing the risk for febrile, nonhemolytic reactions. Proper ABO compatibility and Rh factor matching are required prior to administration.

Platelets

Platelets are blood components developed in the bone marrow and consist of cytoplasmic fragments that contain enzymes necessary for normal clotting response. In the United States, more than 7 million units of platelets are transfused annually. Indications for platelet transfusions include patients who are bleeding because of thrombocytopenia or, in rare cases, the presence of abnormally functioning platelets. Patients experiencing low platelet counts below 5,000 to 10,000/μL may benefit from platelet transfusions on a prophylactic basis. Platelet therapy may also be considered prior to surgery or extensive invasive procedures in those patients with platelet levels below 50,000/μL. Situations that involve chemotherapy, disseminated intravascular coagulation, and massive transfusion may require platelet therapy considerations. ABO identical platelets are ideal whenever available, with Rh-negative platelets matched to Rh-negative patients as frequently as possible for transfusion. If a patient must receive multiple transfusions of platelets, single-donor platelets should be administered. Each bag of platelet concentrate contains 5.5 × 1010 of platelets in 50 to 70 mL of plasma. A single unit raises the platelet count by 5,000/μL. Calculation for dosing of platelet therapy is 6 to 10 U and 1 U/10 kg of body weight.

Administration of platelets requires a filtered component IV drip set with normal saline solution. Premedication of the patient prior to transfusion may include antihistamines or acetaminophen to reduce or prevent chills, fever, and allergic reactions.

Fresh-Frozen Plasma

Fresh-frozen plasma (FFP) is uncoagulated plasma separated from the RBCs. This plasma is primarily composed of water, proteins, salts, and metabolites, and is rich in clotting factors. Annually, approximately 2 million units of FFP are transfused in the United States. Indications for the administration of FFP may include blood loss, coagulation deficiencies, warfarin reversal, and thrombotic thrombocytopenic purpura. Transfusions must be ABO compatible; Rh matching is not required. Blood administration sets and normal saline solution are required for rapid infusion. The citric acid preservative in FFP may bind with calcium during transfusion and can lead to hypocalcemia. Close patient monitoring for hypocalcemia is required during transfusion. FFP must be infused within 24 hours of being thawed.

Cryoprecipitate

Cryoprecipitate is a frozen blood product created from the plasma of a donor. It contains factor VIII, fibrinogen, von Willebrand factor, and factor XIII; it follows that indications for administering cryoprecipitate include hemophilia A, fibrinogen deficiency, von Willebrand's disease, and factor XIII deficiency. Cryoprecipitate is useful in treating patients with clotting disorders, because it replaces fibrinogen. It is typically given to patients with fibrinogen levels of less than 50 to 60 mg/dL. Each bag contains 80 to 100 U of factor VIII. As many as 10 U of cryoprecipitate may be needed to restore the fibrinogen level to 100 mg/dL. Administration of cryoprecipitate does not require compatibility testing,

but ABO compatibility is preferred. An Rh match is not necessary. A blood administration set can be used for infusion, and cryoprecipitate should be administered as rapidly as tolerated. Cryoprecipitate must be administered within 6 hours of thawing. Whole blood donor requirements and the preparation for cryoprecipitate are the same as those for platelets and FFP.

Albumin

Albumin is prepared by the fractionation of pooled plasma. Albumin is available as a 5% or 25% solution and is used for volume replacement in conditions such as burns, trauma, surgery, or infections. The addition of albumin to enhance diuresis in hypoproteinemia is of value. The 25% solution will produce five times its volume from extravascular water into the vascular space. It is important that patients receiving 25% albumin have adequate extravascular water and the compensatory abilities to handle this blood volume expansion.

ABO and Rh compatibility are not a consideration with albumin products. The manufacturer-provided administration tubing should be used for albumin administration, and the infusion rate should be set in accordance with the patient's condition and response. Patients with cardiac and pulmonary disease must be monitored closely during albumin administration because the possibility of heart failure from volume overload.

Plasma Protein Fractions

Plasma protein fraction contains 83% albumin and 17% globulins. Plasma protein fraction is available in a 5% preparation and is indicated for volume expansion in patients with hypovolemia and hypoproteinemia. Clinical settings for the administration of plasma protein fraction may include shock and burn patients. Plasma protein fraction is available in 250-mL containers and can be stored for 5 years at 2°C to 10°C. No transmission of human immunodeficiency virus or hepatitis has been reported with this product.

Synthetic Blood Substitutes

The quest for synthetic substitutes for blood transfusions has been ongoing for the past decade. One promising product known as PolyHeme remains in trial studies with the Food and Drug Administration. PolyHeme is a solution of human hemoglobin extracted from modified RBCs. The product is derived from expired human blood from which the hemoglobin is extracted and purified. The need for ABO antigens, crossmatching, or patient blood typing is not necessary because PolyHeme does not contain intact RBCs. This product can be stored at room temperature for 1 year. This shelf life, when compared with traditional blood products, allows greater versatility in storage locations, making it rapidly available for emergency situations and combat casualties. The processing of this product diminishes the likelihood of viral infection transmission compared with traditional banked blood products and remains an additional benefit to the potential use of this product. PolyHeme continues to be trialed with investigators noting promising results for use during emergency situations when banked blood products are not readily available.

Procedure

The procedure for administering blood is discussed in this section.

Indications

Indications for administering blood include the following:

- Significant hypovolemia as the result of acute blood loss
- Symptomatic anemia

- Decreasing hemoglobin level
- Decreasing hematocrit value
- To increase oxygen-carrying ability
- Decreased clotting factors
- Presurgical care in select cases

Equipment

The following equipment is needed for performing blood administration:

Physician's orders

- Blood product, typed and crossmatched (in some cases may be cryoprecipitate, platelets, or plasma)
- Dedicated venous access line (18-gauge or larger needle)
- Filtered administration set
- Normal saline solution
- Thermometer

Complications

Complications of administering blood include the following:

- Anaphylaxis
- Hemolytic reaction
- DIC
- Transfusion reaction
- Infection

Signs of complications include the following:

- Body temperature of 2°F (1°C) or more above the baseline temperature
- Hives, itching, or skin symptoms
- Swelling, soreness, or hematoma at the venous site
- Flank pain
- Tachycardia
- Respiratory distress (wheezing and dyspnea)
- Hypotension
- Bleeding from widely varied sites or previously clotted wounds
- Blood in the urine
- Anaphylaxis
- Nausea and vomiting

Steps

Skill Drill 9-1 shows the steps for administering blood products. These steps are also described here: Prepare the patient by doing the following four steps.

1. Confirm the order or protocol **Step 1**.

2. Check the patient for the following: right patient, right blood product, and right type. Have a second provider confirm steps 1 and 2 with you **Step 2**.
3. Assess baseline vital signs and temperature **Step 3**.
4. Ensure suitable venous access (usually requires 18-gauge or larger) **Step 4**. At this point, patient preparation is complete and the transfusion procedure begins.
5. Check the blood for the following: right patient, right blood product, right type, and expiration date.
6. Assess the patient for the possibility of a transfusion reaction, and consider the prophylactic administration of ibuprofen or acetaminophen and diphenhydramine.
7. Maintain the temperature of the blood product.
8. Flush the tubing with normal saline **Step 5**.
9. Cover the administration filter with the blood.
10. Connect the blood to the tubing **Step 6**.
11. Piggyback into the IV line of normal saline **Step 7**.
12. Start the transfusion slowly **Step 8**.
13. Monitor every 5 minutes for adverse reactions.

It is important to follow these transfusion precautions:

- Do not mix blood with 5% dextrose in water (causes hemolysis).
- Do not mix with lactated Ringer's (causes clotting).
- Do not mix with medications (may react).
- Have a second venous access available.

Transfusion Reactions

Transfusion reactions are of great concern and play a significant role in the decision to administer blood products. These reactions can be as mild as chills or as catastrophic as death. Reactions can include both endogenous reactions caused by antigen-antibody reaction in the recipient and exogenous reactions caused by external factors related to the transfused blood.

Allergic Reaction

Allergic reactions are caused by allergens in the donated blood with symptoms of anaphylaxis, including chills, facial and laryngeal edema, pruritus, urticaria, and wheezing, as well as fever, nausea, and vomiting. Management includes the use of antihistamines and careful patient monitoring and assessment. Additionally, the indications for epinephrine and corticosteroid administration must be considered.

Isolated reactions such as urticaria or hives may occur during transfusion without additional signs or symptoms of anaphylaxis and without further sequelae. The transfusion does not need to be discontinued and treatment with an antihistamine is usually adequate. When a patient has a known history of this type of allergic reaction, premedication with an antihistamine should be performed prior to the transfusion.

Bacterial Contamination

Bacterial contamination of blood components usually occurs during one of the following phases of blood administration: phlebotomy, during the component preparation or processing, or during thawing of the blood components.

The frequency of bacterial contamination is low. When it does occur, it may have a rapid onset and lead to death. Of the deaths caused by bacterial contamination of blood components reported to the Centers for Disease Control and Prevention, most are contaminated by *Yersinia enterocolitica*. Since

1987, there have been 20 such cases reported, 12 of which resulted in deaths.

Bacterial contamination usually results from endotoxins produced by organisms capable of surviving the cold, such as *Pseudomonas, Staphylococcus*, and *Y enterocolitica*. Presenting symptoms may occur rapidly or within 30 minutes after transfusion and include chills, fever, vomiting, abdominal cramping, bloody diarrhea, hemoglobinuria, shock, renal failure, and DIC. It is essential to rapidly recognize sepsis caused by blood products contaminated by bacteria. The transfusion must be stopped immediately upon the onset of symptoms. The IV line should be kept open, and the protocol for transfusion reactions should be implemented. The blood component unit, any associated fluids, and the transfusion equipment should be sent to the blood bank immediately for thorough inspection, Gram staining, and culture. Blood cultures should be drawn from the patient as soon as possible for the detection of aerobic or anaerobic organisms. The administration of broad-spectrum antibiotics, corticosteroids, and vasopressors; treatment for shock; fluid support; respiratory ventilation; and maintenance of renal function should be considered in the management of these patients.

Febrile Transfusion Reactions

Febrile transfusion reactions are caused by bacterial lipopolysaccharides or antileukocyte recipient antibodies directed against donor WBCs. Temperatures as high as 104°F (40°C), chills, headache, facial flushing, palpitations, cough, chest tightness, increased pulse rate, and flank pain may be present. Treatment of these patients includes administration of antipyretics and antihistamines. Modifications must be considered if future blood transfusions are anticipated in these patients, including the administration of frozen RBCs and the use of leukocyte filters as modifiers.

Hemolytic Transfusion Reactions

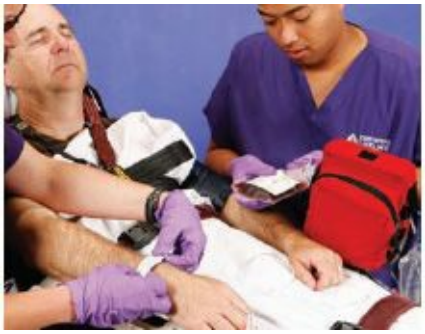
Hemolytic transfusion reactions are caused by ABO or Rh incompatibility, intradonor incompatibility, improper crossmatching, or improper blood storage. These reactions may occur during transfusion or within 3 to 7 days following transfusion.

Immediate hemolytic transfusion reactions usually occur soon after the transfusion of incompatible RBCs. The RBCs are quickly destroyed, resulting in the release of hemoglobin and RBC remnants into the bloodstream with the onset of hemoglobinuria and abnormal bleeding from open sites accompanied by hypotension. The reaction time varies between 1 and 2 hours, with the onset of symptoms within minutes after the blood transfusion begins. Symptoms include chest pain, facial flushing, shortness of breath, chills, fever, hypotension, flank pain, hemoglobinuria, oliguria, bloody oozing at the infusion site, burning along the vein receiving the blood, shock, signs of renal failure, and DIC. A breakdown of these symptoms notes that approximately 35% of the patients with immediate hemolytic transfusion reactions experience fever with or without chills and oliguria with complete recovery, 13% develop anuria, 8% experience coagulopathy, and 10% die after sustained hypotension. Treatment of immediate hemolytic transfusion reactions is directed toward prevention and supportive management. The use of diuretics to augment renal diuresis in an effort to prevent renal failure is recommended. Close monitoring of the patient for the risk factors related to DIC and hypotension is required. Hypotension is managed with fluid resuscitation and vasoactive agents such as dopamine may be required. The use of blood component therapy, including FFP, cryoprecipitate, and platelets, should be considered in patients with bleeding complications or significant coagulation abnormalities. Human error is the cause of most cases of immediate hemolytic transfusion reactions; therefore, these reactions are considered preventable. The extensive policies and procedures necessary to ensure proper patient identification, sample collection and labeling, unit identification, patient testing, handling, and correct transfusion must be strictly enforced.

Skill Drill 9-1

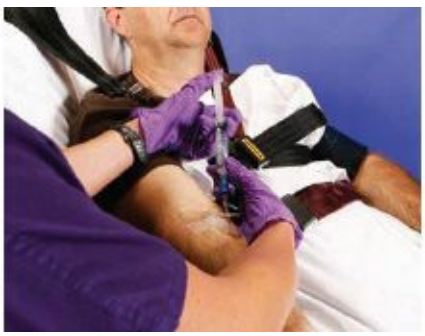
Administering Blood Products

1 Confirm the order or protocol.



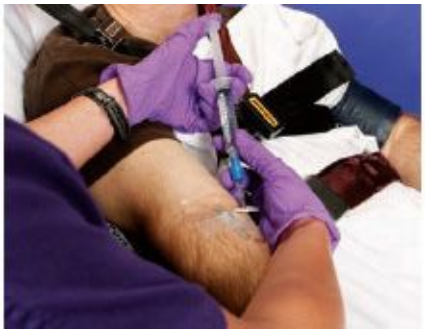
2 Check the patient for the following: right patient, right blood product, and right type. Have a second provider confirm steps 1 and 2 with you.

3 Assess baseline vital signs and temperature.



4 Ensure suitable venous access. Check the blood for the following: right patient, right blood product, right type, and expiration date. Assess the patient for the possibility of a transfusion reaction, and consider prophylactic administration of medications. Maintain the temperature of the

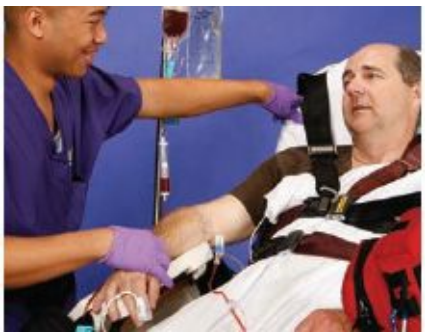
blood product.



5 Flush the tubing with normal saline. Cover the administration filter with blood.

6 Connect the blood to the tubing.

7 Piggyback into the IV line of normal saline.



8 Start the transfusion slowly. Monitor for adverse reactions.

Delayed hemolytic transfusion reactions most often occur in patients who have been sensitized through a previous transfusion, pregnancy, or transplant and who also possess an antibody undetectable by standard pretransfusion testing. In cases of delayed hemolytic transfusion reactions caused by secondary

response, the onset of symptoms does not occur until 3 to 7 days after transfusion. The production of antibodies sufficient to generate the signs and symptoms of delayed hemolytic transfusion reactions occurs during this time period. Generally, they are mild. Severe cases and death are rare. Clinical signs and symptoms usually consist of mild fever, chills, and moderate jaundice with treatment focused on the prevention and the treatment of severe complications should they arise. Support of renal function includes IV fluids to maintain adequate fluid status.

Plasma Protein Incompatibility

Plasma protein incompatibility results from immunoglobulin A incompatibility. Clinical presentations include abdominal pain, diarrhea, shortness of breath, chills, fever, flushing, and hypotension. Management of this form of incompatibility includes oxygen administration, fluids, epinephrine, and corticosteroids as indicated.

Other Causes

Additional exogenous transfusion reactions include bleeding tendencies, circulatory overload, hypocalcemia, hypothermia, and potassium intoxication. Bleeding tendencies may be caused by low platelet counts in banked blood causing thrombocytopenia. Clinical presentations include abnormal bleeding and oozing from breaks in the skin or gums, abnormal bruising, and petechiae. Management involves the consideration of transfusion of platelets, FFP, or cryoprecipitate as needed.

Management Considerations

Circulatory overload is a potential complication during blood administration. As mentioned, the decision to administer whole blood has the possible disadvantage of an increased total volume of blood product that may be poorly tolerated by certain patients. Caution with the administration of whole blood should be noted in those patients with cardiac and pulmonary disease who possess the potential for experiencing congestive heart failure. The use of PRBCs in lieu of whole blood administration is recommended in this patient group. The use of diuretics should be considered if circulatory overload is suspected.

Hypocalcemia results from citrate toxicity during blood transfusions when blood products containing citrate are infused too quickly and bind calcium. The depletion of circulating calcium results in calcium deficiency. This calcium deficit may lead to arrhythmias, hypotension, muscle cramping, nausea, vomiting, seizure activity, and/or a tingling sensation in the fingers. Should the onset of such symptoms occur, slowing or stopping the transfusion may be indicated if ordered. These reactions may be more severe in patients with hypothermia or in those with elevated potassium levels. Management considerations include the administration of calcium gluconate IV infused slowly.

High potassium levels in stored plasma caused by the hemolysis of RBCs may result in potassium intoxication during blood administration. Clinical manifestations may include diarrhea, intestinal colic, flaccidity, muscle twitching, oliguria, signs of renal failure, bradycardia, ECG changes with visible tall peaked T waves, and/or cardiac arrest. Management considerations include a diagnostic 12-lead electrocardiographic (ECG) tracing, sodium polystyrene (Kayexalate) administration, administration of an albuterol nebulizer, administration of furosemide, and the infusion of 50% glucose, insulin, sodium bicarbonate, or calcium in an effort to reduce serum potassium by shifting potassium into cells, as well as reducing total body potassium and making the heart less reactive to high potassium.

The induction of hypothermia is possible during the rapid infusion of large amounts of cold blood products. Hypothermia symptoms include chills, shivering, hypotension, arrhythmias, bradycardia, and/or cardiac arrest if body temperatures fall below 30°C (86°F). Treatment considerations may include stopping the transfusion, warming the patient, obtaining a diagnostic 12-lead ECG, and warming the blood if the transfusion is resumed.

The risk-benefit analysis of blood administration must be met with caution. Blood transfusion can be

a life-saving intervention; however, the many complications and adverse reactions that accompany the decision to deliver blood products to a patient require highly skilled health care providers and vigilance in the health care setting.

Flight Considerations

In the transport environment, a patient in shock or with sepsis or MODS will most likely be very labile and require diligence in monitoring and adjustments to therapy to maintain oxygenation and hemodynamic status. It is important to give close attention and ensure that all infusions are dosed properly and are compatible. Care with ventilator transfers and settings must also be maintained because oxygen desaturation may occur easily. Patients can be negatively affected by movement and agitation.

Although patients with sepsis are infrequently transported long distances by fixed-wing transport, when they are, altitude and cabin pressure require ventilator adjustments to compensate for pressures at altitude.

Summary

Often in the transport environment, a patient with sepsis will be critically ill, and multiple support devices and treatments will be in progress. The referring facility may have exhausted its resources to care for the patient. CCTPs must be well versed in many aspects of the management of shock, sepsis, SIRS, and MODS. Multiple therapies may be needed concurrently in the care of patients in critical condition because of these disorders.

Case Study

YOU ARE CALLED TO A REGIONAL HOSPITAL ICU for emergency ground transfer of a man to a tertiary facility. Three days earlier, the patient fell, sustained splenic trauma, and underwent emergency repair of a splenic tear. Transport time by ground will be 1 hour.

Upon arrival, staff reports this is a 35-year-old, 77-kg man who fell from a scaffold 3 days ago and was brought by EMS to the emergency department. He has no medical history and is a half-pack-a-day smoker and an occasional drinker. He was diagnosed with a splenic tear and underwent surgical repair. He was admitted to the ICU after surgery, has had worsening hemodynamic status, and is being referred to a tertiary facility for ongoing care. During his stay, he received packed RBCs, normal saline, and FFP for surgery and stabilization. A PA catheter was inserted, and dopamine therapy was initiated to maintain hemodynamic status. Cefazolin (Ancef) was given as surgical prophylaxis. He continues to need a ventilator as well.

He is currently mildly responsive, with a Glasgow Coma Scale score of 7T (T indicates that the patient is intubated). He is intubated with a 7.5 endotracheal tube and ventilated with synchronized intermittent mandatory ventilation. The tidal volume is 700 mL, the rate is 10 breaths/min, FIO_2 is 50%, and PEEP is 5 cm. Occasional suctioning is required. Two peripheral lines are in place, with normal saline running at 100 mL/h. Dopamine is infusing at 6 µg/kg/min. He is receiving enteral nutrition via a nasally inserted duodenal tube. His PA catheter is revealing lowered PCWP, decreased SVR, and increased CO. An available chest x-ray reveals bilateral diffuse infiltrates. His temperature is 102°F (38.9°C). Current monitoring reveals the following: heart rate, 133 beats/min; blood pressure, 84/43 mm Hg; temperature, 38.9°C; MAP, 55 mm Hg; CVP, 3 mm Hg; PA pressure, 20/10 mm Hg; PCWP, 7 mm Hg; SpO_2, 89%; and end-tidal carbon dioxide, 35 mm Hg. Current available lab values are as follows: hemoglobin, 11.0 g/dL; hematocrit, 33.1%; WBC count, 24, 100/µL; platelet count, 51 × 103/µL; BUN,

61 mg/dL; creatinine, 2.1 mg/dL; Na^+, 146 mEq/L; K^+ 4.8 mEq/L; Cl^-, 111 mEq/L; glucose, 225 mg/dL; pH, 7.29; P_{CO_2}, 52 mm Hg; Pa_{O_2}, 47 mm Hg; and HCO_3^-, 14 mEq/L. Urine output has been scant, at less than 50 mL/h.

Your assessment confirms the preceding information. The patient feels febrile to touch. The examination reveals equal and reactive pupils at 5 mm. The enteral feeding tube is placed nasally and secured. The endotracheal tube is secured at 22 cm at the lip line. The trachea is midline. Other portions of the head, eye, ear, nose, and throat exam have normal findings. The chest has equal rise and fall with ventilations. A PA catheter is placed in the right side and is secure. Lung sounds reveal bilateral, diffuse, light crackles. The abdomen reveals a splenic surgical incision that is intact and dressed. No exudate is noted. An indwelling urinary catheter is noted with a pelvic exam. The lower extremities are cool and pale. Pedal pulses can be detected by Doppler only. Faint radial pulses are noted. Capillary refill is delayed.

You transfer his IVs to your pumps. The ECG and hemodynamic lines are transferred and calibrated to your monitors. The patient is moved to your litter, and ventilator settings are duplicated on your portable ventilator, increasing the FIO_2 to 80%. You discharge the patient from the ICU, obtain all records, and move to your mobile ICU. He is placed in the mobile ICU, all oxygen and power needs are transferred to onboard systems, and transport is begun.

En route, no change in the preceding hemodynamic status is noted, and the patient continues to be hypoxic, with an oxygen saturation of 89%. Contact with medical control is made, and standing protocols are initiated.

Immediate attention is given to increasing the oxygenation and perfusion. Dopamine is increased to 10 µg/kg/min, and norepinephrine is considered as another pressor agent to increase the blood pressure and SVR. The ventilator F_{IO_2} is increased to 100% and PEEP to 10 cm. Only slight improvements of oxygenation (90%) and hemodynamics (blood pressure, 88/46 mm Hg) are noted. Hydrocortisone is also considered. Further discussions were to maintain current patient status, and further therapy will ensue on admission to the tertiary facility. No further changes are noted during the transport course, and the patient is delivered to the tertiary facility ICU without further incident.

1. What is your differential diagnosis?
2. What other treatment may be beneficial to this patient?
3. Is this an appropriate transfer based on the patient's condition?

Analysis

This patient is most likely experiencing sepsis and SIRS. MODS should be considered because of the respiratory failure and renal insufficiency. The infection could be secondary to the surgical exploration, or a result of mechanical ventilation (pneumonia) or perhaps an invasive line or catheter (intravascular or urinary).

Treatments en route to the receiving facility may include the following:

- Further increases in ventilator support by increasing F_{IO_2}, minute volume, and PEEP. Tidal volume should not be increased.
- Administration of chemical paralytics to facilitate ventilation. Sedative agents must be used with caution owing to the potential hypotensive qualities of these drugs.
- Ensuring that adequate fluid resuscitation has been performed *before* increasing any vasopressor doses; 4 to 6 L may be required. Blood products would be considered if the hematocrit value is less than 30%.

- Adding an infusion of another vasopressor agent, such as norepinephrine, epinephrine, phenylephrine, or vasopressin *after* adequate fluid resuscitation.
- Dobutamine therapy may be instituted only after adequate fluids and vasopressor support and may enhance tissue oxygen delivery.
- Steroid therapy may be instituted to combat inflammatory responses.
- Blood glucose management because patients with sepsis often are hyperglycemic.

This is an appropriate transfer because the patient will benefit from further treatment at a tertiary facility, such as the following:

- Further ventilator support
- Studies and determination of appropriate antibiotic therapy
- Possible surgical consult and intervention
- Anticoagulation and inflammatory therapies

Sepsis and MODS continue to be complex challenges to CCTPs. Despite recent advances in the critical care arena, sepsis and MODS continue to have high mortality and morbidity rates. New therapies are helpful, but early detection and intervention are still the mainstay of positive outcomes. Identification of risk factors and subtle changes in the condition of the patient are required to identify early onset of one of the disorders and for initiation of aggressive therapy to reverse the process and gain a favorable outcome. Although the critical care transport arena is limited in therapy that may reverse the process, it still serves a vital role in support of patients. Transport staff have the resources and must remain vigilant in the support of failing organ systems, restoration of intravascular volume, and adequate oxygenation to meet metabolic demand.

Prep Kit

Ready for Review

- All cells require energy, a continuous supply of oxygen, and nutrients. From those nutrients, mitochondria manufacture energy in the form of adenosine triphosphate (ATP). That process is called cellular respiration.
- Cellular respiration has three parts: glycolysis, the citric acid cycle, and the electron transport chain (oxidative phosphorylation). Unlike the other two parts of cellular respiration, which require oxygen, glycolysis also can occur in an anaerobic environment. But this solo attempt to support cellular respiration causes a reduction in ATP and an increase in lactic acid, which can result in cell damage, impaired function of tissue, and a cascade of injurious effects.
- To get oxygen to the body's cells, the respiratory system must allow oxygen and carbon dioxide exchange across the alveolar-capillary membrane. Once this transfer occurs, arterial blood transports oxygen to the tissues (the oxygen is dissolved in plasma [3%] or is bound to hemoglobin [97%]).
- Various factors can affect the delivery of oxygen-rich blood to the tissues, including blood volume, viscosity, and arterial elasticity. However, the primary determinates of transport are blood pressure (it must be stable) and cardiac output (it must be adequate).
- Under normal conditions, only 25% of the oxygen delivered to the tissues of the body is actually extracted. This allows the body a buffer zone for periods of low oxygen delivery, during which the cells must extract more oxygen. Therefore, management of shock is geared toward maximizing oxygen

consumption by maintaining adequate oxygen supply, providing hemoglobin through administration of blood products, and optimizing cardiac output. If there is a breakdown in one or more of these components, homeostasis is lost.

- Shock is a progressive, whole-body response to an inadequate supply of oxygen within cells, tissues, and organs, from one or multiple causes. It is commonly classified in four stages, and the mortality rate increases with progression:
  - During initial or early shock, blood flow into the micro-circulatory beds decreases and lactic acid levels begin to rise. However, compensatory mechanisms, such as an increase in oxygen consumption, are able to keep vital signs at baseline.
  - During the compensatory stage of shock, neural, hormonal, and chemical mechanisms attempt to compensate for the now severely reduced delivery of oxygen. They, in effect, cover up low circulatory volumes, poor stroke volume, and falling cardiac output, especially in children and healthy young adults. Common signs and symptoms are as subtle as mild tachypnea and tachycardia. The MAP may decrease 10 to 15 mm Hg from baseline. Cardiac output also may drop slightly.
  - During the progressive or decompensating stage of shock, multifaceted systemic responses to cell breakdown occur, affecting nearly every organ of the body. Compensatory mechanisms have begun to fail. Patients have more pronounced signs of shock, including altered mental status, tachycardia, and a drop in MAP of more than 20 mm Hg from baseline. Laboratory analysis shows a progression of acidosis, hyperkalemia, and climbing lactate levels. This is a life-threatening emergency that requires immediate treatment.
  - During the refractory or irreversible stage of shock, compensatory mechanisms have failed. At this stage, the patient is unresponsive to verbal stimuli. Blood pressure will be inadequate and the heart rate will be increased. The respiratory rate is increased and respirations are shallow. Skin will be cold, cyanotic, and/or mottled and peripheral pulses are weak and thready to absent. Urinary output will decrease and bowel sounds will be absent. Anaerobic metabolism progresses to permanent organ dysfunction and treatment no longer can reverse the mass effects. Multiple organ failure ensues and the risk of mortality is at its highest.
- MODS is diagnosed when two or more organs stop functioning. The organs affected early in MODS are the brain, kidneys, liver, adrenal glands, and heart. Sepsis is the leading cause of MODS.
- Shock traditionally is classified by cause. More than one type of shock may be present at the same time.
- Cardiogenic shock results when the ventricles are unable to pump blood forward, ultimately decreasing cardiac output. Manifestations vary depending on the underlying cause, but common signs and symptoms are low blood pressure; altered mental status; cool, pale, diaphoretic skin; decreased urine output; weak and thready pulse; distant or abnormal S_3 or S_4 heart sounds; fluid accumulation in the lungs or limbs; and tachypnea. Management focuses on enhancing cardiac output while decreasing left ventricular work load. Hypovolemic shock is present when there is too little circulating blood volume within the vascular system, resulting in hypotension. Manifestations include tachycardia; hypotension; signs of poor tissue perfusion; altered mental status; and cold, mottled, and pulseless extremities. Management revolves around treating the underlying condition, administering oxygen, and initiating volume replacement.
- Distributive shock is a term used to describe several types of shock involving loss of vasomotor tone or increased vascular permeability: neurogenic shock, anaphylactic shock, and septic shock.
- Neurogenic shock results from conditions that impede the ability of the sympathetic nervous system to control the constriction and dilation of vessel walls, such as trauma to the brain or spinal cord. The signs and symptoms associated with neurogenic shock can be very different from those found in

hypovolemic shock; most profound is the presence of bradycardia in the face of hypotension. It is vital to piece together all possible causes of hypotension to determine the best treatment, which generally focuses on maintaining an MAP of greater than 70 mm Hg or a systolic blood pressure of greater than 90 mm Hg.

- Anaphylactic shock is a severe, life-threatening allergic reaction producing systemic vasodilation in response to histamine release. The reaction can be massive, and it can occur minutes after exposure or it may be delayed for hours or days. Manifestations include bronchoconstriction, laryngeal edema, and angioedema resulting in airway compromise, which requires rapid treatment to limit effects. Standard oxygen therapy may not be enough; patients may require intubation, an emergency cricothyrotomy, or tracheostomy to maintain an adequate airway.
- Septic shock is a progressive disease that typically occurs from an infection. Septic shock usually starts as a localized infection and then develops into a widespread inflammatory process, referred to as systemic inflammatory response syndrome (SIRS). When hypotension develops, septic shock is present.
- Infection is the leading cause of SIRS and septic shock. However, any shock state can cause the beginning of SIRS and lead to sepsis before reaching multiple organ failure. The minimum criteria for early recognition of SIRS relate to body temperature, respiratory rate, pulse rate, and white blood cell count.
- A patient who has SIRS with a known or highly suspected source of infection has sepsis. Sepsis may be a mild systemic response to infection or suspected infection. In contrast, severe sepsis is sepsis with a confirmed or highly suspected diagnosis of infection and organ dysfunction of at least one organ. Septic shock incorporates hemodynamic instability with SIRS and a confirmed or highly suspected source of infection that is unresponsive to fluid resuscitation. Sepsis may progress through septic shock and result in organ failure and MODS.
- The prevalence of sepsis-related infections is increasing. They affect all populations and age groups and are associated with (in order of prevalence) diabetes, hypertension, congestive heart failure, COPD, cirrhosis, HIV infection, cancer, and pregnancy. Prolonged hospitalization is a risk factor, as are invasive procedures, comorbidities, and use of immunosuppressants. The mortality rate for sepsis increases with severity; sepsis is the leading cause of death in noncoronary ICUs and the 10th leading cause of death in the United States.
- Bacteria are the pathogens most commonly associated with the development of sepsis, although fungi, viruses, and parasites also can cause sepsis. Infection can occur at any body site, including lungs, abdomen, skin, and urinary tract, and as a result of primary bloodstream infection. Gramnegative and gram-positive bacteria are the two most common organisms responsible for sepsis.
- Sepsis is part of a dynamic hypermetabolic, hyperinflammatory response to an insult that grossly affects the endothelium. The three identifiable physiologic processes involved with the development of microvascular abnormalities associated with severe sepsis are proinflammation, procoagulation, and decreased fibrinolysis.
- Microvascular disruption and injury are responsible for hypoperfusion and the shunting of oxygen, resulting in decreased oxygen delivery to the tissues. The profound imbalance between oxygen delivery and consumption causes hypoxia within the cells and tissues. This complex cascade of events is the primary cause of severe sepsis and its progression to organ dysfunction and failure.
- The first priority in recognizing sepsis is to maintain a high index of suspicion for distributive shock in two phases: hyperdynamic (warm) and hypodynamic (cold). The hyperdynamic phase has high cardiac

output lasting hours or days. It can progress to the hypodynamic phase, a rapid deterioration with a sudden drop in cardiac output. Other manifestations of sepsis include fever (may or may not be present), hypothermia in later stages, tachycardia, normal-to-low blood pressure progressing to hypotension, normal skin progressing to cold and clammy skin with pallor or cyanosis, petechiae and blood oozing from mucous membranes and procedure sites, decreased pulse pressure, increased respiratory rate and depth, respiratory alkalosis, and ARDS.

- The management of sepsis is aimed at maintaining organ perfusion while enhancing tissue oxygenation. Management requires balancing preload, afterload, and contractility to achieve targeted criteria for an optimal level of homeostasis.
- Blood administration is sometimes performed to manage shock and should be considered in cases of hemorrhagic blood loss as a result of trauma, internal hemorrhage, perioperative and postoperative complications, specific disease entities, anemia due to illness, or coagulation disorders.
- The decision to give a transfusion during transport is complex. Factors to consider include the urgency of the transfusion, the "out-of-hospital" time, availability of the blood products, type and crossmatch information, and appropriate transport and care of the blood product.
- Steps for proper blood administration include appropriate typing and crossmatching, appropriate IV access, blood tubing administration sets, priming of the tubing with normal saline, validation of the blood product's match with the specific patient before administration, and continued supervision by a skilled health care provider.
- In an emergency situation in which the time for crossmatching cannot be taken, the use of the universal donor type O, Rh-negative blood or plasma can be transfused until crossmatched blood becomes available.
- Blood contains a variety of antigens that influence the compatibility of blood between persons. Three types of blood antigens are the ABO blood group, Rh factor, and the HLA blood group. These are considered when crossmatching.
- A person with only an A antigen is type A. A person who has only a B antigen is type B. If both A and B antigens are identified, the person is type AB. When neither A nor B are identified, the person's blood type is O.
- People with type AB blood are universal recipients; they can receive blood types A, B, AB, and O without the threat of an ABO reaction. Persons with type O blood are universal donors; their blood can be transfused into patients with type A, B, and AB blood.
- Rh factors are antigens found on the cell membrane of red blood cells. An Rh-negative person may develop an Rh antibody if exposed to Rh-positive blood. A second Rh-positive exposure could result in a fatal hemolytic reaction.
- HLA is present on the cell membrane surface of circulating platelets, white blood cells, and most tissue cells. Patients receiving platelets from multiple donors may experience febrile transfusion reactions as a result of HLA. This type of reaction destroys the platelets, reducing the benefit of the transfusion.
- Blood administration can occur in many different forms: whole blood, packed red blood cells, platelets, FFP, cryoprecipitate, albumin, plasma protein fractions, and synthetic blood substitutes.
- Whole blood contains all blood components (red blood cells, white blood cells, platelets, plasma, and electrolytes). Transfusion with whole blood is uncommon and indicated only in acute massive blood loss for blood volume expansion and increased oxygen-carrying capacity. Disadvantages are significant, including fluid volume overload in patients with cardiac compromise and degradation after 24 hours of storage.

- PRBCs retain all of the characteristics of whole blood with the exception of the extraction of approximately 250 mL of platelet-rich plasma from each unit of whole blood. Anemia and blood loss are possible indications for PRBC administration.
- Platelet therapy may be considered for patients with extremely low platelet levels, those who are bleeding due to thrombocytopenia, or patients with abnormally functioning platelets. If a patient must receive multiple transfusions of platelets, single-donor platelets should be administered.
- FFP is uncoagulated plasma separated from the red blood cells. Indications for the administration of FFP may include blood loss, coagulation deficiencies, warfarin reversal, and thrombotic thrombocytopenia purpura. Close patient monitoring for hypocalcemia is required during transfusion.
- Cryoprecipitate is a frozen blood product created from the plasma of a donor. It contains factor VIII, fibrinogen, von Willebrand factor, and factor XIII, and is useful in treating patients with clotting disorders. Administration of cryoprecipitate does not require compatibility testing.
- Albumin is prepared by the fractionation of pooled plasma. Albumin is used for volume replacement in conditions such as burns, trauma, surgery, or infections. Patients with cardiac and pulmonary disease must be monitored closely during albumin administration.
- Plasma protein fraction contains 83% albumin and 17% globulins. Plasma protein fraction is indicated for volume expansion in patients with hypovolemia and hypoproteinemia. Clinical settings for the administration of plasma protein fraction may include shock and burn patients.
- When performing blood administration, it is important to follow transfusion precautions:
  - Do not mix blood with 5% dextrose in water (causes hemolysis).
  - Do not mix with lactated Ringer's solution (causes clotting).
  - Do not mix with medications (may react).
  - Have a second venous access line available.
- Blood administration can result in transfusion reactions. Transfusion reactions can be as mild as chills or as catastrophic as death. Signs include body temperature of 2°F (–16°C) or more above the baseline temperature; hives, itching, or skin symptoms; swelling, soreness, or hematoma at the venous site; flank pain; tachycardia; respiratory distress (wheezing and dyspnea); hypotension; bleeding from widely varied sites or previously clotted wounds; blood in the urine; anaphylaxis; and nausea and vomiting.
- Main types of transfusion reactions include allergic reactions, bacterial contamination, febrile transfusion reactions, hemolytic transfusion reactions, and plasma protein incompatibility.
- Allergic reactions caused by allergens in the donated blood have symptoms of anaphylaxis (ie, chills, facial and laryngeal edema, pruritus, urticaria, and wheezing) as well as fever, nausea, and vomiting. Management includes the use of antihistamines, careful patient monitoring and assessment, and possible epinephrine and corticosteroid administration.
- Bacterial contamination of blood components usually occurs during phlebotomy, component preparation or processing, or thawing of the blood components. Presenting symptoms may occur within 30 minutes after transfusion begins and include chills, fever, vomiting, abdominal cramping, bloody diarrhea, hemoglobinuria, shock, renal failure, and DIC. It is essential to rapidly recognize these symptoms, stop the transfusion immediately, and implement the protocol for transfusion reactions.
- Febrile transfusion reactions occur when symptoms include a temperature as high as 104°F (40°C), chills, headache, facial flushing, palpitations, cough, chest tightness, increased pulse rate, and flank pain. Treatment includes the administration of antipyretics and antihistamines.
- Hemolytic transfusion reactions are caused by ABO or Rh incompatibility, intradonor incompatibility,

improper crossmatching, or improper blood storage.

- Immediate hemolytic transfusion reactions may occur during transfusion or within 3 to 7 days following transfusion. Red blood cells are destroyed, and hemoglobin and red blood cell remnants are released into the bloodstream. Symptoms include chest pain, facial flushing, shortness of breath, chills, fever, hypotension, flank pain, hemoglobinuria, oliguria, bloody oozing at the infusion site, burning along the vein receiving the blood, shock, signs of renal failure, and DIC. Treatment of immediate hemolytic transfusion reactions is directed toward prevention and supportive management.
- Delayed hemolytic transfusion reactions do not occur until 3 to 7 days after transfusion. Signs and symptoms are usually mild and include mild fever, chills, and moderate jaundice, with treatment focused on the prevention and the treatment of severe complications should they arise.
- Plasma protein incompatibility is caused by immunoglobulin A incompatibility. Clinical presentations include abdominal pain, diarrhea, shortness of breath, chills, fever, flushing, and hypotension. Management includes oxygen administration, fluids, epinephrine, and corticosteroids as indicated.
- Hypocalcemia may occur when blood products containing citrate are infused too quickly and bind calcium, causing calcium deficiency and leading to arrhythmias, hypotension, muscle cramping, nausea, vomiting, seizure activity, and/or a tingling sensation in the fingers. Slowing or stopping the transfusion may be indicated. Management considerations include the administration of calcium gluconate IV infused slowly.
- High potassium levels in stored plasma may result in potassium intoxication during blood administration. Clinical manifestations include diarrhea, intestinal colic, flaccidity, muscle twitching, oliguria, signs of renal failure, bradycardia, ECG changes with visible tall peaked T waves, and/or cardiac arrest. Management considerations include a diagnostic 12-lead ECG tracing; Kayexalate administration; administration of an albuterol nebulizer; administration of furosemide; and the infusion of 50% glucose, insulin, sodium bicarbonate, or calcium.
- Hypothermia may result from a rapid infusion of large amounts of cold blood products. Hypothermia symptoms include chills, shivering, hypotension, arrhythmias, bradycardia, and/or cardiac arrest. Treatment may include stopping the transfusion, warming the patient, obtaining a diagnostic 12-lead ECG, and warming the blood if the transfusion is resumed.

Vital Vocabulary

ABO-incompatible transfusion reactions A type of transfusion reaction in which the patient possesses antigens to a blood type and receives that blood type.

adaptive immune system The secondary mechanism that protects the host by reacting with and eliminating specific antigens after requiring more time than the innate immune system to mobilize its defense against unknown pathogens.

aerobic metabolism A form of energy production in which mitochondria utilize glucose, amino acids, and fatty acids combined with oxygen and ADP to produce ATP, carbon dioxide, water, and heat.

albumin A blood product containing this specific protein found in the blood, and prepared by the fractionation of pooled plasma; used for volume replacement in certain conditions.

anaerobic metabolism A less efficient form of energy production in which an alternative pathway converts glucose to pyruvic acid with the simultaneous production of ATP and that also results in the production of lactate.

anaphylactic shock A severe hypersensitivity reaction that involves bronchoconstriction and cardiovascular collapse.

anaphylactoid reaction A non–IgE-mediated response that causes the rupture of mast cells and basophils, which then release histamine and other defense mediators.

antigen Any substance that can create an immune response in the body.

arterial oxygen content (CaO_2) The total amount of oxygen in the arterial blood.

cardiac index A hemodynamic value that adjusts a patient's cardiac output to take into accout his or her total body surface area.

cardiac output (CO) The amount of blood pumped by the heart per minute, calculated by multiplying the stroke volume by the heart rate per minute.

cardiogenic shock A condition caused by loss of 40% or more of the functioning myocardium; the heart is no longer able to circulate sufficient blood to maintain adequate oxygen delivery.

cryoprecipitate A blood product created from plasma and in which clotting factors, especially factor VIII, are concentrated; used to treat patients with coagulation disorders.

cytokines Chemical messengers that enhance cell growth, promote cell activation, direct cellular traffic, stimulate macrophage function, and destroy antigens; interleukins are a type of cytokine.

delayed hemolytic transfusion reactions A type of transfusion reaction that does not occur until 3 to 7 days after transfusion.

disseminated intravascular coagulation (DIC) A complex condition arising from different causes that activate coagulation mechanisms, resulting in obstructed blood flow as a result of microdots; in fibrinolysis, the attempt by the body to reopen the microcirculation. Bleeding and thrombosis and, potentially, organ dysfunction result.

distributive shock A condition that occurs when there is widespread dilation of the resistance vessels, the capacitance vessels, or both.

ejection fraction The portion of the blood ejected from the ventricle during systole.

fresh-frozen plasma (FFP) A blood product in which uncoagulated plasma has been separated from the red blood cells; primarily composed of water, proteins, salts, metabolites, and clotting factors.

hemolysis A situation in which red blood cells are destroyed, resulting in the release of hemoglobin and red blood cell remnants into the bloodstream.

hemolytic transfusion reaction A type of transfusion reaction caused by ABO or Rh incompatibility, intradonor incompatibility, improper crossmatching, or improper blood storage.

human leukocyte antigen (HLA) An antigen present on the cell membrane surface of circulating platelets, white blood cells, and most tissue cells.

hypercapnia Greater than normal amounts of carbon dioxide in the blood.

hyperdynamic state A condition that occurs as the first stage of distributive shock and is characterized primarily by high cardiac output and low peripheral vascular resistance; also known as warm shock.

hypodynamic state A condition that occurs as the second stage of distributive shock and is characterized primarily by a subnormal temperature, a low white blood cell count, profound hypotension and hypoperfusion, and a sudden drop in cardiac output; also known as cold shock.

hypovolemic shock A condition that occurs when the circulating blood volume is inadequate for delivering adequate oxygen and nutrients to the body.

immediate hemolytic transfusion reactions A type of transfusion reaction that usually occurs soon (between 1 and 2 hours) after the transfusion of incompatible red blood cells.

innate immune system The primary antigen and immunogen-nonspecific defense mechanism that protects the host by eliminating microbes to prevent infection and other antigens in an attempt to prevent allergic reactions.

leukotrienes A class of biologically active compounds that occur naturally in leukocytes and that produce allergic and inflammatory reactions.

mean arterial pressure (MAP) The average (or mean) pressure against the arterial wall during a cardiac cycle.

microcirculation Circulation that occurs in the microvasculature, the body's smallest vessels (arterioles, capillaries, and venules).

multiple organ dysfunction syndrome (MODS) Altered organ function in acutely ill patients, which is diagnosed when two or more organs stop functioning.

neurogenic shock Circulatory failure caused by paralysis of the nerves that control the size of the blood vessels, leading to widespread dilation; seen in spinal cord injuries.

neutrophils One of the types of leukocytes (white blood cells); these numerous phagocytic microphages usually are the first of the mobile phagocytic cells to arrive at an area of injury or infection.

oxygen consumption ($\dot{V}O_2$) The amount of oxygen used by the cells and tissues.

oxygen delivery (DO_2) The amount of oxygen delivered to the tissues each minute.

oxygen extraction ratio (ERO_2) The relationship between oxygen consumption and oxygen delivery, or the cells' ability to utilize oxygen.

packed red blood cells (PRBCs) A blood product that retains all of the characteristics of whole blood with the exception of the extraction of approximately 250 mL of platelet-rich plasma from each unit of whole blood.

pathogens The microorganisms responsible for activating the immune system and that cause disease; includes viruses, parasites, fungi, and bacteria.

plasma protein fraction A blood product that contains 83% albumin and 17% globulins.

primary MODS Multiple organ dysfunction syndrome that results from a direct insult such as trauma.

refractory stage A stage in shock in which there is persistently low mean arterial blood pressure despite vasopressor therapy and adequate fluid resuscitation.

Rh factors Antigens found on the cell membrane of red blood cells.

secondary MODS Multiple organ dysfunction syndrome that is a slower, more progressive insult to organs and frequently results from the sepsis cascade.

sepsis Systemic response to infection manifested by two or more symptoms noted with SIRS.

septic shock A form of shock that occurs in septicemia when endotoxins or exotoxins are released from certain bacteria in the bloodstream, resulting in a persistently low mean arterial blood pressure despite adequate fluid resuscitation.

severe sepsis A type of sepsis with associated acute organ failure; hypoperfusion or hypertension is present.

systemic inflammatory response syndrome (SIRS) A widespread inflammatory process associated with

infectious and noninfectious causes, without end-organ damage.

transfusion reaction A reaction resulting from an endogenous or exogenous factor related to transfused blood.

true anaphylaxis An anaphylactic reaction that results when the allergen binds to IgE on the cell membranes of basophils and when mast cells stimulate the release of histamine from the cell.

tumor necrosis factor (TNF) A protein mediator released primarily by macrophages and T lymphocytes that help regulate the immune response.

type-and-crossmatch The test to determine compatibility between patient serum and donor red blood cells prior to transfusion.

universal donors Persons who have type O blood.

universal recipients Persons who have type AB blood.

Bibliography

The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med.* 2000;342(18):1301–1308.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support for Doctors: ATLS Student Course Manual,* 8th ed. Chicago, IL: American College of Surgeons; 2009.

Angus DC, Linde-Zwirble WT, Lidicker J, et al. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. *Crit Care Med.* 2001;29(7):1303–1310.

Bakker J, Coffernils M, Leon M, et al. Blood lactate levels are superior to oxygen-derived variable in predicting outcome in human septic shock. *Chest.* 1991;99(4):956–962.

Beale RJ, Hollenberg SM, Vincent JL, et al. Vasopressor and inotropic support in septic shock: an evidence-based review. *Crit Care Med.* 2004;32(11 suppl):S455–S465.

Bone RC, Balk RA, Cerra FB, et al. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis: the ACCP/SCCM Consensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine. *Chest.* 1992;101(6):1644–1655.

Cauwels A, Janssen B, Buys E, et al. Anaphylactic shock depends on PI3K and eNOS-derived NO. *J Clin Invest.* 2006;116(8):2244–2251.

Cohen J, Brun-Buisson C, Torres A, et al. Diagnosis of infection in sepsis: an evidence-based review. *Crit Care Med.* 2004;32(11 suppl):S466–S494.

Convertino VA, Ryan KL, Rickards CA, et al. Inspiratory resistance maintains arterial pressure during central hypovolemia: implications for treatment of patients with severe hemorrhage. *Crit Care Med.* 2007;35(4):1145–1152.

Dellinger RP, Levy MM, Carlet JM, et al; for the International Surviving Sepsis Campaign Guidelines Committee. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008 [published correction appears in *Crit Care Med.* 2008;36(1):296]. *Crit Care Med.* 2008;36(4):1394–1396. Available at: http://www.survivingsepsis.org/system/files/images/2008_Guidelines_Final_.pdf. Accessed March 24, 2009.

Eastridge BJ, Salinas J, McManus JG, et al. Hypotension begins at 110 mm Hg: redefining "hypotension"

with data. *J Trauma.* 2007;63(2):291–299.

El-Banayosy D, Cobaugh A, Zittermann L, et al. A multidisciplinary network to save the lives of severe, persistent cardiogenic shock patients. *Ann Thorac Surg.* 2005;80(2):543–547.

Filbin MR. Septic shock. Emedicine. Available at: http://emedicine.medscape.com/article/786058-overview. Updated December 17, 2008. Accessed February 23, 2009.

Finfer S, Bellomo R, Boyce N, et al; for the SAFE Study Investigators. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med.* 2004;350(22):2247–2256.

Guly HR, Bourmra O, Lecky FE; for the Trauma Audit and Research Network. The incidence of neurogenic shock in patients with isolated spinal cord injury in the emergency department [published online ahead of print August 3, 2007]. *Resuscitation.* 2008;76:57–62. doi:10.1016/j.resuscitation.2007.06.008.

Hochman JS, Boland J, Sleeper LA, et al; and the SHOCK Registry Investigators. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an international registry. *Circulation.* 1995;91(3):873–881.

Hoyert DL, Arias E, Smith BL, et al. Deaths: final data for 1999. *Natl Vital Stat Rep.* 2001;49(8):1–113.

Ince C, Sinaasappel M. Microcirculatory oxygenation and shunting in sepsis and shock. *Crit Care Med.* 1999;27(7):1369–1377.

Kaplow R, Hardin S. *Critical Care Nursing: Synergy for Optimal Outcomes.* Sudbury, MA: Jones and Bartlett Publishers; 2007;611.

Krausz MM. Initial resuscitation of hemorrhagic shock [published online April 27, 2006]. *World J Emerg Surg.* 2006;1:14. doi:10.1186/1749-7922-1-14.

Kress JP, Pohlman AS, O'Connor MF, et al. Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. *N Engl J Med.* 2000;342(20):1471–1477.

Kutko MC, Calarco MP, Flaherty MB, et al. Mortality rates in pediatric septic shock with and without multiple organ system failure. *Pediatr Crit Care Med.* 2003;4(3):333–337.

Labelle C, Kitchens CS. Disseminated intravascular coagulation: treat the cause, not the lab values. *Cleveland Clinic J Med.* 2005;72(5):377–397.

LaRosa, SP. Sepsis. The Cleveland Clinic Center for Continuing Education. Available at: http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/infectious-disease/sepsis/. Accessed March 25, 2009.

Lee CC, Marill KA, Carter WA, et al. A current concept of trauma-induced multiorgan failure. *Ann Emerg Med.* 2001;38(2):170–176.

Marshall JC, Cook DJ, Christou NV, et al. Multiple organ dysfunction score: a reliable descriptor of complex clinical outcome. *Crit Care Med.* 1995;23(10):1638–1652.

Martin GS, Mannino DM, Eaton S, et al. The epidemiology of sepsis in the United States from 1979 through 2000. *N Engl J Med.* 2003;348(16):1546–1554.

Mehta RH, Grab JD, O'Brien SM, et al; for the Society of Thoracic Surgeons National Cardiac Database Investigators. Clinical characteristics and in-hospital outcomes of patients with cardiogenic shock undergoing coronary artery bypass surgery: insights from the Society of Thoracic Surgeons National Cardiac Database. *Circulation.* 2008;117(7):876–885.

Meybohm P, Renner J, Boening A, et al. Impact of norepinephrine and fluid on cerebral oxygenation in experimental hemorrhagic shock. *Pediatr Res.* 2007;62(4):440–444.

Moore FA, McKinley BA, Moore EE. The next generation in shock resuscitation. *Lancet.* 2004;363(9425):1988–1996.

Morgan B. Inflammation and coagulation overview. *Critical Care Concepts.* 2005:1–8.

National Heart, Lung, and Blood Institute (NHLBI) ARDSNet. NIH NHLBI ARDS Clinical Network Mechanical Ventilation Protocol Summary. Available at: http://www.ardsnet.org/system/files/Ventilator+Protocol+Card.pdf. Accessed February 23, 2009.

Nguyen HB, Rivers EP, Abrahamian FM, et al; Emergency Department Sepsis Education Program and Strategies to Improve Survival (ED-SEPSIS) Working Group. Severe sepsis and septic shock: review of the literature and emergency department management guidelines. *Ann Emerg Med.* 2006;48(1):28–50.

Oppert M, Engel C, Brunkhorst FM, et al. Acute renal failure in patients with severe sepsis and septic shock: a significant independent risk factor for mortality: results from the German Prevalence Study. *Nephrol Dialysis Transplant.* 2008;2(3):904–909.

Parks JK, Elliott AC, Gentilello LM, et al. Systemic hypotension is a late marker of shock after trauma: a validation study of Advanced Trauma Life Support principles in a large national sample. *Am J Surg.* 2006;192(6):727–731.

Parks R. Clotting Factor Replacement for Hemophilia. WebMD. 2007. Available at: http://www.webmd.com/a-to-z-guides/clotting-factor-replacement-for-hemophilia. Accessed May 11, 2009.

Pesaturo AB, Jennings HR, Voils SA. Terlipressin: vasopressin analog and novel drug for septic shock. *Ann Pharmacother.* 2006;40(12):2170–2177.

Phua J, Koay ES, Lee KH. Lactate, procalcitonin, and amino-terminal pro-B-type natriuretic peptide versus cytokine measurements and clinical severity scores for prognostication in septic shock. *Shock.* 2008;29(3):328–333.

Prough D, Kramer G, Uchida T, et al. Effects of hypertonic arginine on cerebral blood flow and intracranial pressure after traumatic brain injury combined with hemorrhagic hypotension. *Shock.* 2006;26(3):290–295.

Proulx F, Fayon M, Farrell CA, et al. Epidemiology of sepsis and multiple organ dysfunction syndrome in children. *Chest.* 1996;109(4):1033–1037.

Resuscitation: fluid therapy: early goal-directed resuscitation of sepsis: from the emergency room to the ICU. Program and abstracts of the 32nd Critical Care Congress; January 28–February 2, 2003; San Antonio, TX.

Revell M, Greaves I, Porter K. Endpoints for fluid resuscitation in hemorrhagic shock. *J Trauma.* 2003;54(5 suppl):S63–S67.

Reynolds HR, Hochman JS. Cardiogenic shock: current concepts and improving outcomes. *Circulation.* 2008;117(5):686–697.

Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med.* 2001;345(19):1368–1377.

Rossaint R, Cerny V, Coats TJ, et al. Key issues in advanced bleeding care in trauma. *Shock.* 2006;26(4):322–331.

Sakr Y, Dubois M, De Backer D, et al. Persistent microcirculatory alterations are associated with organ failure and death in patients with septic shock. *Crit Care Med.* 2004;32(9):1825–1831.

Sepsis Management Guidelines. Eli Lilly and Co. 2008. Available at: http://www.xigris.com/350-sepsis-management-guidelines.jsp. Accessed March 25, 2009.

Su F, Wang Z, Cai Y, et al. Fluid resuscitation in severe sepsis and septic shock: albumin, hydroxyethyl starch, gelatin or Ringer's lactate: does it really make a difference? *Shock.* 2007;27(5):520–526.

Vervloet MG, Thijs LG, Hack CE. Derangements of coagulation and fibrinolysis in critically ill patients with sepsis and septic shock. *Semin Thromb Hemost.* 1998;24(1):33–44.

Vincent JL, Gerlach H. Fluid resuscitation in severe sepsis and septic shock: an evidence-based review. *Crit Care Med.* 2004;32(11 suppl):S451–S454.

Zaza T, Thameem D, Oba Y. High PEEP levels may save lives in ARDS/ALI: meta-analysis of randomized clinical trials. Presented at the 2008 meeting of the American College of Chest Physicians; October 29, 2008. Available at: http://meeting.chestjournal.org/cgi/content/abstract/134/4/p89002. Accessed February 23, 2009.

Zeerleder S, Hack CE, Wuillemin WA. Disseminated intravascular coagulation in sepsis. *Chest.* 2005;128(4):2864–2875.


CUE

Trauma

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Objectives

1. Understand the CCTP's impact on preventing trauma deaths by performing proper prehospital care and transporting to the appropriate trauma center (p 308).
2. Understand the significance of trauma management on morbidity and mortality (p 308).
3. Understand the various trauma scoring systems, including the Glasgow Coma Scale, the trauma score, the revised trauma score, the abbreviated injury scale, the injury severity score, and the trauma injury severity score, and how they are used (p 314–316).
4. Understand Newton's first, second, and third laws of motion and how they relate to patterns of injury (p 308–309).
5. Discuss the types of trauma (p 309).
6. Describe the steps of the assessment process that are specific to critical care trauma patients (p 309–310).
7. Explain the concept of triage and standard triage systems (START and JumpSTART) (p 310–313).
8. Discuss the classifications of trauma centers defined by the American College of Surgeons' Committee on Trauma, including level I, II, III, and IV trauma centers (p 316–317).
9. Discuss the American College of Surgeons' Committee on Trauma patient classifications (p 316–317).
10. Understand how to assess a trauma critical care patient (p 317).
11. Know how to recognize, assess, and manage the most common critical care injuries, including pneumothorax (open, tension, and simple), hemothorax, flail chest, pericardial tamponade, aortic dissection, myocardial contusion, diaphragmatic rupture, tracheobronchial disruption, pulmonary contusion, esophageal perforation, and traumatic asphyxia (p 321–327, 330–337).
12. Know how to recognize, assess, and manage critical care ear, eye, neck, throat, and thyroid injuries (p 337–342).
13. Understand how and when to use diagnostic imaging, including the standard radiograph, computed tomography, magnetic resonance imaging, ultrasonography, intra-abdominal pressure monitoring, and related transport considerations (p 318–321).
14. Know the signs and symptoms as well as how to manage abdominal and pelvic injuries, including both hollow and solid organ injuries (p 342–346).
15. Recognize the different types of fractures and know how to manage them (p 346–348, 349–350).
16. Know how to recognize, monitor, and manage compartment syndrome, crush injuries, and rhabdomyolysis (p 350–352).
17. Understand pharmacology as it relates to trauma and know the specific treatments and medications (p

352–353).

18. Understand the specific trauma considerations for special populations (p 353–354).

Advances in Trauma Care

Trauma poses a significant threat to life. Since the time EMS began to evolve in the 1960s, more emphasis has been placed on rapidly identifying injuries and transporting patients to an appropriate trauma center for definitive care. The severity of the injuries in a traumatic incident will vary from minor to life threatening. Most local community hospitals should be able to manage and treat patients with minor injuries. All trauma patients need a trauma assessment to determine if they have serious injuries. The ability of the CCTP to appropriately care for trauma patients en route will help to decrease the incidence of morbidity and mortality related to their injuries.

Identifying life-threatening illnesses and injuries as soon as possible and transporting the patient to an appropriate facility have improved patient outcomes. This is a popular topic of discussion because trauma is a leading cause of death in persons younger than 40 years. According to the Centers for Disease Control and Prevention, traumatic injuries account for more than 117,000 deaths each year in the United States. Many preventable trauma deaths occur in the prehospital environment, further emphasizing the importance of proper prehospital care.

Renewed emphasis on education has helped to focus prehospital care providers' priorities at the scene of a trauma incident. Priorities include scene safety, rapid immobilization/stabilization, and transport.

CCTPs may find themselves dealing with trauma patients in two very different situations. One instance is the scene call, usually to back up field BLS or ALS providers. In these cases, the CCTP will be working in more austere and less controlled conditions with less sophisticated assessment tools than in the interfacility transport. On a scene call, the CCTP may be called to provide care not available to the standard field responder, such as rapid sequence intubation (RSI); or to provide speed of transport to surgery or blood transfusion, such as by rotor wing.

The other instance involves the CCTP transferring critical patients, usually from smaller facilities to trauma centers. In this situation, the CCTPs will be able to avail themselves of the results of laboratory results, imaging, and other sophisticated assessment findings prior to transport. The CCTPs may also be able to enlist the aid of the transferring physician or other house staff to help with stabilization efforts prior to transport by helping secure difficult airways, inserting chest tubes, and hanging blood or other treatments that may be better performed in the referring hospital rather than in a moving transport vehicle. Diplomatically enlisting this aid and advocating for the patient are also the jobs of the CCTP, in addition to providing care.

In both types of calls, the time it takes to stabilize the patient's condition for transport must be balanced against the need to transport an unstable patient immediately for care that cannot be provided in the field, such as surgery. A top priority of the CCTP is to determine the treatment that needs to be provided before transport vs what can be done en route.

Morbidity and Mortality

Trauma systems in the United States spend considerable time and effort in researching ways to decrease morbidity and mortality rates. Morbidity and mortality are often used synonymously with disability and death, because this is ultimately what advanced trauma systems are working to prevent. **Morbidity** refers to nonfatal injury and disability, whereas **mortality** refers to deaths caused by injury and disease. This is an area of concern for all trauma systems in the United States, because trauma is the leading cause of death in the pediatric population and a leading cause of death overall. Half of all deaths in children result

from trauma, accounting for more than 14,000 deaths annually.

Prevention and quality trauma care are two major contributors to decreasing morbidity and mortality rates. Quality prevention programs keep the trauma system and its patients from suffering needlessly from life-threatening and/or lifealtering injuries related to trauma. This is significant because a vast majority of trauma deaths are related to motor vehicle crashes. These crashes are often caused by factors such as alcohol, excessive speed, lack of physical restraints, and poor judgment. Most of these factors are easily preventable with an established and efficiently functioning prevention program.

An alarming statistic relating to motor vehicle crashes is the number of individuals who actually survive the crash, but suffer a debilitating injury that leaves them incapacitated. Many of these injuries involve the head and the spinal cord, and occur at the time of injury. The CCTP must realize the importance of avoiding secondary injury caused by improper handling. Patients with long-term debilitating injuries will spend the rest of their lives dependent on others for basic everyday needs and may require advanced nursing care, resulting in a huge cost to society (more than $200 billion annually) and to the patients and their families, both financially and emotionally.

Overview of Trauma

Newton's Laws of Motion

By far one of the foremost scientific intellects of all time, Sir Isaac Newton discovered three laws of motion. We review them here.

Newton's First Law

Newton's first law of motion deals with force and velocity, both of which are well exemplified in automobile crashes. Essentially, Newton says, "A body in motion remains in motion in a straight line unless acted upon by an outside force." The CCTP must understand this concept in order to predict patterns of injuries. For example, envision that a vehicle traveling at 50 mph hits a tree head on and comes to an abrupt stop. The chain of events during the collision includes the car striking the tree, the patient's chest striking the steering wheel, and internal organs striking the rib cage. Therefore, the exchange of energy within the body causes many different types of damage. A solid understanding of Newton's first law of motion better prepares the CCTP to deliver the appropriate standard of care to patients with traumatic injuries.

Newton's Second Law

Newton's second law of motion refers to an object's behavior when an outside force is applied. According to this law, acceleration is dependent on two variables—the mass of the object and the force upon the object. Therefore, if force is increased, so is acceleration. If mass is increased, the acceleration is decreased. Essentially, an object accelerates in the direction it is pushed. If you push twice as hard, it accelerates twice as much. Conversely, if the object has twice the mass, it will accelerate at half the speed.

Newton's Third Law

Newton's third law of motion is most commonly paraphrased as, "To every action, there is an equal and opposite reaction." In other words, if an automobile strikes a wall at 70 mph, the wall pushes back at 70 mph. A solid understanding of the third law is of particular importance to the CCTP when evaluating the mechanism of injury and predictability of injuries. For example, the CCTP would have a higher index of suspicion of a chest injury from an automobile that hit a tree head on going 70 mph than one going 5 mph.

Types of Trauma

Traumatic injuries are generally classified into one of two categories: blunt or penetrating. Patients may have injuries that fit into both classifications at the same time. For example, an individual who is stabbed in the back (penetrating injury) and then falls down a flight of steps (blunt injury) could experience both types of injuries. Blunt injuries result from energy exchange between an object and the body, without intrusion through the skin **Figure 10-1**. Examples include rapid forward decelerations (collisions), rapid vertical decelerations (falls), and energy transferred from blunt instruments such as a stick or bat. Penetrating injuries refer to injuries caused by external forces in which the tissue is penetrated by an object. Examples include projectiles, such as bullets, knives, and fragments from explosions and falls upon fixed objects.

Deceleration injuries are caused by a sudden stop of the body's forward motion. These blunt injuries can result from falls, automobile crashes, or abuse such as shaken baby syndrome. Common injuries involve shearing, avulsing, or rupturing of body organs, fascia, nerves, and other soft tissue. Often these types of injuries are not visible to the CCTP; therefore, a thorough understanding of the types of mechanisms of injury and effective assessment skills is imperative.

External force injuries are caused by forces that violate the tissues of the body. These injuries can result from gunshot wounds, stabbings, and/or projectiles. Injuries depend upon the anatomic area involved, the mass, and, most importantly, the velocity of the foreign object that enters the body. The kinetic energy (KE) of an object is the energy associated with that object in motion. It reflects the relationship between the weight (mass) of the object and the velocity at which it is traveling and is expressed mathematically as the following equation (m = mass, v = velocity):

Figure 10-1 A front-end collision should clue the CCTP to suspect potential head, neck, and chest injuries.

$$KE = \tfrac{1}{2}\, mv^2$$

These injuries require the CCTP to provide excellent patient assessment skills in order to prioritize injuries and treatments. The CCTP must be cautious not to develop tunnel vision. Tunnel vision occurs when people focus all of their attention on what is immediately in front of them, ignoring their peripheral vision **Figure 10-2**. As a result, the CCTP's focus would be on a visible injury, which may or may not be life threatening, while no attention is focused on other injuries that may well be life threatening. Maintaining a calm, professional demeanor and using a systematic approach allows the CCTP to maintain a high index of suspicion and attention to detail.

Trauma Assessment

Although the CCTP has had extensive training in patient assessment during initial training, these skills must now be applied to the advanced practice environment of critical care transport.

Trauma assessment, much like medical assessment, encompasses both a physical examination and a

subjective interview (when possible). For the CCTP, the subjective information often has to come from someone other than the patient. Therefore, it must always be evaluated critically and not taken as gospel. Even on an interfacility call, the sending diagnosis is subjective and should be evaluated by the CCTP. If information can be gained directly from the patient, it may help focus the physical exam. For example, a trauma patient with a chest injury might say, "I'm having trouble catching my breath." The CCTP's physical examination could then lead to a field diagnosis and subsequent treatment of a tension pneumothorax. It is especially important to remember that patients are not static, and that conditions may become evident as time progresses.

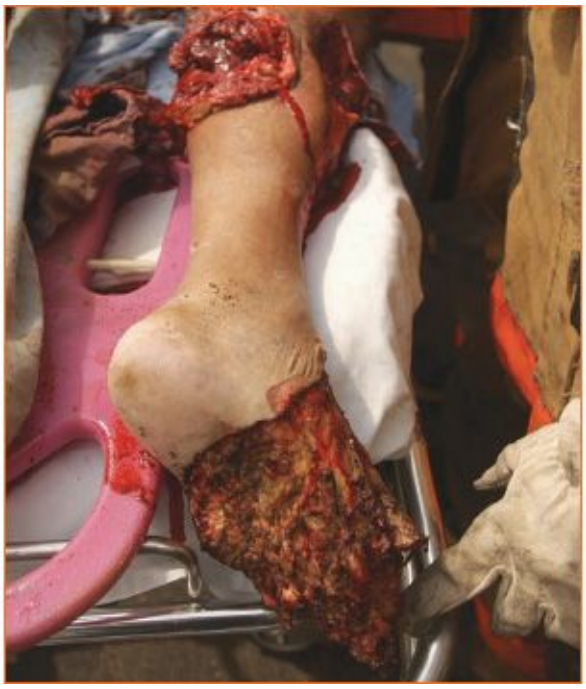


Figure 10-2 An overwhelming injury can cause a CCTP to focus on the visual horror, rather than more serious emergencies such as severe internal blood loss.

As always, the assessment process begins with scene size-up, then primary assessment. The level of consciousness and status of ABCs can be assessed simultaneously. The CCTP should undertake manual cervical spine immobilization while introducing himself or herself to the patient. If the patient responds, the CCTP can easily obtain vital information in regard to mental, airway, and breathing status. Any response that is less than alert should immediately direct the CCTP to perform a more detailed assessment of the ABCs and begin to search for causes.

Once the initial trauma primary assessment is accomplished, the CCTP uses these findings along with the mechanism of injury to dictate whether a rapid trauma or a focused assessment is most appropriate. Examine the trauma scene for evidence of high-energy trauma. If one of the following is present, consider transport to a trauma center:

- Ejection from an automobile
- Death in the same passenger compartment
- Pedestrian thrown or run over or auto-pedestrian injury at greater than 5 mph
- High-speed automobile crash (greater than 40 mph)
- Intrusion into the passenger compartment of greater than 12″
- Major vehicle deformity of greater than 20′
- Vehicle rollover with an unrestrained passenger

- Extrication time of greater than 20 minutes
- Falls of greater than 20′
- Motorcycle crash at greater than 20 mph or with separation of rider and bike

Trauma patients who have an isolated injury require a focused examination, whereas a rapid trauma assessment is used for multisystem trauma patients. A secondary assessment allows the CCTP to focus on the patient's main problem, such as an isolated injury.

A rapid trauma assessment allows the CCTP to rapidly assess the head, neck, chest, abdomen, pelvis, and extremities to find and correct any life threats and should be completed in under 1 minute. When conducting the rapid trauma assessment, the CCTP should not only do a visual assessment, but also a palpation assessment to look for life-threatening injuries.

The secondary assessment is a more comprehensive examination to determine additional injuries that may have been overlooked in the primary assessment. It is extremely important to document all findings in the secondary assessment because it also serves as a baseline for future treatment decisions in the continuity of care. Critical patients who fall into the "load and go" category should have the secondary assessment performed during transport, whereas noncritical patients may be assessed on the scene.

When conducting a secondary assessment, the questions to ask will vary depending on the patient's specific injury. The patient's answers will lead you to other questions.

The more comprehensive secondary assessment is generally done during transport to a trauma center. This assessment is done to develop a baseline for future treatment decisions. This exam should include:

1. **Head-to-toe exam**: Inspect, auscultate, palpate, and percuss.
2. **Reassess vital signs**: This includes pulse, respirations, blood pressure, and monitors such as cardiac, capnography, and pulse oximetry.

Once the primary and secondary assessments are completed, the CCTP must determine the criticality of the patient and make a transport decision. Patients who meet the appropriate critical criteria should immediately be loaded into the critical care transport unit for transport to the appropriate trauma center. The CCTP should alert the receiving facility as soon as possible so that specialized health care providers can be awaiting the patient upon his or her arrival.

Patients who cannot be stabilized by interventions available to the CCTP at the point of pickup need to be rapidly transported. Patients who are bleeding internally need blood and surgery immediately. Delay of transport of these patients for crystalloid IVs, for example, is not in their best interest. Issues of airway, breathing, and stopping external bleeding are the only reasons to delay transport, and it should only be delayed long enough to intervene with these problems.

All multisystem trauma patients should be transported rapidly, even those who do not need immediate surgery to stop internal bleeding and those not in need of immediate transfusion. The CCTP must be alert and maintain a high index of suspicion for deterioration with all trauma patients.

Reassessment is continued during transport to the trauma center. Essentially, it is an ongoing reassessment of the patient and any interventions performed. If at any time during reassessment the patient's condition deteriorates, the CCTP should immediately reassess the primary assessment and address any negative findings that can be corrected.

The CCTP must also be prepared for special patients, such as those who are morbidly obese, those who are anatomically different than normal, elderly patients, children, and those with comorbid factors that will be exacerbated with traumatic injury. Patients with special circumstances may also require a creative approach and application of critical thinking skills by the CCTP in order to meet their needs. It is necessary to be both proficient at advanced-level procedures and to have excellent assessment and decision-making skills. Deployment of an effective assessment technique is of paramount importance in

order to provide appropriate care and treatment for the trauma patient. Remember that most patient catastrophes occur or are started during hand-offs of patient care.

Triage

Triage is a system of prioritizing patients based on injury severity. More simply put, triage is doing the greatest good for the greatest number. There are four common triage categories: immediate (red), delayed (yellow), minimal (green), and expectant (black).

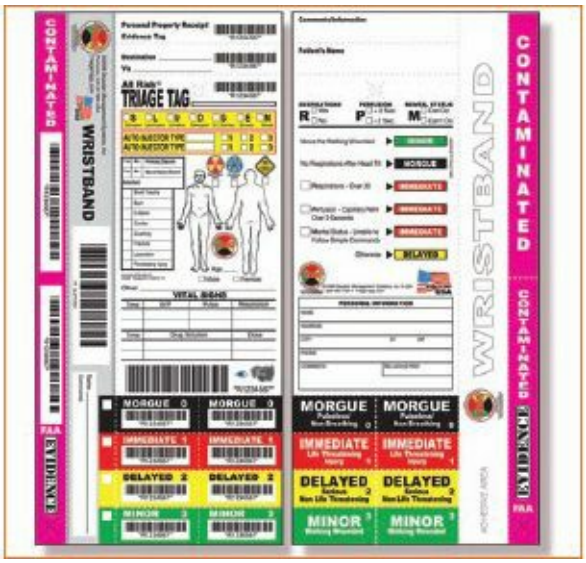


Figure 10-3 Standard triage tag.

Figure 10-3 shows a standard triage tag. Red tags denote the highest priority. Patients in this category need immediate care and transport to definitive treatment centers. Typical injures in this classification are injuries related to the ABCs, head trauma, and/or signs of inadequate tissue perfusion (shock).

Yellow tags are reserved for second- or delayed-priority patients. Patients in this category need treatment and transport, but it can be delayed. Typical injuries in this classification are serious but stable, such as orthopaedic or back injuries.

Green tags are the third priority and are often referred to as "walking wounded." Patients in this category have minor soft-tissue injuries such as contusions, abrasions, and lacerations.

Black tags are for patients for whom there is no treatment indicated: those who are dead, alive but nonsalvageable, or not injured. This may include cardiac arrest and obvious nonsurvivable injuries. The black triage category is also sometimes called expectant.

In order for triage to work, a systematic approach must be deployed that clearly defines individual roles and responsibilities of EMS providers. The triage officer must have ultimate control of the counting and prioritizing of patients. The treatment officer oversees the treatment area, the transportation officer oversees transport of patients, and the staging officer must oversee and direct the staging area in order to deploy needed resources to the appropriate treatment sectors. Individuals assigned to these positions do not take part in patient care, but rather direct the activities of their specific assignment.

START Triage

START triage is one of the easiest methods of triage. START is an acronym for simple triage and rapid treatment. The staff members at Hoag Memorial Hospital, Newport Beach, California, are responsible for developing this method of triage. It is easily mastered with practice and will give you the ability to rapidly categorize patients at a multiple casualty incident. START triage uses a limited assessment of the

patient's ability to walk, respiratory status, hemodynamic status, and neurologic status.

The first step of the START triage system is performed on arrival at the scene by calling out to the disaster site, "If you can hear my voice and are able to walk…" and then directing patients to an easily identifiable landmark. The injured persons in this group are the "walking wounded" and are considered minimal priority, or third-priority, patients.

The second step in the START process is directed toward nonwalking patients. You move to the first nonambulatory patient and assess the respiratory status. If the patient is not breathing, you should open the airway by using a simple manual maneuver. A patient who still does not begin to breathe is triaged as expectant (black). If the patient begins to breathe, tag him or her as immediate (red), place the patient in the recovery position, and move on to the next patient.

If the patient is breathing, a quick estimation of the respiratory rate should be made. A patient who is breathing faster than 30 breaths/min is triaged as an immediate priority (red). If the patient is breathing fewer than 30 breaths/min, move to the next step of the assessment.

The next step is to assess the hemodynamic status of the patient by checking for a radial pulse. An absent radial pulse implies the patient is hypotensive and should be triaged as an immediate priority. If the radial pulse is present, go to the next assessment.

The final assessment in START triage is to assess the patient's neurologic status, which simply means to assess the patient's ability to follow simple commands, such as "show me three fingers." This assessment establishes that the patient can understand and follow commands. A patient who is unconscious or cannot follow simple commands is an immediate-priority patient. A patient who complies with a simple command should be triaged in the delayed category. The START system is outlined in **Figure 10-4**.

JumpSTART Triage for Pediatric Patients

Lou Romig, MD, recognized that the START triage system does not take into account the physiologic and developmental differences of pediatric patients. She developed the **JumpSTART triage** system for pediatric patients. JumpSTART is intended for use in children younger than 8 years or who appear to weigh less than 100 lb. As in START, the JumpSTART system begins by identifying the walking wounded. Infants or children who are not developed enough to walk or follow commands (including children with special needs) should be taken as soon as possible to the treatment sector for immediate secondary triage. This action assists in getting children who cannot take care of their own basic needs into the care of someone who can. There are several differences within the respiratory status assessment compared with that in START triage. First, if you find that a pediatric patient is not breathing, immediately check the pulse. If there is no pulse, label the patient as expectant (black). If the patient is not breathing but has a pulse, open the airway with a manual maneuver. If the patient does not begin to breathe, give five rescue breaths and check respirations again. A child who does not begin to breathe should be labeled expectant (black). The primary reason for this difference is that the most common cause of cardiac arrest in children is respiratory arrest.

The next step of the JumpSTART process is to assess the approximate rate of respirations. A patient who is breathing less than 15 breaths/min or more than 45 breaths/min is tagged as immediate priority, and you move on to the next patient. If the respirations are within the range of 15 to 45 breaths/min, the patient is assessed further.

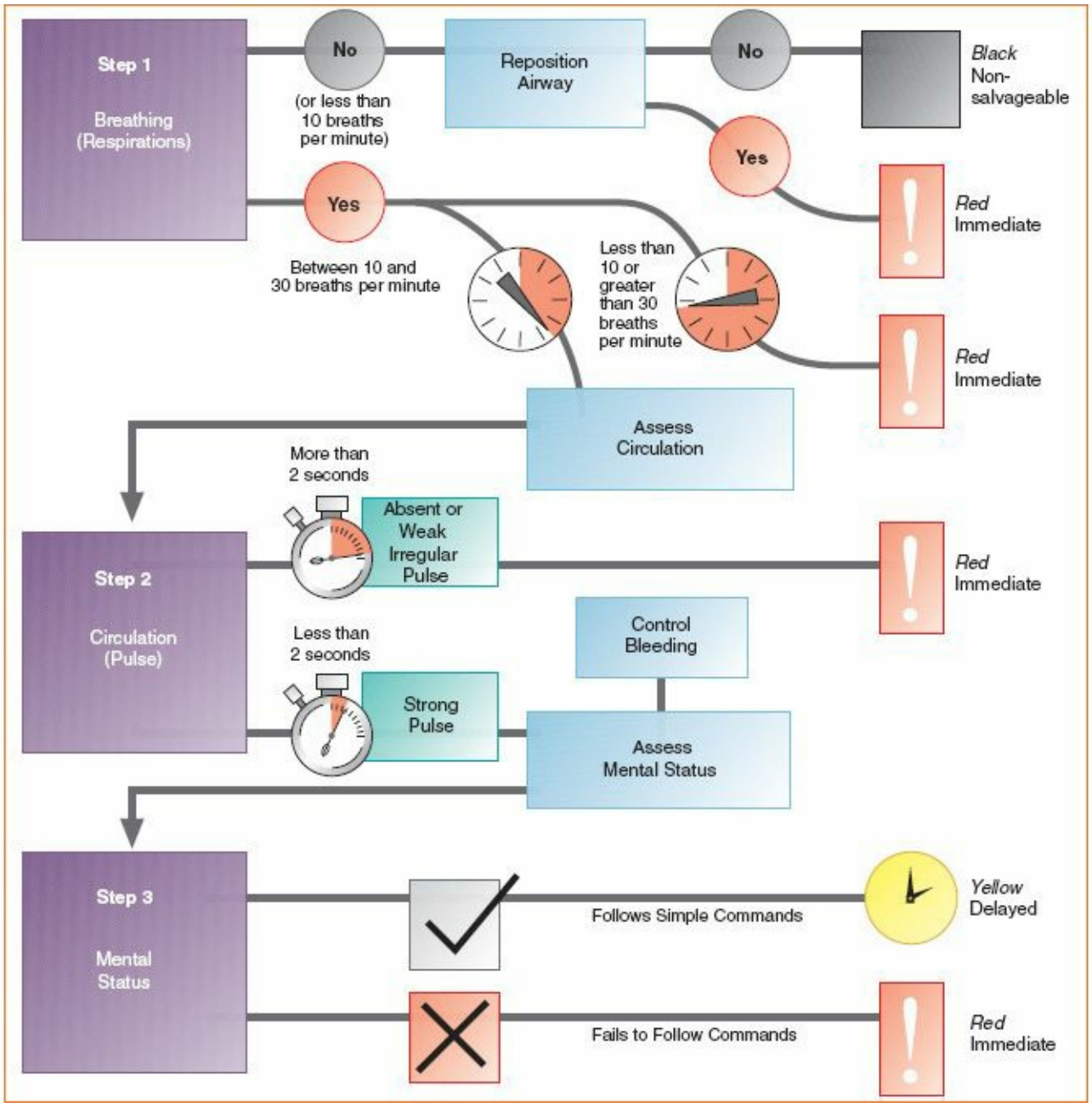


Figure 10-4 The START algorithm.

The next assessment in JumpSTART triage evaluates the hemodynamic status of the patient. As in START, you are checking for a distal pulse. This does not need to be the brachial pulse; assess the pulse that you are comfortable checking. If there is an absence of a distal pulse, label the child as an immediate priority and move to the next patient. If the child has a distal pulse, move on to the next assessment.

The final assessment is neurologic status. Responses vary in children because of their developmental differences. For JumpSTART, a modified AVPU (A, alert; V, responding to voice; P, responding to pain; U, unconscious) score is used. A child who is unresponsive or responds to pain by posturing or with incomprehensible sounds or is unable to localize pain is considered an immediate priority and tagged as such. A child who responds to pain by localizing it or withdrawing from it or is alert is considered a delayed priority patient. The JumpSTART system is described in **Figure 10-5**.

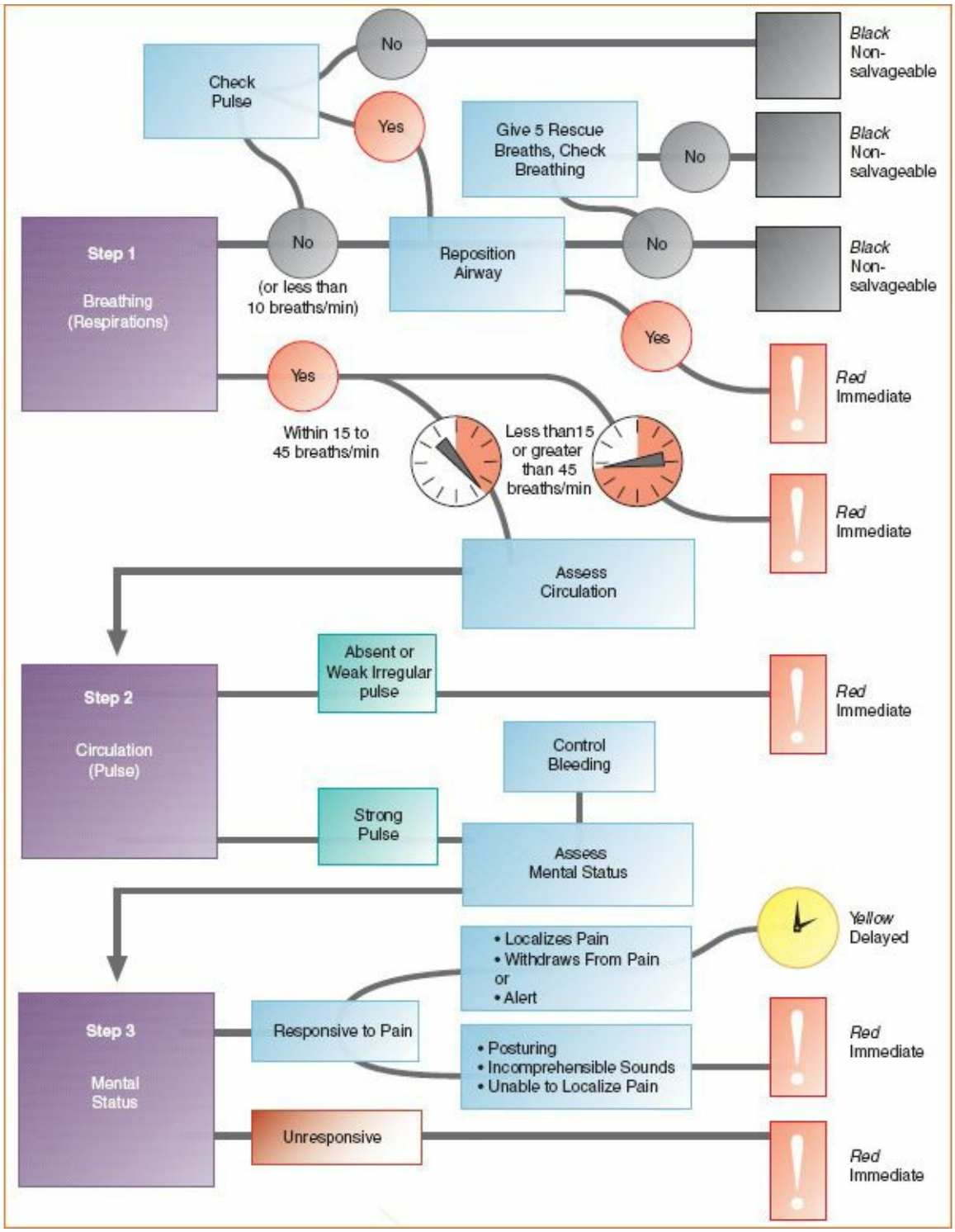


Figure 10-5 The JumpSTART algorithm.

Trauma Scoring Systems

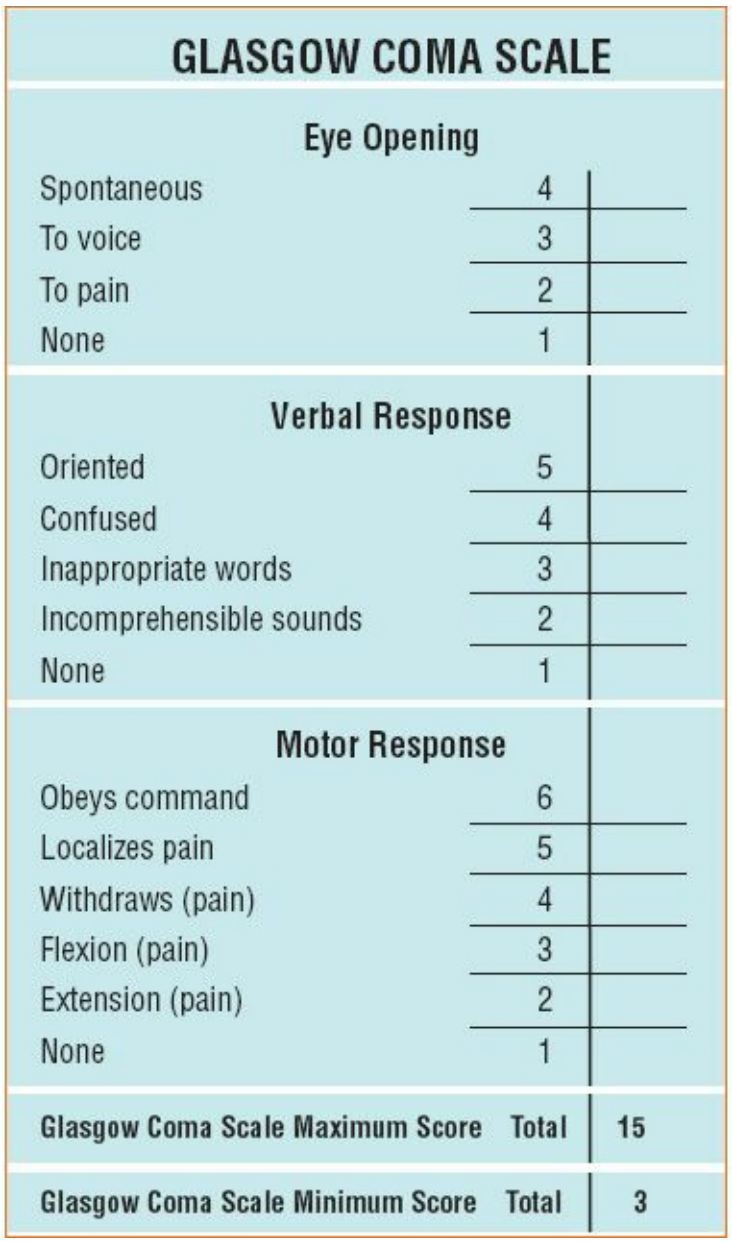
Several trauma scoring systems are used in the critical care transport environment. These provide assessment information and indicators of patient survivability. Trauma scoring systems are an important tool for regional trauma centers that use them in conjunction with their quality assurance program.

Glasgow Coma Scale

The **Glasgow Coma Scale (GCS)** is a tool that many health care professionals use to assess the neurologic function of their patients. This scoring system is used to determine a patient's level of consciousness by measuring and assigning point values, or scores, for eye opening, verbal response, and

motor response. The highest score is 15; the lowest score is 3. The GCS chart in **Figure 10-6** shows how the numerical values are determined.

Although the GCS score is often used when caring for trauma patients, it is also a great assessment tool for patients with medical conditions. Medical emergencies, such as a drug overdose, metabolic disorders, and hypercapnia, benefit from frequent patient evaluation using the GCS. Ongoing assessments assist CCTPs in rapidly identifying treatable conditions.



GLASGOW COMA SCALE

Eye Opening		
Spontaneous	4	
To voice	3	
To pain	2	
None	1	
Verbal Response		
Oriented	5	
Confused	4	
Inappropriate words	3	
Incomprehensible sounds	2	
None	1	
Motor Response		
Obeys command	6	
Localizes pain	5	
Withdraws (pain)	4	
Flexion (pain)	3	
Extension (pain)	2	
None	1	
Glasgow Coma Scale Maximum Score	**Total**	**15**
Glasgow Coma Scale Minimum Score	**Total**	**3**

Figure 10-6 Glasgow Coma Scale scores are used to assess a patient's level of consciousness, for example, in patients with head injuries. The lower the score, the more severe the extent of brain injury.

Special Populations

It is just as important to obtain an accurate neurologic exam in the pediatric population as in the adult population, although doing so may be more difficult with pediatric patients. Depending on the age of the child, modifications may need to be made in order to accurately identify any neurologic deficits.

A poor GCS score usually indicates that the patient requires some type of immediate intervention, such as airway control or aggressive resuscitation. Chapter 23 discusses the pediatric GCS and its application in the management of children.

Patients who present with GCS scores of less than 8 require intubation or another form of advanced airway intervention because they are experiencing significant neurologic impairment. Remember, the GCS score is one of the most accurate means of objectively assessing the central nervous system when invasive monitoring devices are not available. Even in an intubated patient, the GCS score provides accurate, useful information; the CCTP merely needs to note when a patient's inability to verbalize is the result of the device in his or her airway, rather than a decrease in neurologic function.

Always give detailed explanations of abnormal GCS findings in the narrative section of your patient care record. In many situations, successful management of the unresponsive patient with a head injury depends on ongoing examinations. Neurologic changes can occur in a matter of minutes, which necessitates obtaining a GCS score as often as every 5 minutes. Ongoing GCS assessment scores must always be compared to the baseline score. It is important to look for changes that signal deterioration as well as improvement. Any changes must be reported to the receiving facility.

Trauma Score

The **trauma score** is a predictor of the likelihood of patient survival. The score ranges from 1 to 16, with 16 being the best possible score, and takes into account the GCS score, respiratory rate, respiratory expansion, systolic blood pressure, and capillary refill. The trauma score, however, does not accurately predict survivability in patients with severe head injuries. Therefore, the revised trauma score, discussed next, replaced the trauma score.

Special Populations

The pediatric trauma score takes into account the child's weight, systolic blood pressure, airway status, central nervous system status, whether or not he or she has an open wound, and whether there is musculoskeletal trauma. Chapter 23 discusses the pediatric trauma score and its application in the management of children.

Revised Trauma Score

The numeric scoring of trauma patients for determining the severity of their injury is common practice. There are several different trauma scoring systems, but the one that is the most commonly used is the **revised trauma score**.

The revised trauma score is a system that measures the following three physiologic parameters: respiratory rate, systolic blood pressure, and GCS score **Table 10-1**. The total score will range from 0 to 13, with the more debilitated patient receiving a lower score. The score can also be weighted, giving the three physiologic parameters different weights; the result then ranges from 1.0 to 7.8408. When the scores are weighted, the GCS score is given more weight to account for a patient's compensatory mechanisms that could prevent vital signs from accurately reflecting patient condition. The weighted score is not practical for use in the field, but is a useful research tool when studying mortality among trauma patients. In the field of trauma, it is important to understand that much time and effort is spent researching ways to decrease morbidity and mortality in trauma patients. Extensive research has proven that there is a correlation between a low revised trauma score and a high mortality rate. This simply states and proves the effectiveness of the various trauma scores **Figure 10-7**.

The revised trauma score does not readily identify the small percentage of severely injured trauma patients whose vital signs, because of compensatory mechanisms, do not accurately represent patient condition. For example, a patient with severe trauma who is in the early stages of shock may not pre-sent

with abnormal vital signs (eg, low systolic blood pressure and increased respiratory rate) because the body is compensating for the blood loss. The body's ability to compensate for this blood loss may only last for a brief period. When the patient begins to decompensate, the vital signs will begin to deteriorate. Aggressive resuscitation should begin, however, before the patient's vital signs deteriorate. Regardless of the score, the CCTP needs to be prepared for each trauma patient's condition to deteriorate with time, once the body's ability to compensate for the injury ends. For patients with a revised trauma score of less than 11, the American College of Surgeons recommends transfer to a Level I or II trauma center. A high index of suspicion should be maintained in all trauma patients.

Abbreviated Injury Scale

Developed in 1969, the **Abbreviated Injury Scale (AIS)** is an anatomic scoring system that is designed to provide a reasonably accurate means of ranking the severity of injury. This scale categorizes injuries into six body regions and assigns an individual score to each injury. The body regions include the head, neck, thorax, abdomen, spine, and extremities. Injuries are ranked on a scale of 1 to 6, with 1 being a minor injury and 6 being an injury that has the highest risk of mortality. The following list indicates the categories and the corresponding AIS scores:

- Minor injury: 1
- Moderate injury: 2
- Serious injury: 3
- Severe injury: 4
- Critical condition: 5
- Unsurvivable: 6

Modified from *Survival Probability* by Revised Trauma Score. © Trauma.org.

The AIS allows for the determination of the severity of an individual injury but does not take into account multisystem injuries.

TABLE 10-1 Components of the Revised Trauma Score

Revised Trauma Score	Components
4	GCS: 13–15 Systolic blood pressure: > 89 mm Hg Respiratory rate: 10–29 breaths/min
3	GCS: 9–12 Systolic blood pressure: 76–89 mm Hg Respiratory rate: > 29 breaths/min
2	GCS: 6–8 Systolic blood pressure: 50–75 mm Hg Respiratory rate: 6–9 breaths/min
1	GCS: 4–5 Systolic blood pressure: 1–49 mm Hg Respiratory rate: 1–5 breaths/min
	GCS: 3

0	Systolic blood pressure: 0 mm Hg Respiratory rate: 0 breaths/min

Abbreviation: GCS, Glasgow Coma Scale.
Reproduced from Revised Trauma Score. © Trauma.org

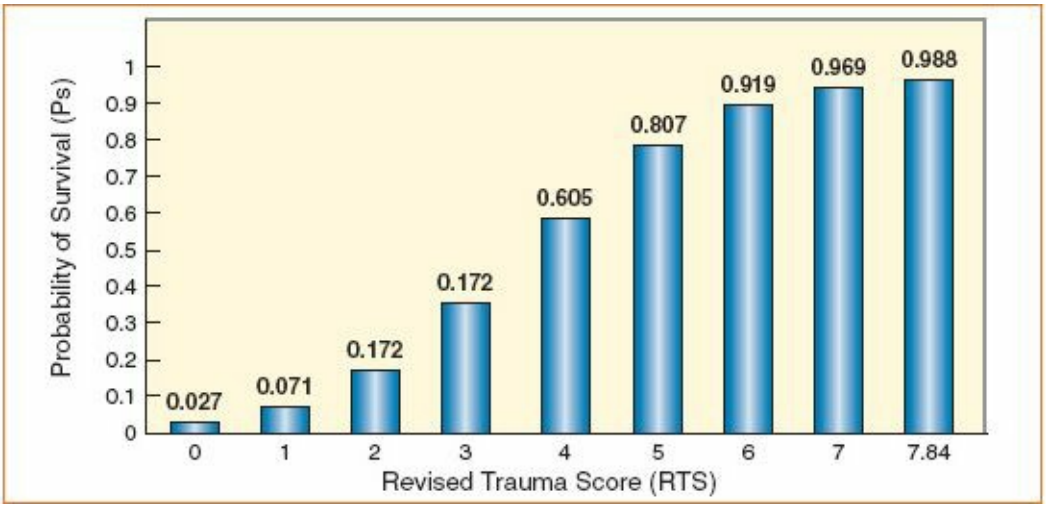


Figure 10-7 Survival probability by revised trauma score. Modified from *Survival Probability* by Revised Trauma Score. © Trauma.org.

TABLE 10-2 Sample ISS Calculation

ISS Region	Injury	AIS	Square of the Top Three AIS Scores*
Head and neck	No injury	0	
Face	Unstable facial fracture	4	16
Chest	Flail chest	5	25
Abdomen	Liver laceration	4	16
Extremity	No injury	0	
External	Abrasion to right upper quadrant of abdomen	3	

Abbreviations: AIS, Abbreviated Injury Score; ISS, Injury Severity Score.
*The ISS in this example is 57.
Reproduced from Injury Severity Score. © Trauma.org

Injury Severity Score

The **Injury Severity Score (ISS)** is an anatomic scoring system that provides an overall score for patients with multiple injuries. The ISS quantifies multisystem injuries with the use of AIS scores. The ISS is determined by adding the highest AIS scores squared in the three most severely injured body systems. The ISS is a number between 1 and 75, with 1 being a minor injury and 75 being an injury with a high mortality rate. Any ISS body region that is assigned an AIS of 6 (unsurvivable) equates to an ISS score of 75. An example of the ISS calculation is shown in **Table 10-2**.

Throughout the trauma system community, a patient with an ISS greater than 15 is frequently considered a major trauma patient who requires immediate attention and in most cases transfer to a Level I facility.

The ISS determined by health care providers is often used by trauma registry personnel for data

collection and research purposes. Once data such as ISS scores are collected, some variances can occur with this scoring system. For example, any error that is calculated with AIS scoring will increase the likelihood of errors with the ISS. Remember that you must always refer back to your triage skills in the field for determining the destination of your patient. The various trauma scoring systems are not as useful as your triage skills because a full description of the patient injuries is not known.

Trauma Injury Severity Score

The **trauma injury severity score** is another scoring system that calculates the survival probability of the critically ill or injured patient. This scoring system uses the results of the ISS and the revised trauma score along with the patient's age, which incorporate the patient's physiologic and anatomic indicators. This score is rarely used in the transport setting.

Trauma Systems

Levels of Trauma Care

The CCTP is often summoned to accident scenes and/or outlying facilities to transport critically ill trauma patients to more definitive care. For this reason, it is important to be familiar with how the American College of Surgeons' Committee on Trauma classifies trauma care. Trauma centers are classified into Levels I through IV, with Level I having the most resources followed by Levels II, III, and IV, respectively.

A Level I facility is a regional resource center and generally serves large cities or heavily populated areas. Level I facilities must be capable of providing every aspect of trauma care from prevention through rehabilitation; therefore, the facility must have adequate personnel and resources. Because of the extensive requirements, most Level I facilities are university-based teaching hospitals.

A Level II facility is typically located in less densely populated areas. Level II centers are expected to provide initial definitive care, regardless of injury severity. These facilities can be academic institutions or public/private community facilities. Because of location and resources, the Level II trauma center may not be able to provide the same comprehensive care as a Level I trauma center.

Level III facilities serve communities that do not have access to Level I or II facilities. Level III facilities provide assessment, resuscitation, emergency care, and stabilization. Level III facilities must have transfer agreements with a Level I or II trauma center, and must have protocols in place to transfer patients whose needs exceed the facility's resources.

Level IV facilities are typically found in remote, outlying areas where no higher level of care is available. These facilities provide advanced trauma life support prior to transfer. Such a facility may be a clinic or urgent care facility, with or without a physician.

Although an inclusive trauma system should leave no facility without a direct link to a Level I or II facility, all facilities are expected to provide the same high quality of initial care regardless of the classification level.

Trauma centers are categorized as either adult trauma centers or pediatric trauma centers, not necessarily both. Pediatric trauma centers are not nearly as common as adult trauma centers. When transporting a pediatric trauma patient, you must be certain to transport to the closest appropriate facility —a pediatric trauma center if that is closest.

The American College of Surgeons' Committee on Trauma also provides criteria for Level I trauma patient classification. When one or more of the following criteria listed below are present in the trauma patient, he or she is classified as a Level I trauma patient.

- Confirmed blood pressure less than 90 mm Hg at any time in adults, and age-specific hypotension

in children

- Respiratory compromise, obstruction, and/or intubation (with the exception that a patient with an unmanageable airway should go to the closest hospital)
- Transfer patients from other hospitals receiving blood to maintain vital signs
- Emergency physician's discretion

TABLE 10-3 American College of Surgeons Recommendations for a Level II Patient

Patient characteristic/condition indicators	1. Glasgow Coma Scale score of < 14 when associated with trauma 2. Respiratory rate of < 10 or > 29 breaths/min (< 20 breaths/min in an infant younger than 1 year) when associated with trauma 3. Penetrating wounds (other than gunshot wounds) to the head, neck, torso, and extremities proximal to the elbow and knee 4. Flail chest 5. Combination of trauma with burns 6. Two or more proximal long bone fractures 7. Pelvic fractures 8. Limb paralysis and/or spinal cord injury 9. Amputation proximal to the wrist and/or ankle
Mechanism of injury indicators	1. High-speed automobile crash • Initial speed > 40 mph • Major automobile deformity • Intrusion into the passenger compartment 2. Ejection from the automobile 3. Death in same passenger compartment 4. Extrication time > 20 minutes 5. Falls > 20′ or significant falls in children or elderly 6. Automobile rollover 7. Automobile vs pedestrian or automobile vs bicycle impact > 5 mph 8. All-terrain vehicle or motorcycle crash > 20 mph or separation of rider from vehicle
Consider Level II classification with these preexisting conditions	1. Age younger than 5 years or older than 55 years 2. Cardiac or respiratory disease 3. Insulin-dependent diabetes mellitus, cirrhosis of the liver, or morbid obesity 4. Pregnancy 5. Immunosuppressed patients 6. Patients with a bleeding disorder or receiving anticoagulants

- GCS score of 8 or less, with mechanism attributed to trauma
- Gunshot wound to the abdomen, neck, or chest

Although the American College of Surgeons does not cite required criteria for a Level II patient, they do provide recommendations, which are listed in **Table 10-3**.

General Trauma Management

Paramedics are already well schooled in prehospital management of trauma. Interfacility management of the trauma patient will often involve patients who are very ill and in need of close assessment and continued monitoring. Some patients may undergo damage-control surgery prior to transfer. This treatment involves surgical correction of life-threatening injuries followed by transfer to a critical care unit or tertiary hospital for correction of acidosis, hypothermia, or coagulopathies. Definitive surgery is performed after the patient's condition has been stabilized. In some patients, injuries may be managed without surgery. Regardless of the approach, management following arrival at a hospital follows Advanced Trauma Life Support guidelines. As in the prehospital setting, immediate attention is directed at the ABCs.

Immediately assess the placement of the endotracheal tube, presence of breath sounds, and capnography waveform. If any doubt exists as to proper placement of the endotracheal tube, perform direct laryngoscopy to confirm tube location. Cardiovascular status should be assessed, including the patency of all vascular access devices and the total volume of fluids infused since the patient presented at the receiving facility. A thorough neurologic exam should be performed and documented. Although it may seem duplicative, it is imperative to assess the patient from head to toe for external injuries. Previously unseen injuries can prove fatal during interfacility transport if they are not discovered and addressed prior to departure.

Elicit as much medical history as possible from the patient or hospital staff. Consider drug or alcohol intoxication; up to one third of all trauma patients are drug or alcohol dependent. Repeated head-to-toe assessments decrease the possibility of missed injuries, which occur more often in multisystem trauma patients. The CCTP must also be prepared to detect and treat complications on initial evaluation and treatment, including fluid overload, transfusion reactions, contrast-induced renal issues, abdominal compartment syndrome, bleeding, and shock.

Hypothermia in Trauma

Hypothermia is still a commonly encountered problem that the CCTP must address. Hypothermia in trauma can be attributed to environment, the injury itself, or the treatment provided. The CCTP must address hypothermia by attempting to reverse it, or prevent the onset.

Multiple studies have shown that the incidence of hypothermia in trauma can be as high as 50%. These studies also indicate that hypothermia in trauma can result in mortality rates that approach 100%. Although hypothermia does benefit select patients, it has a profound detrimental effect on trauma patients **Table 10-4**. Coagulopathy is the most profound effect of hypothermia.

Hypothermia in trauma can result from the patient being exposed to the environment, such as cold weather during a prolonged extrication. Even on a warm day, blood loss and thermoregulation loss in trauma can result in hypothermia. Hypothermia can also be induced or exacerbated by the CCTP by exposing the patient for an exam, failing to increase ambient temperature in patient care areas, and infusing fluids.

Nonwarmed infusions require the body to warm up the fluid internally, which can result in further

decreases in core temperature, expend needed energy, and stress thermoregulation mechanisms.

TABLE 10-4 Effects of Hypothermia on Trauma Patients

Impaired cardiorespiratory function
Cardiac depression Myocardial ischemia Arrhythmias Peripheral vasoconstriction Impaired tissue oxygen delivery Elevated oxygen consumption during rewarming Blunted response to catecholamines Increased blood viscosity Metabolic acidosis
Bleeding diathesis
Decreased kinetics of coagulation factors Reduced platelet function
Reduced clearance of drugs
Decreased hepatic blood flow Decreased hepatic metabolism Decreased renal blood flow
Increased risk of infection
Decreased white blood cell number and function Impaired cellular immune response Wound infection • Thermoregulatory vasoconstriction • Decreased subcutaneous oxygen tension • Impaired oxidative killing by neutrophils • Decreased collagen deposition Pneumonia Sepsis
Insulin resistance with hyperglycemia
Adapted from: Smith CE. Prevention and treatment of hypothermia in trauma patients. TraumaCare International ITACCS. Available at: http://www.itaccs.com/traumacare/archive/04_01_Spring_2004/prevention.pdf. Accessed April 23, 2009.

Treatment should focus on prevention or reduction in present hypothermia. Limit exposure of the patient as much as possible. Increase ambient temperature by using warm blankets and increasing patient compartment temperatures. This increase in temperature may be needed even on a warm day. Use of only warmed fluids is also indicated. Oxygen may be warmed and humidified as well if possible. The CCTP

should also be cognizant of the multiple other side effects of hypothermia, such reduced drug clearance. The trauma center will also employ multiple invasive and noninvasive techniques.

Diagnostic Imaging for Trauma

Obtaining and understanding imaging studies related to trauma is an integral component of the trauma care paradigm for the CCTP. The use of diagnostic imaging aids helps manage and direct further care for the trauma patient. Diagnostic imaging also confirms previous care that has been delivered, such as endotracheal tube placement and invasive catheters.

Types of diagnostic imaging were introduced in Chapter 8. There have been many exciting advances and new procedures in diagnostic imaging. We will briefly cover those that are related to the practice of the CCTP in trauma care.

Standard Radiographs

Standard radiographs, used in a stationary or portable environment, provide a beneficial starting point in most paradigms of trauma care. As mentioned in Chapter 8, standard radiographs can provide confirmation of endotracheal tube placement, cervical fractures, chest injuries, and most major bone fractures. Examples of normal and abnormal radiographs are shown in **Figure 10-8** and **Figure 10-9**.

Plain radiographs are limited in their use by access to the area to be imaged, such as obtaining all seven cervical vertebrae, and are not typically useful for advanced imaging.

Typical interpretation involves:

- Structure and landmark verification
- Proper size and placement of anatomic structure or devices Symmetry of structure
- Foreign object identification

Computed Tomography

Computed tomography is another imaging study that is of essential use for the trauma patient. For trauma patients, it is typically used for head injury identification **Figure 10-10**, identification of bleeding, and complex fractures. Typical studies will scan the head, c-spine, chest, and abdominal-pelvic regions. Interpretation is similar to that of plain radiographs because the contrast of different structures still applies.

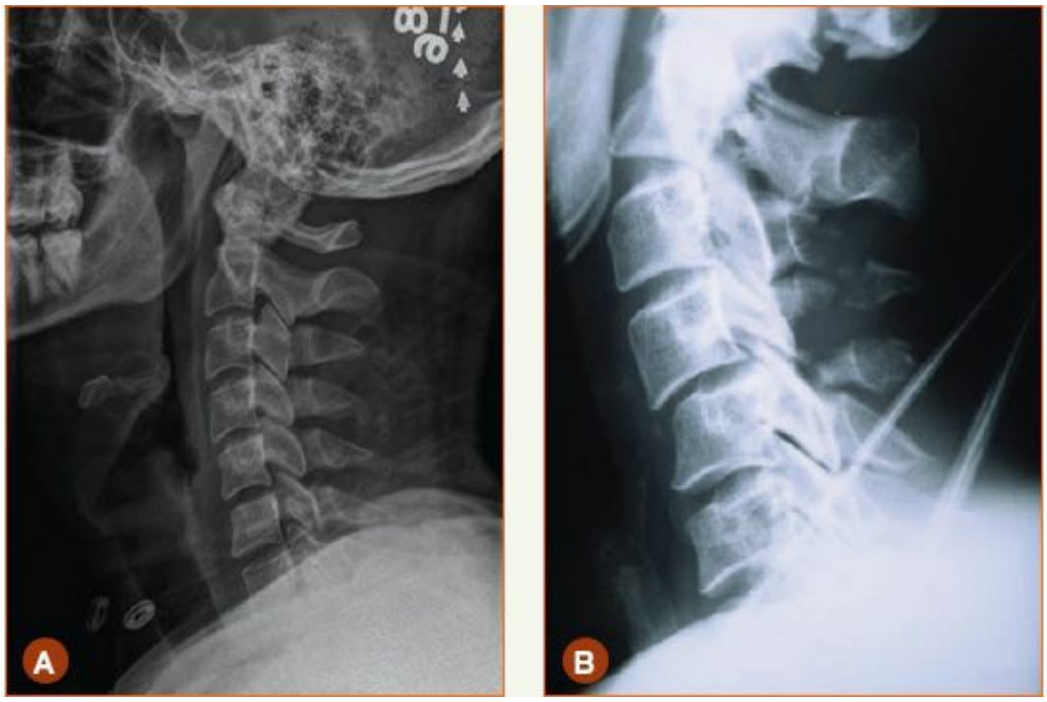


Figure 10-8 **A.** Normal cervical radiograph. **B.** Abnormal cervical radiograph.

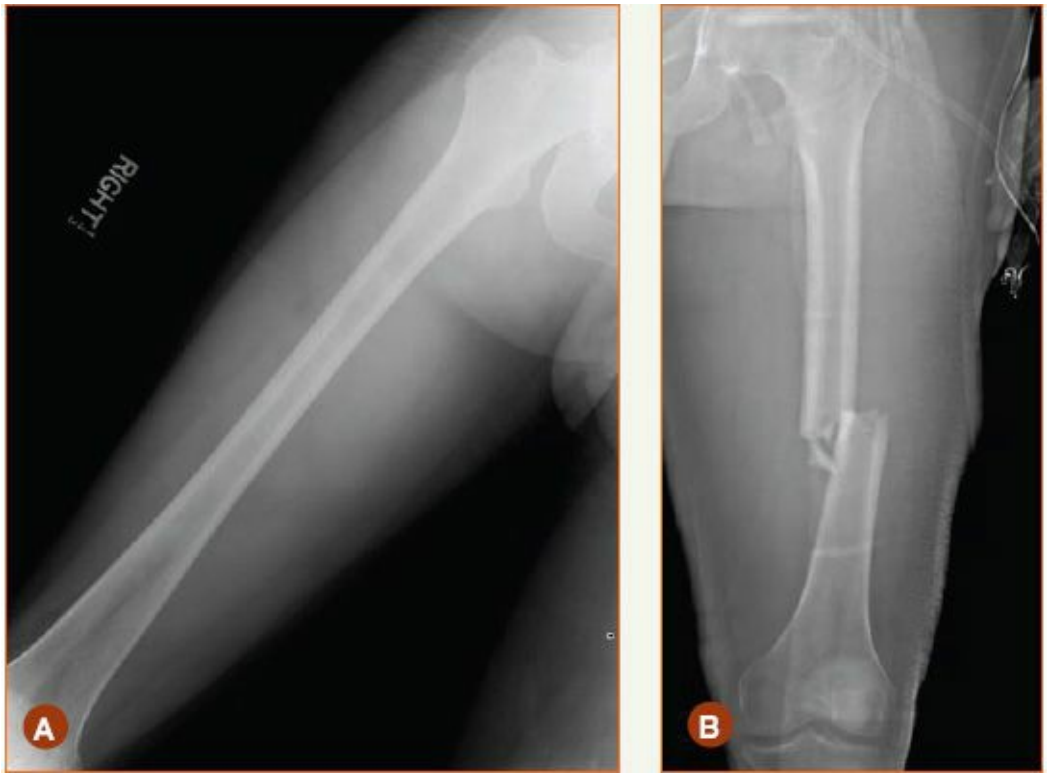


Figure 10-9 **A.** Normal long-bone radiograph. **B.** Abnormal long-bone radiograph.

Magnetic Resonance Imaging

Once again, the contrast differences of magnetic resonance imaging (MRI) are used for identification of abnormalities in trauma **Figure 10-11**. MRI, however, may be of limited use in the major trauma patient as a result of availability and the time required to obtain images.

FAST Ultrasonography

One of the recent advances in trauma care is the **FAST** ultrasound. FAST is an acronym for Focused

Assessment with Sonography for Trauma. FAST is an ultrasound exam directed at identifying the presence of free intraperitoneal or pericardial fluids. It is typically performed in the emergency department or trauma room by trained staff. It has replaced diagnostic peritoneal lavage (DPL) and has decreased the need for laparotomy, or the exploration of the abdomen for blood by incision. However, if the FAST ultrasound is equivocal, then diagnostic peritoneal lavage or laparotomy may be performed. FAST has been adopted by some transport services as well as for use in the prehospital setting, using smaller and portable equipment **Figure 10-12**. It is showing much promise in the early identification of abdominal bleeding in trauma. Studies have also shown that the E-FAST or extended FAST exam can reliably identify chest injuries such as pneumothorax and hemothorax.

The FAST is performed by imaging four distinct areas of the abdomen. Training standards to perform this procedure vary, but at a minimum usually require some credentialing and experience.

Intra-abdominal Pressure Monitoring

Intra-abdominal pressure (IAP) is the static pressure inside the abdominal compartment. Normally, IAP is approximately 5 mm Hg with variations caused by respirations. Nonpathologic increases in IAP can occur with obesity. Recent studies of IAP in critically ill medical and surgical patients have found elevated pressures to be relatively common. The deleterious effects of intra-abdominal hypertension (IAH) can lead to **abdominal compartment syndrome (ACS)** and death if untreated. ACS is thought to play a significant role in end-organ damage and multisystem organ failure during critical illness.

IAP can be measured with a variety of commercial devices or with user-assembled equipment. The current reference standard uses a transducer connected to an indwelling urinary drainage catheter located within the bladder. IAP is expressed in mm Hg and measured at end expiration with the patient supine and the pressure transducer at the midaxillary line. The recommended technique uses a pressure transducer connected by tubing at or near the aspiration port of the urinary catheter drainage tubing. The following steps are performed.

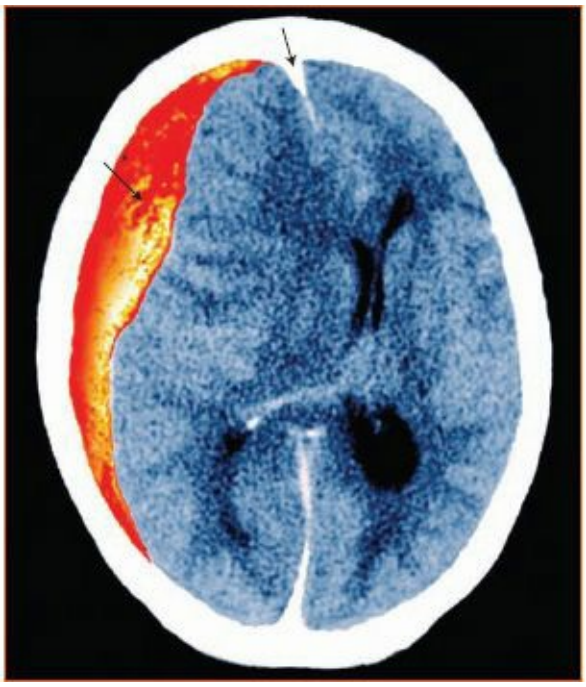


Figure 10-10 A computed tomography scan showing a head bleed with a midline shift.

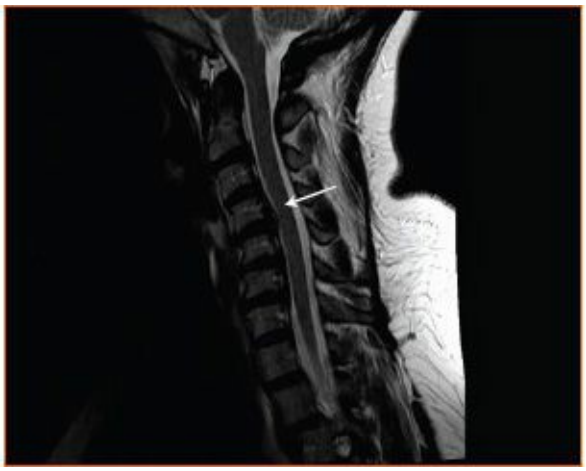


Figure 10-11 Magnetic resonance image showing abnormalities of the spinal cord.

Figure 10-12 A portable FAST (Focused Assessment with Sonography for Trauma) unit.

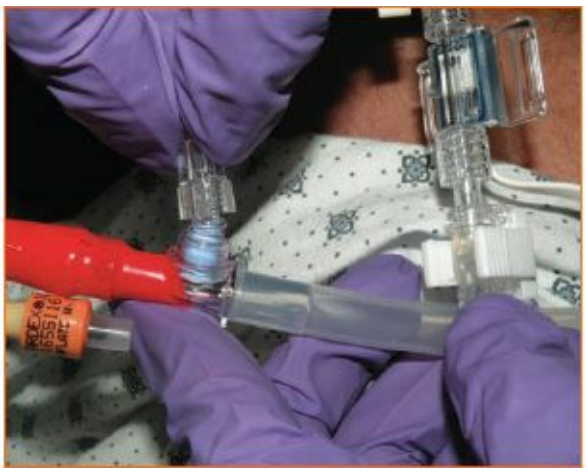


Figure 10-13 The CCTP may be asked to obtain intra-abdominal pressure measurements during transport.

1. Clamp the tubing distal to the connection.

2. Instill 50 mL or less of sterile room temperature saline through the pressure measurement system into the bladder.
3. Record the measured pressure after a 30- to 60-second stabilization period **Figure 10-13**.

IAH is defined as a sustained IAP of greater than 12 mm Hg. ACS is defined as a sustained IAP of greater than 20 mm Hg and is often associated with new-onset single- or multiorgan system failure.

There are multiple causes of IAH and not all are associated with trauma or surgical procedures to the abdomen. ACS is classified into three types:

1. Primary: Resulting from surgical interventions or injuries to the abdominal-pelvic region. Examples include damage-control exploratory laparotomy, massive retroperitoneal hematomas, liver transplantation, and failed nonoperative management of injury or disease.
2. Secondary: Develops as a result of conditions outside the abdomen, such as sepsis, burns, and capillary leakage resulting from massive fluid resuscitation.
3. Recurrent: Recurrence of ACS following initially successful surgical or medical treatment of primary or secondary ACS.

Although definitive treatment for acute ACS is surgical decompression (often with the abdomen left open until swelling is decreased), prevention through careful monitoring and targeted medical management is highly encouraged. Interventions to reduce the risk of IAH and/or to prevent IAH from progressing to ACS include neuromuscular blockade, drainage of free fluid in the abdominal cavity (paracentesis), decompression of the gastrointestinal tract using gastric tubes, prokinetic drugs (to stimulate intestinal motility), and close attention to prevention of fluid volume overload.

The CCTP may observe IAP monitoring equipment being used in the ICU or emergency department (ED) setting and may be asked to obtain IAP measurements during transport. Familiarity with the equipment in use is necessary to accurately obtain measurements.

Transport Considerations

With the rapid care of a trauma patient, not all available studies may have been performed. The CCTP should have and maintain some proficiency in interpreting imaging for obvious abnormalities. Reviewing imaging that has been performed serves to limit liability, confirm suspicions in diagnostics, and document accurate placement or performance of certain procedures. Various methods of training are available to the CCTP, and most services provide internal training as well.

Even with technology and the electronic transmission of imaging, it is still important to always ask for and ensure that all copies of the imaging studies are with patient records. Some may be as small as a disk, so care is needed not to misplace or lose them.

Thoracic Trauma

The Centers for Disease Control and Prevention reports that thoracic trauma injuries cause more than 700,000 ED visits and more than 18,000 deaths per year in the United States. Of all trauma deaths, one in four (25%) are directly associated with thoracic injuries. Given these alarming statistics, it is imperative for the CCTP to have a complex understanding of the anatomy and physiology of the thoracic cavity. However, only 15% to 20% of these potentially catastrophic injuries require open chest surgery. The majority can be managed with relatively simple interventions within the scope of the CCTP.

The thorax is essentially a bony cage consisting of 12 pairs of ribs that join anteriorly with the sternum and posteriorly with the thoracic spine **Figure 10-14**.

The chest consists of two thoracic cavities, each containing one lung. The mediastinum, which contains the heart, superior and inferior vena cava, aorta, bronchi, trachea, and esophagus, is located between the two cavities. The diaphragm inserts into the thoracic cage below the fifth rib and separates the chest and abdominal cavities. The primary function of the thorax is to allow for adequate oxygenation and circulation. Therefore, injuries to the chest must be promptly found and managed. Injuries to the thoracic region can result from either blunt or penetrating injuries; therefore, it is important for the CCTP to conduct a thorough assessment in order to avoid missing life-threatening injuries. The chest is normally a closed box with a single opening; the changes in pressure necessary for ventilation are dependent on the ribs, diaphragm, and pleural membrane all being intact and working properly. Half of all chest injuries involve the chest wall. Recent research has emphasized the importance of this relationship and normal interthoracic pressures for adequate circulation and ventilation. Especially in hypotension, excessive interthoracic pressure can interfere with blood returning to the heart. Therefore, care must be taken in trauma patients to ensure adequate positive pressure ventilations without allowing interthoracic pressure to rise too high. This may also become an issue during flight. The atmospheric pressure declines as the aircraft goes higher and gas volume expands in a closed space. This may make a small tension pneumothorax larger, and less gas will be needed to ventilate a patient using a gas-powered ventilator.

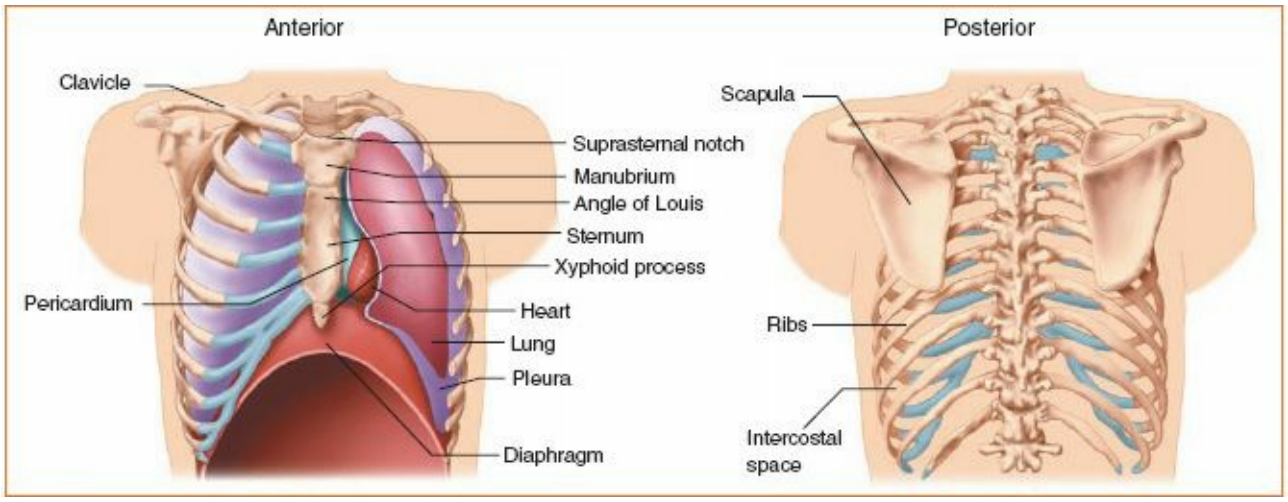


Figure 10-14 The thorax, front and back views.

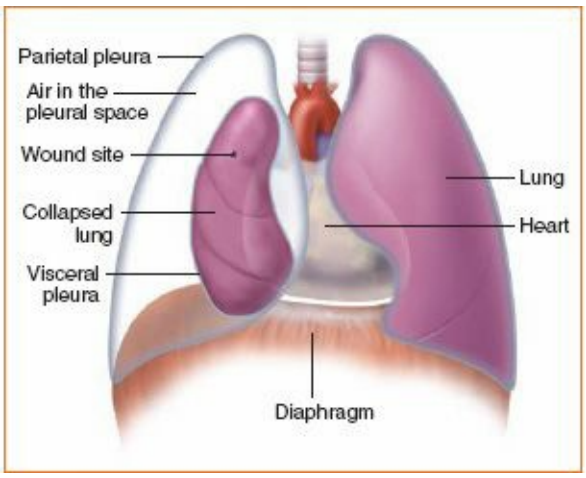


Figure 10-15 A pneumothorax occurs when air leaks into the space between the pleural surfaces from an opening in the chest or the surface of the lung. The lung collapses as air fills the pleural space.

Pneumothorax

A pneumothorax can be either open or closed **Figure 10-15** and is reported to be present in 15% to 50% of chest injuries. An open pneumothorax (also known as a communicating pneumothorax) is commonly called a sucking chest wound **Figure 10-16**, even though it does not always make a sucking noise. A tension pneumothorax occurs when a defect in the chest wall allows air to enter the thoracic space, breaking the normal adherence between the pleura (think of the suction between a wet glass and the counter top), which allows the lung to collapse **Figure 10-17**. This is often a result of a penetrating injury to the outer chest, such as a stab wound, or it can occur internally from a sharp broken rib end.

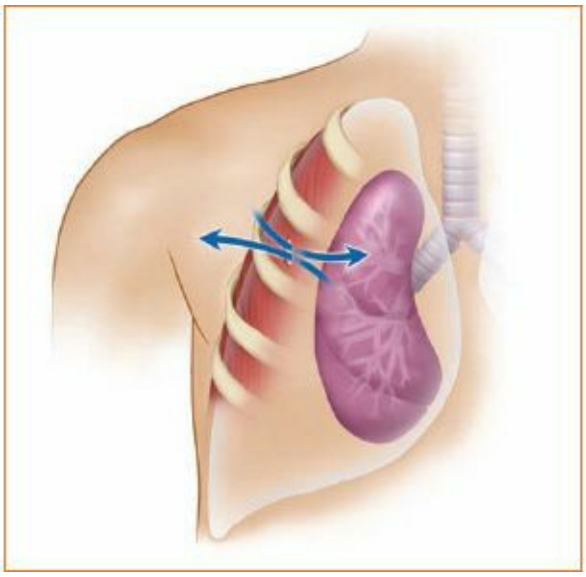


Figure 10-16 With a sucking chest wound, air passes from the outside into the pleural space and back out with each breath. The size of the defect does not need to be large to compromise ventilation.

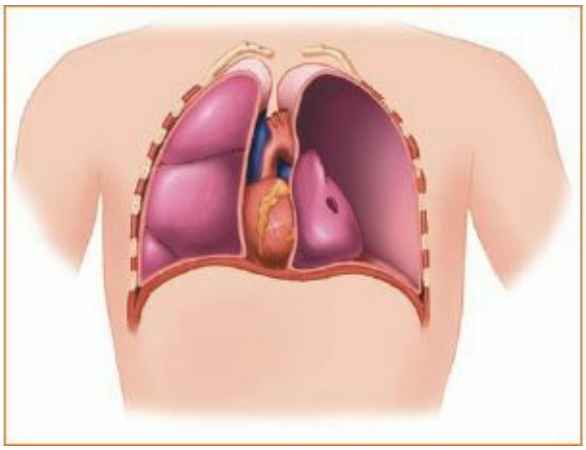


Figure 10-17 In a tension pneumothorax, air accumulates in the pleural space, eventually causing compression of the heart and great vessels.

These types of injuries cause air to be drawn into the thoracic cavity with each breath. The air enters the dead space in the pleural cavity instead of the lung; therefore, it does not oxygenate blood and it causes hypoxia. A collapse as small as 10% can be life threatening if there are other comorbidities, although a pneumothorax this small may be hard to appreciate by auscultation.

The differential diagnosis for respiratory distress in chest trauma includes the following:

- Simple pneumothorax
- Tension pneumothorax
- Flail chest

- Hemothorax
- Tracheobronchial injury
- Pulmonary contusion

Open Pneumothorax

In order to promote adequate oxygenation and ventilation, it is critical that the CCTP recognize and treat an open pneumothorax as soon as possible.

Equipment needed for managing an open pneumothorax includes the following:

- Body substance isolation equipment
- Occlusive dressing
- Medical tape

Indications include:

- Emergency relief of dyspnea secondary to open pneumothorax

Contraindications include:

- Tension pneumothorax

Complications of sealing a sucking chest wound include a tension pneumothorax. This can occur if air is still able to enter the pleural cavity from the lung side of the wound and builds up, or if the seal is incomplete. If this happens, the seal should be "burped" by releasing an edge. Many techniques of sealing sucking chest wounds attempt to address these issues, but pressure build-up or an ineffective seal may still occur. Once a seal is applied, constant surveillance is necessary to guard against either possibility. Assisted ventilation and/or needle decompression may be necessary.

Signs and Symptoms

Open Pneumothorax

- Penetrating chest trauma with a sucking sound ("sucking chest wound")
- Dyspnea
- Tachypnea

Transport Management

Open Pneumothorax

- Maintain an open airway and administer high-flow oxygen.
- Immediately close the chest wound, initially with a gloved hand.
- Apply an occlusive dressing, taped down on three sides so that a flutter value is created.
- Initiate a large-bore IV.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.
- Watch for the development of a tension pneumothorax or an inadequate seal and the need for needle decompression or assisted ventilation.
- Consider inserting a chest tube if protocol allows.
- Monitor the chest tube drainage system during transport.

Skill Drill 10-1 illustrates the clinical procedure for managing an open pneumothorax, which is described below:

1. Maintain an open airway and administer high-flow oxygen **Step 1**.
2. Immediately close the chest wound **Step 2**. This can be initially accomplished by the use of a gloved hand. However, to reduce the risk of a tension pneumothorax, apply an occlusive dressing, taped down on three sides so that a flutter valve is created **Step 3**. Adequate overlap on the open edge is required to create a good seal. Aluminum foil does not seal on inhalation; a soft air-occlusive dressing is preferable. Several commercial devices are also available.
3. Initiate a large-bore IV to maintain peripheral perfusion **Step 4**.
4. Monitor oxygen saturation, vital signs, and the cardiac monitor **Step 5**.
5. Watch for development of a tension pneumothorax or an inadequate seal, and the need for needle decompression or assisted ventilation.
6. Transport to an appropriate facility.

Possible complications include converting an open pneumothorax to a tension pneumothorax. The CCTP should watch for increasing difficulty in breathing, peak inspiratory pressure (PIP), tracheal tugging or deviation, and jugular vein distention (JVD) as late signs.

Simple Pneumothorax

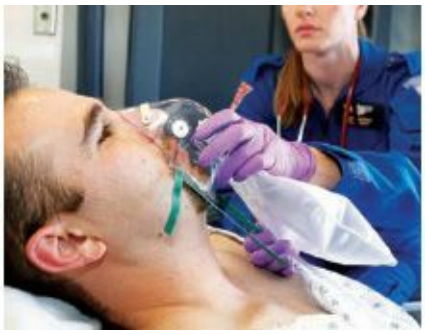
A simple pneumothorax is most often associated with a closed chest injury, such as from a fractured rib being driven inward into the lung or a collapsed lung that results from a medical cause, which is sometimes called a spontaneous pneumothorax. Loss of the negative pressure holding the two layers of the pleura together allows the lung to collapse like an empty balloon. The term actually refers to any pneumothorax that is not a tension pneumothorax and, therefore, may be used to refer to an open and a closed pneumothorax as long as there is no build-up of pressure in the pleural cavity outside the lung.

Tension Pneumothorax

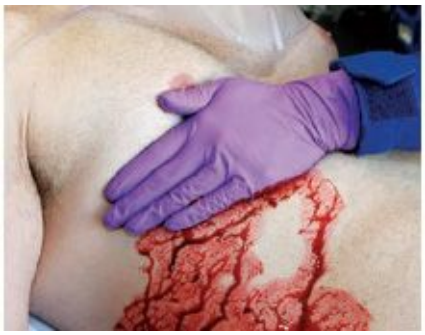
A tension pneumothorax is a life-threatening injury that results from a continual influx of air into the pleural space. It may occur secondary to a closed simple pneumothorax or after sealing an open pneumothorax, which increases interthoracic pressure, hampers the body's ability to oxygenate blood or eliminate carbon dioxide from blood, and eventually collapses the affected lung. The collapse in the lung causes a shift in the mediastinum away from the injured side, resulting in ventilatory and circulatory compromise from the collapsed lung. The great vessels and the heart become deformed under the pressure. Failure to treat tension pneumothorax will cause the patient to progress to pulseless electrical activity and cardiopulmonary arrest.

Skill Drill 10-1

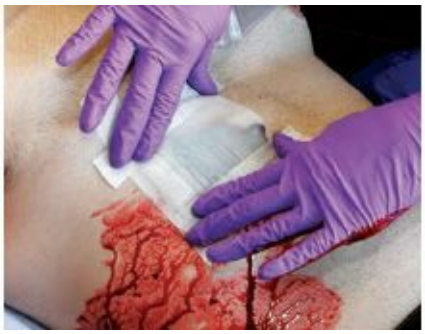
Managing an Open Pneumothorax



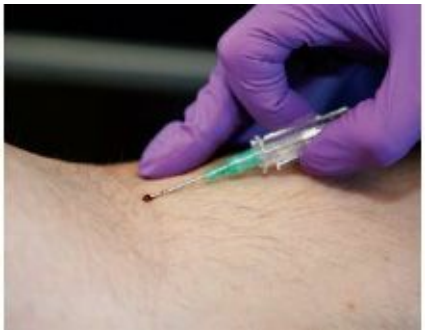
1 Maintain an open airway and administer high-flow oxygen.



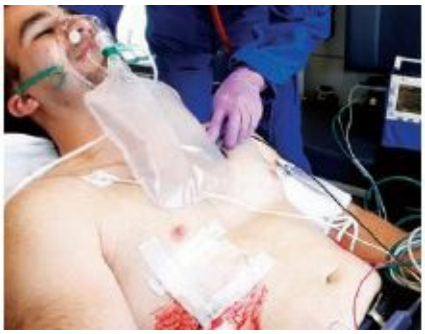
2 Immediately close the chest wound, initially with a gloved hand.



3 Apply an occlusive dressing, taped down on three sides so that a flutter valve is created.



4 Initiate a large-bore IV.



5 Monitor oxygen saturation, vital signs, and the cardiac monitor. Watch for development of a tension pneumothorax or an inadequate seal, and the need for needle decompression or assisted ventilation.

Clinical signs and symptoms of a tension pneumothorax include dyspnea, anxiety, JVD (although this may be absent due to hypovolemia), tachypnea, and **tracheal deviation** (tracheal deviation is a late sign not often observed in the prehospital setting). Tracheal tugging during inspiration, a much more subtle sign, is observed much earlier in the process, but the CCTP should actively look for this sign. The most important signs, however, are increasing dyspnea or decreasing ventilatory compliance to bagging and increasing PIP or peak airway pressure, depending on settings if the patient is receiving ventilation. Auscultation of lung sounds will reveal diminished or absent breath sounds (which are difficult to appreciate in aircraft and on noisy scenes) on the affected side if the pneumothorax is large enough. Pulsus paradoxus (a pulse that disappears on inspiration that also can be associated with a drop in systolic blood pressure), electrical alternans (an alternating large QRS complex with a small QRS complex), and elevated central venous pressure (CVP) may also be present. PIP will go up despite an open airway. Management of tension pneumothorax is performed by immediate needle decompression.

Equipment for managing a tension pneumothorax includes the following:

- Body substance isolation equipment, including sterile gloves
- 14-gauge or larger IV catheter (2.5″ to 3″ is not a standard IV needle length and requires special stocking)
- 10-mL syringe with 1 to 2 mL of sterile saline in it in order to see bubbling air escaping because it is too difficult to hear in many cases
- 2% Chlorhexidine
- Flutter valve (Heimlich or nonlubricated condom or glove finger) optional
- Sterile dressings

Indications include:

- Emergency relief of tension pneumothorax

Contraindications include:

- Patient without signs of a tension pneumothorax

Skill Drill 10-2 demonstrates the clinical procedure for managing a tension pneumothorax, which is described below:

1. Assess the patient to ensure that the presentation matches that of a tension pneumothorax **Step 1**.

- Difficult ventilation despite an open airway (decreasing compliance or increasing PIP or peak airway pressure)
- JVD (may not be present with associated hemorrhage)
- Absent or decreased breath sounds on the affected side
- Hyperresonance to percussion on the affected side
- Tracheal deviation away from the affected side (this late sign is not always present)
- Electrical alternans and pulsus paradoxus
- Elevated CVP
- Signs of impending cardiovascular collapse

2. Prepare and assemble the necessary equipment **Step 2**.
   - Large-bore IV catheter, preferably 10- to 14-gauge and at least 2″ long on a syringe filled with 1 to 2 mL of sterile saline
   - Alcohol or 2% chlorhexidine
   - Adhesive tape
3. Select the appropriate site **Step 3**: midclavicular, second intercostal space, above the third rib **Figure 10-18**; or, less commonly, the midaxillary, above the fifth or sixth rib. Go over the rib to avoid the neurovascular bundle under each rib.
4. Select the largest available needle. It is optional to attach it to a syringe with a few milliliters of saline so the bubbles are visible.
5. Cleanse the site with an appropriate aseptic technique **Step 4**.
6. Make a flutter valve by inserting the needle through the end of a condom or use a commercial device such as the Asherman Chest Seal (not necessary in extremis) **Step 5**.
7. Insert the needle at a 90° angle over the rib **Step 6**.
8. Remove the needle and listen for a rush of air or look for bubbles in the syringe **Step 7**.
9. Advance the catheter over the needle and secure it in place **Step 8**.
10. Dispose of the needle in a sharps container.
11. Closely monitor vital signs, oxygen saturation, and lung compliance.
12. Be prepared to repeat decompression if necessary.

Complications include improper placement, which could lead to injury to the intercostal vessels and significant hemorrhage. Also, passing the needle into the chest may injure the lung parenchyma.

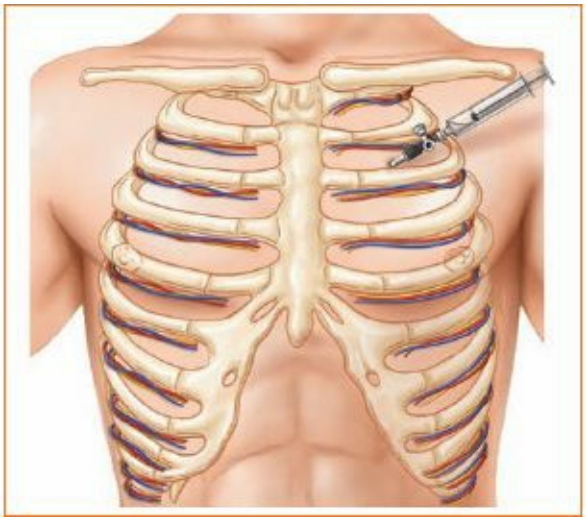


Figure 10-18 Correct placement for needle decompression. The position of nerves, arteries, and veins is shown in relation to the ribs.

Signs and Symptoms

Tension Pneumothorax

- Dyspnea
- Anxiety
- JVD
- Tachypnea
- Tracheal deviation
- Tracheal tugging during inspiration
- Decreasing ventilatory compliance to bagging
- Increasing PIP or peak airway pressure, if the patient is on a ventilator
- Diminished or absent breath sounds on the affected side, if the pneumothorax is large enough
- Pulsus paradoxus
- Electrical alternans
- Elevated CVP

Transport Management

Tension Pneumothorax

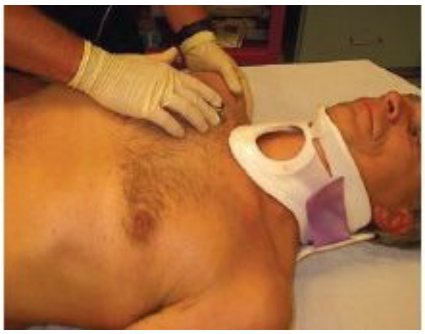
- Perform immediate needle decompression.
- Consider inserting a chest tube if protocol allows.
- Maintain the chest tube on suction during transport.

Hemothorax

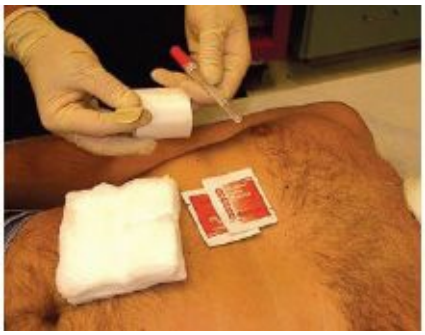
Blood in the pleural space is called a hemothorax **Figure 10-19**. Each of the body's thoracic cavities can accumulate up to 3,000 mL of blood. A massive hemothorax occurs when at least 1,500 mL of blood accumulates in a thoracic cavity. A torn intercostal artery can bleed at a rate of 50 mL/min. The obvious threat from a hemothorax is hypovolemic shock, although a large amount of blood in the pleural cavity can interfere with lung function. Hemothorax can be caused by blunt trauma; however, it is more commonly a result of penetrating trauma. Medical causes include a tumor eroding through great vessels.

Skill Drill 10-2

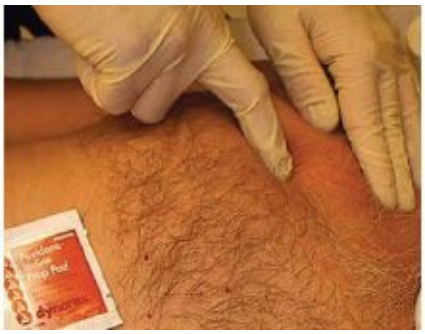
Needle Decompression (Thoracentesis) of a Tension Pneumothorax



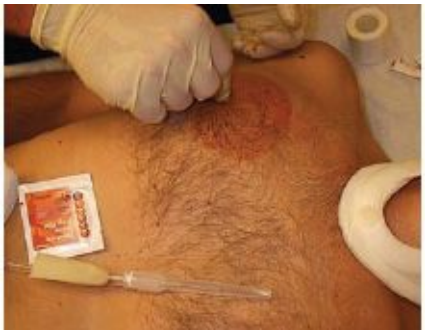
1 Assess the patient.



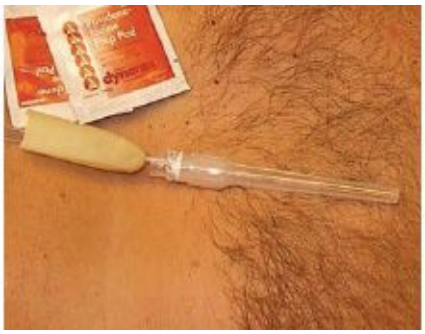
2 Prepare and assemble all necessary equipment.



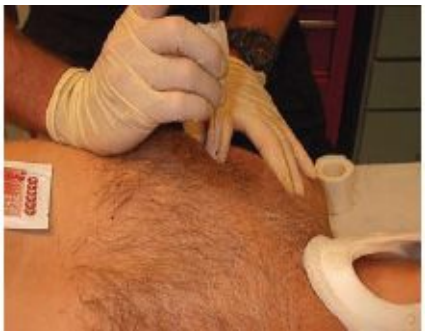
3 Locate the appropriate site between the second and third rib.



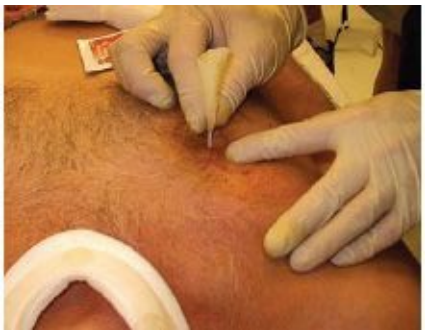
4 Cleanse the appropriate area using an aseptic technique.



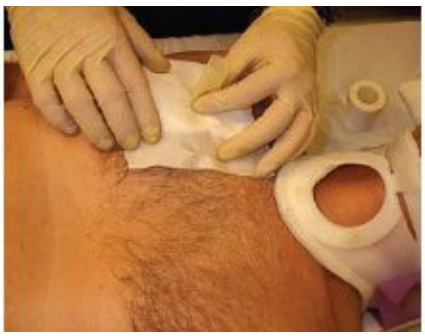
5 Make a one-way or flutter valve (optional).



6 Insert the needle at a 90° angle.



7 Remove the needle and listen for release of air or look for bubbling in the syringe. Properly dispose of the needle in a sharps container.



8 Secure the catheter in place. Monitor the patient closely for recurrence of the tension pneumothorax.

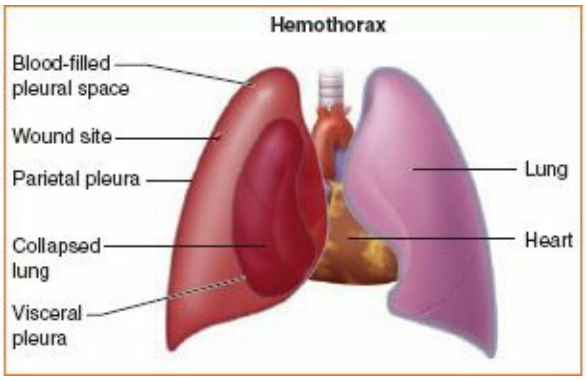


Figure 10-19 A hemothorax is a collection of blood in the pleural space produced by bleeding within the chest.

Clinical signs and symptoms of a hemothorax include hypoxia, agitation, hypotension, tachycardia, tachypnea, decreased breath sounds on the affected side, dullness to percussion on the affected side, hemoptysis, worsening shock, and falling CVP. A patient with a hemothorax rarely presents with JVD or tracheal deviation. Management of a hemothorax is supportive and is conducted as follows:

1. Maintain an open airway and apply high-flow oxygen.
2. Load and go if in the field.
3. Initiate IV fluids to maintain peripheral perfusion; however, raising blood pressure above 90 mm Hg systolic or 65 mm Hg mean arterial pressure (MAP) may increase blood loss or dislodge a clot and restart bleeding.
4. Be alert for development of a tension pneumothorax.

Management: Chest Tube Insertion

Patients with a pneumothorax, a tension pneumothorax, a hemothorax, a hemopneumothorax (combination of a pneumothorax and hemothorax) **Figure 10-20**, or an empyema ultimately may need a chest tube inserted. A chest tube is a flexible plastic tube that is inserted through the side of the chest into the pleural space **Figure 10-21**. It was traditionally attached to an underwater seal to create a one-way system allowing for air or fluid to drain from the chest with each exhalation, reestablishing the interpleural negative pressure and reinflating the lung. Modern systems no longer use a water chamber, but they have the same effect. They are used to remove air, fluid, or pus from the pleural cavity. In some systems, the CCTP may place chest tubes, but in every critical care transport system the CCTP will care for and transport patients with chest tubes in place.

Signs and Symptoms

Hemothorax

- Hypoxia
- Agitation
- Hypotension
- Tachycardia
- Tachypnea
- Decreased breath sounds on the affected side

- Dullness to percussion on the affected side
- Hemoptysis
- Worsening shock
- Falling CVP

Transport Management

Hemothorax

- Maintain an open airway; apply high-flow oxygen.
- Load and go if in the field.
- Initiate IV fluids.
- Be alert for the development of a tension pneumothorax.
- Consider inserting a chest tube if protocol allows.
- Maintain the chest tube on suction during transport.

Controversies

Use of fluids for uncontrolled internal bleeding is controversial. Although raising the blood pressure too high may increase blood loss or dislodge a clot, an adequate blood pressure of 90 mm Hg systolic or an MAP of 65 mm Hg must be maintained to perfuse the heart and brain. The ideal fluid for use is one that carries oxygen, such as blood.

The procedure for insertion of a chest tube begins with collecting the needed equipment (often packaged as a "tray"):

- Body substance isolation equipment, including sterile gloves
- Scalpel
- Chest tube: 36F to 40F

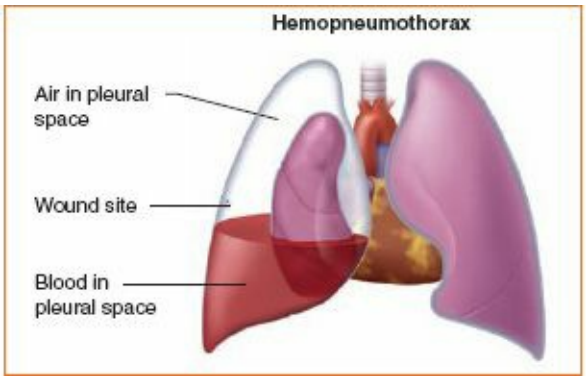


Figure 10-20 In a hemopneumothorax, both blood and air are present.

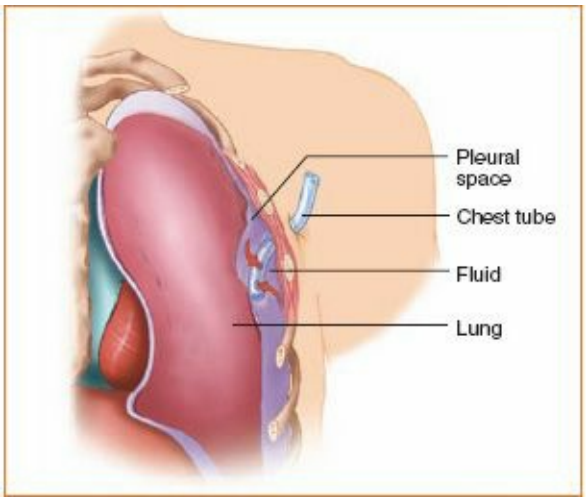


Figure 10-21 A chest tube is inserted through the side of the chest into the pleural space.

- Kelly clamps (curved and straight)
- Sterile occlusive dressing
- Suturing material
- 2% Chlorhexidine
- A local anesthetic if the patient is awake
- A fluid collection device with a one-way valve that allows air, fluid, or pus to flow out of the chest. If not available, an indwelling catheter bag with a one-way valve may be used
- Tape
- Mechanical suction device (not always used)

Indications include:

- Pneumothorax
- Hemopneumothorax
- Empyema

Complications include:

- Recurrent pneumothoraces
- Accidental removal Broken collection chamber
- Parenchymal injury
- Laceration of intercostal vessels
- Creation of a hemothorax or bleeding
- Misplacement below the diaphragm
- Infection

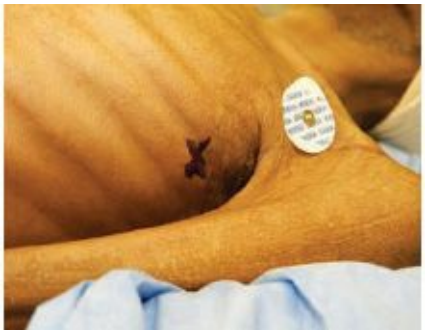
Skill Drill 10-3 shows the clinical procedure for inserting a chest tube, which is described below:

1. Select the appropriate site: midaxillary over the fifth rib **Step 1**.
2. Connect the indwelling catheter bag or the collection device to the distal end of the one-way valve with a rubber band **Step 2**. Be certain that the arrow on the valve is facing away from the patient.
3. Cleanse the site with an appropriate aseptic technique **Step 3**.

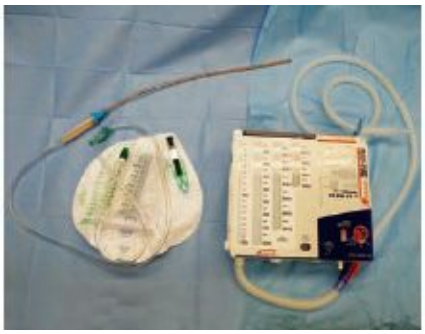
4. Anesthetize the area, including the pleura and periosteum, over the fifth rib **Step 4**, if the patient is conscious and time permits. If possible, place the patient in a 30° reverse Trendelenburg (head up) position so blood drains to the low end of the lung.
5. Mark the tube for the desired length of insertion **Step 5**.
6. Clamp the distal end of the tube with a large clamp (such as a Kelly clamp) and the proximal end of the tube with a curved clamp.
7. Make a transverse incision over the fifth rib in the midaxillary line **Step 6**. If the patient has fractured ribs, use the midclavicular line (between the midaxillary line and the nipple line).
8. Tunnel over the fifth rib with a large curved clamp, push through the pleura, spread the clamp, and leave it in place **Step 7**.
9. Grasp the clamp attached to the end of the chest tube and advance it through the space created by the first clamp, directing it posteriorly and downward toward the diaphragm **Step 8**.
10. Remove the clamps and advance the tube to the predetermined mark indicated on the tube, at least 5 cm beyond the last hole **Step 9**.
11. Connect the collection device **Step 10**.
12. Remove the distal clamp **Step 11**.
13. Suture the tube in place and close the wound **Step 12**.
14. Note the depth of the tube at the skin (marked on the tube) and mark with a felt-tip pen.
15. Cover the insertion site with an occlusive dressing and reinforce all connections with tape **Step 13**.
16. Upon arrival at the receiving facility, obtain a chest radiograph to confirm placement.
17. Document the procedure, including the size of the chest tube inserted, the amount of return of air/fluid after insertion of the tube, and any changes in patient condition (including oxygen saturation).

Skill Drill 10-3

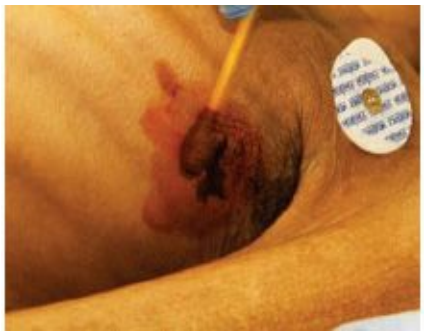
Chest Tube Insertion



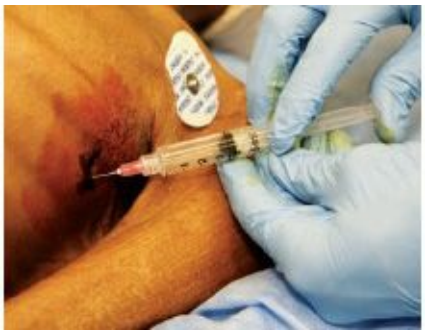
1 Select the appropriate site.



2 Connect the collection device to the distal end of the one-way valve with a rubber band.

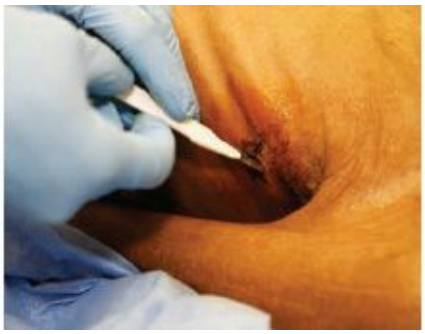


3 Cleanse the site with an appropriate aseptic technique.

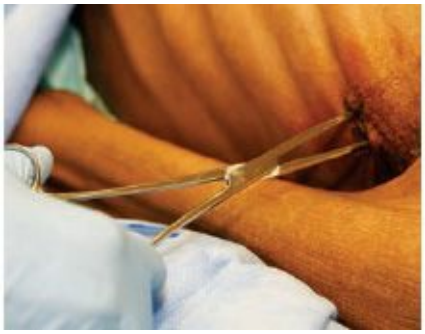


4 Anesthetize the area, including the pleura and periosteum, over the fifth rib.

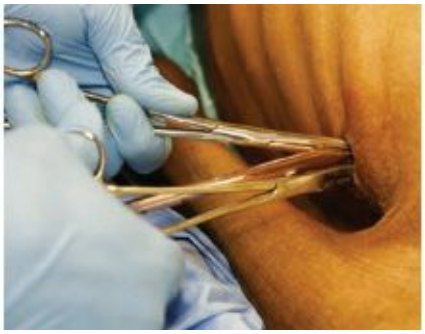
5 Mark the tube for the desired length of insertion. Clamp the distal end of the tube with a large clamp and the proximal end of tube with a curved clamp.



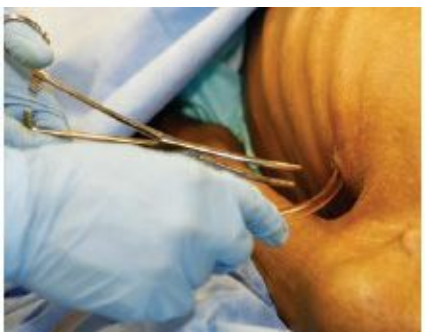
6 Make a transverse incision over the fifth rib at the midaxillary line. If the patient has fractured ribs, use the midclavicular line (between the midaxillary line and the nipple line).



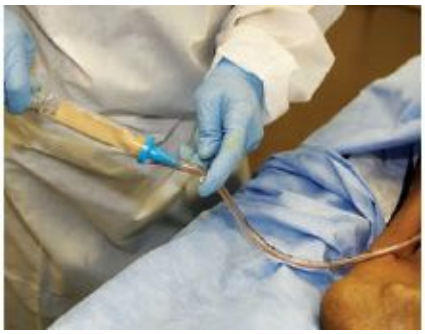
7 Tunnel over the fifth rib with a large curved clamp, push through the pleura, spread the clamp, and leave it in place.



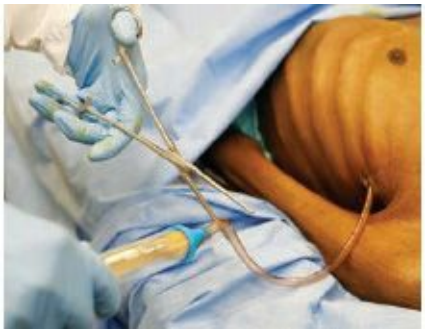
8 Grasp the clamp attached to the end of the chest tube and advance it through the space created by the first clamp.



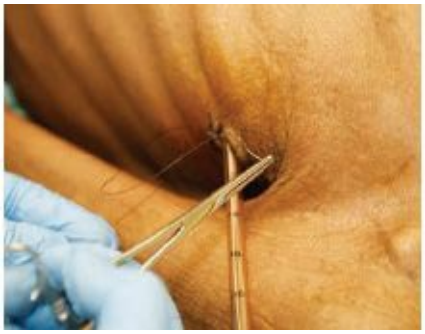
9 Remove the clamps and advance the tube to the predetermined mark indicated on the tube.



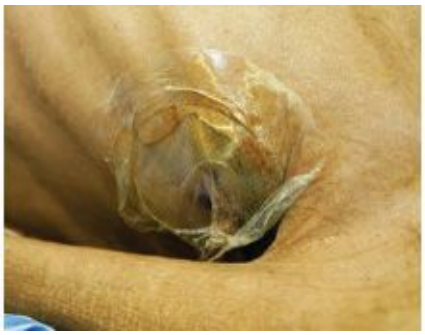
10 Connect the collection device with the one-way valve.



11 Remove the distal clamp.



12 Suture the tube in place and close the wound.



13 Cover the insertion site with an occlusive dressing. Upon arrival at the receiving facility, obtain a chest film to confirm placement. Document the procedure.

When transporting a patient with a chest tube in place:

- Make sure all connections are taped or banded with wire to prevent accidental separation.
- Ensure that the dressing over the insertion site is securely taped and occlusive. Use a felt-tip marker to mark the depth of the tube; if there are markings, note the depth of the tube on the transfer chart.

Make sure the tube is sutured, wired, or taped so it cannot be accidentally pulled out.

- Maintain the drainage unit below the level of the chest at all times during transport. Many units have bed hangers so that the unit can be hung on the stretcher. If there is water in the unit, keep it upright at all times. If attached to a suction device, find out if the suction can be discontinued for transport; if not, attach it to portable suction.
- Tubing should be kept coiled to prevent kinks or dependent loops.
- Access and document bubbling in the water seal (does not have to be continuous), any output in the collection chamber, and its type (eg, frank blood).
- Do not clamp tubes for transport. This is likely to cause a tension pneumothorax.
- Continuous bubbling may be a sign of tracheobronchial laceration. Large amounts of frankly bloody drainage need to be balanced by transfusion.

Management: Blood Administration

Because of the potential for massive hemorrhage from thoracic trauma, the CCTP must be familiar with the administration of blood. The steps for administering blood are covered in Chapter 9.

Flail Chest

A fracture in two or more places to two or more adjacent ribs is a flail chest or a flail segment **Figure 10-22**. Flail chest was found to be present in as many as a third of serious chest injuries in one study. Many escape detection for as long as 6 hours after hospital admission. A central flail involves the sternum. A flail segment may appear to move paradoxically relative to the chest wall but actually is stationary while the rest of the chest moves around it. This movement may be more obvious in unconscious patients who are no longer "splinting." Often, bruises to the underlying tissue can cause a pulmonary or myocardial contusion and represent the most dangerous sequelae of a flail chest. Flail segments are generally caused from blunt trauma, such as striking a steering wheel in a motor vehicle collision (MVC).

Signs and Symptoms

Flail Chest

- Dyspnea
- Severe pain
- Decreased breath sounds on the affected side
- Tenderness
- Crepitus to palpation
- Bruising over the area

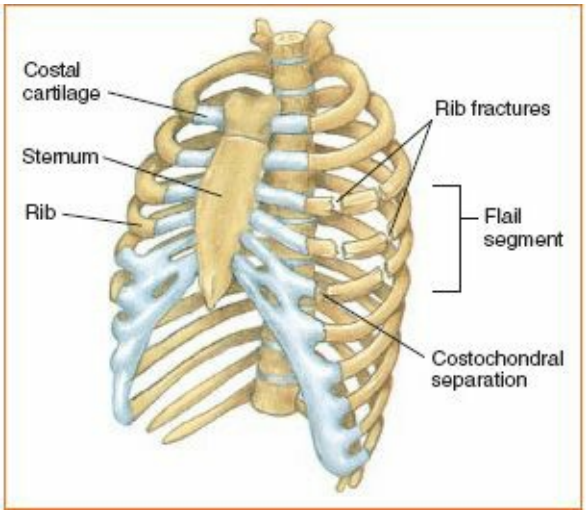


Figure 10-22 In flail chest injuries, two or more adjacent ribs are fractured in two or more places. A flail segment may move paradoxically when the patient breathes, but this is usually only present in the very young, the elderly, or other patients with underdeveloped pectoral and lattisimus muscles.

Transport Management

Flail Chest

- Maintain an open airway; apply high-flow oxygen.
- Assist ventilation with a trial of continuous positive airway pressure; be prepared to intubate.
- Load and go if the patient is a field transport.
- Initiate IV fluids.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.
- Be alert for development of tension pneumothorax, hemothorax (shock), or respiratory failure secondary to a pulmonary contusion.

Clinical signs and symptoms of a flail chest are dyspnea, severe pain, decreased breath sounds on the affected side, tenderness, and crepitus to palpation with possible bruising over the area. Signs and symptoms are often difficult to assess as a result of the patient's efforts to splint the area and ease respiration, and may mask abdominal tenderness.

Flail segments pose a threat to the patient's ability to breathe, increase the risk for developing a hemothorax or pneumothorax, and require immediate treatment.

Management

Management of a flail chest is as follows:

1. Maintain an open airway and apply high-flow oxygen.
2. Assist ventilation with a trial of continuous positive airway pressure and be prepared to intubate.
3. Load and go if a field transport.
4. Initiate IV fluids to maintain peripheral perfusion.
5. Monitor oxygen saturation, vital signs, and the cardiac monitor. Be alert for development of tension pneumothorax, hemothorax (shock), or respiratory failure secondary to a pulmonary contusion.
6. The gold standard treatment for flail chest is mechanical ventilation (also known as internal fixation)

with a sedate, paralyzed patient who has been adequately treated for pain. It may be possible to manage some patients conservatively with pain medications and oxygen supplementation alone.

7. Use of attempts to splint (also called external fixation) or wrap the chest has been shown to be ineffective, and this practice has been abandoned.

Pericardial Tamponade

Pericardial tamponade is a life-threatening condition that requires immediate treatment. This condition is reported in 2% of penetrating injuries to the chest and upper abdomen. It is more common in stab wounds to the heart (as much as 80%) than in gunshot wounds. The pericardium normally contains a small amount of fluid that cushions and lubricates the heart as it expands and contracts. When an abundance of blood or fluid accumulates, it compresses the ventricles of the heart, thus compromising cardiac filling and output **Figure 10-23**. Abnormal amounts of fluid may result from:

- Pericarditis caused by infection and inflammation
- Trauma
- Surgery or other invasive procedures performed on the heart
- Cancer
- Myocardial infarction and congestive heart failure
- Renal failure

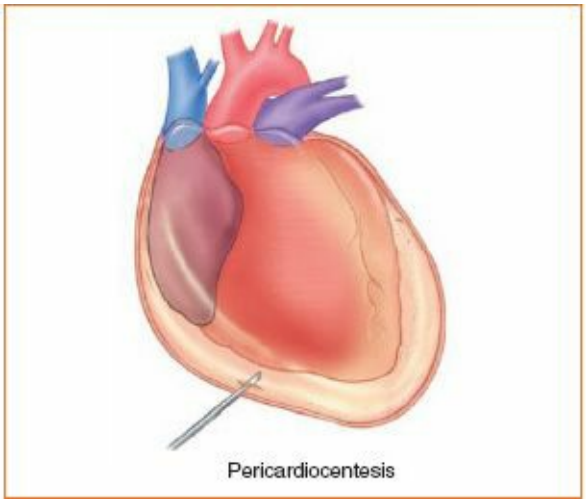


Figure 10-23 Needle aspiration in pericardial tamponade.

Signs and Symptoms

Pericardial Tamponade

- Beck's triad: narrowing pulse pressure, jugular vein distention, and muffled heart tones
- Paradoxical pulse
- Electrical alternans
- Progressive decreases of electrocardiographic (ECG) voltage
- Hypotension
- Cyanosis
- Dyspnea
- Tachycardia

- Pulseless electrical activity
- Rising CVP greater than 15 cm H_2O
- Widened mediastinum on chest radiograph

Transport Management

Pericardial Tamponade

- Provide supportive care.
- Administer a fluid bolus.
- If necessary, perform an emergency pericardiocentesis.

Classic clinical signs and symptoms of a pericardial tamponade are called **Beck's triad**: narrowing pulse pressure, JVD, and muffled heart tones. However, Beck's triad is only present 10% to 40% of the time and can be difficult to assess in the transport environment. With a low blood volume, there may be no JVD. Additionally, the patient may present with a paradoxical pulse (loss of radial pulse upon inspiration, often with a drop of 10 to 15 mm Hg systolic pressure as well), electrical alternans, progressive decreases of ECG voltage, hypotension, cyanosis, dyspnea, tachycardia, and pulseless electrical activity. A rising CVP greater than 15 cm of water may also be present (until precipitous decompensation), especially if the patient is otherwise normovolemic. Chest radiographs may show a widened mediastinum. Actual diagnosis is usually done with an ultrasound exam (FAST exam, discussed previously in this chapter), using a portable FAST unit.

Management

Most often, the prehospital management is only supportive, and a fluid bolus may buy some time; however, the CCTP may be called upon to perform or assist in an emergency **pericardiocentesis**.

The equipment needed for this procedure includes:

- Body substance isolation equipment, including sterile gloves, gown, and mask
- Cardiac monitor and defibrillator
- Pericardiocentesis kit (Mansfield catheter)
- 50-mL Luer-lock syringes
- Sterile tubes for specimen collection
- 2% Chlorhexidine
- Appropriate medications (cardiac, sedating, and numbing)
- Pigtail catheter
- Drapes
- 16- to 18-gauge by 5½″ spinal needle
- Three-way stopcock
- Sterile alligator clip
- Scalpel
- Assorted needles and syringes
- Preferably, the availability of ultrasound imaging

Indications include:

- Chest trauma with patient in extremis such as pulseless electrical activity
- FAST exam showing pericardial blood with clinical signs

Contraindications include:

- No demonstrable pericardial effusion
- Severe thrombocytopenia or noncorrectable coagulopathy
- Skin or soft-tissue infection at the proposed needle site (except in a critical patient)

Complications include:

- Malignant ventricular arrhythmias, including cardiac arrest
- Puncture of the ventricles or atria (most commonly the right ventricle)
- Laceration of the coronary arteries and lung
- Pericardial tamponade from a myocardial laceration
- Air embolism
- Acute pulmonary edema
- Puncture of the liver or stomach
- Infection

Skill Drill 10-4 illustrates the clinical procedure for pericardiocentesis, which is described below:

1. Prepare the equipment and obtain baseline vital signs, including pulse oximetry findings.
2. Place the patient into a supine position with the head of the stretcher elevated 20° to 30° as tolerated.
3. Premedicate per protocol or online medical control.
4. Don a mask, gown, hair cover, and sterile gloves.
5. Cleanse the site with an appropriate aseptic technique and drape the area **Step 1**.
6. Identify the needle entry site directly below the xiphoid—approximately 1 cm left of midline **Step 2**.
7. Infiltrate the area with 1% lidocaine into the skin and deeper tissues, if the patient is conscious **Step 3**.
8. Insert the needle-on-syringe with 2 to 3 mL of sterile saline in it at the identified entry site, at a 30° angle directed toward the left shoulder or directly cephalad. Once the needle is deeper than the skin, inject the saline to remove any skin from the needle **Step 4**.
9. Gently aspirate while inserting the needle **Step 5**. Some resistance may be felt as the needle enters the pericardial sac. *If the needle is advanced too far, the myocardium will become irritated. The needle may vibrate with the pulse. The needle should be pulled back slightly until fluid can be aspirated.*
10. If blood is withdrawn and placed in an open container, it should not clot if it comes from the pericardial sac. A sample can be placed into the collection container and transported with the patient.
11. Administer medications per protocol or online medical control.
12. Monitor for postprocedure complications.
13. Monitor vital signs and for JVD at 15-minute intervals for 1 hour, and then every hour for 4 hours.
14. The entire procedure is often done under ultrasound guidance as well.

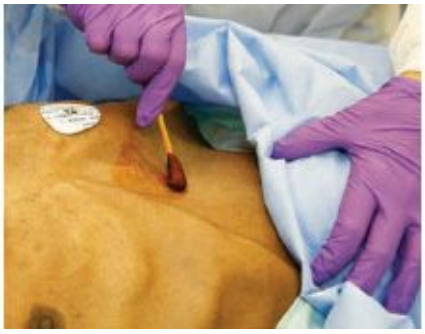
15. In some cases, a catheter may be placed with a stopcock for periodic drainage as needed.

Aortic Dissection/Transection

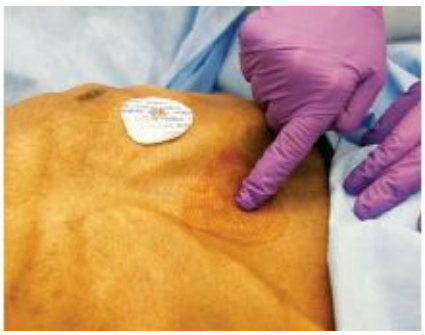
Traumatic aortic rupture is the most common cause of immediate death in MVCs. The body's entire blood volume passes through the aorta; therefore, it is easy to understand how death could be immediate. Most victims die within minutes, often prior to the arrival of the CCTP. Those who do survive require surgical intervention as soon as possible.

Skill Drill 10-4

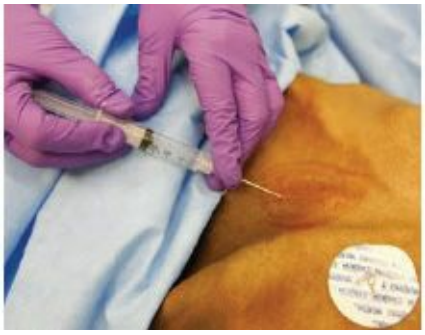
Pericardiocentesis



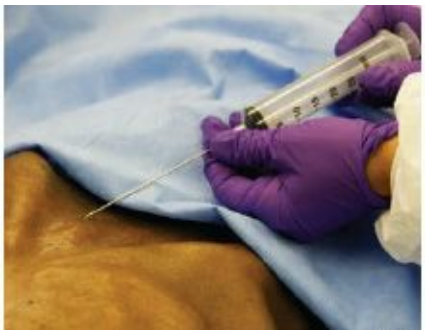
1 Prepare the equipment and obtain baseline vital signs, including pulse oximetry findings. Place the patient into a supine position with the head of the stretcher elevated 20° to 30° as tolerated. Premedicate per protocol or on-line medical control. Don a mask, gown, hair cover, and sterile gloves. Cleanse the site with an appropriate aseptic technique and drape the area.



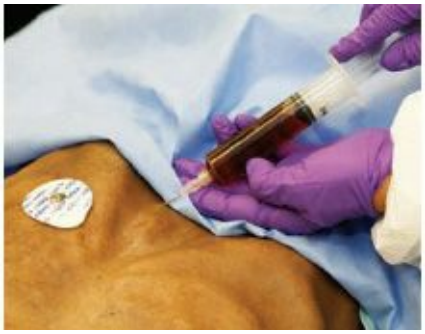
2 Identify the needle entry site directly below the xiphoid.



3 Infiltrate the area with 1% lidocaine into the skin and deeper tissues.



4 Insert the needle-on-syringe at the identified entry site, at a 30° angle directed toward the left shoulder or directly cephalad.



5 Gently aspirate while inserting the needle. *If the needle is advanced too far, the myocardium will become irritated. The needle should be pulled back slightly until fluid can be aspirated.* A blood sample can be placed into the collection container and transported with the patient. Administer medications per protocol or on-line medical control. Monitor for postprocedure complications. Monitor vital signs and for JVD at 15-minute intervals for 1 hour, and then every hour for 4 hours.

The most widely accepted etiology of traumatic aortic tears is that the aorta is injured at the ligamentum arteriosum, the remnant of the fetal ductus arteriosus, as a result of decelerating injuries and sheer force. Tears in the ascending aorta are almost uniformly immediately fatal, but patients with tears on the descending side have about a 15% chance of staying alive until they reach the operating room. There is a 30% mortality rate among those who make it to the hospital. Some trauma centers are now using endovascular stenting with some success. Diagnosis of traumatic aortic tears is very difficult in the prehospital setting and is even missed in the hospital. The CCTP should closely evaluate the mechanism of injury to assess the likeliness of an aortic injury. There may be a pulse deficit or blood pressure difference from one arm to the other if the dissection is at the top of the arch of the aorta. Other signs include hypotension; decreased level of consciousness (due to decreased carotid pressure); hypertension of the upper extremities; decrease of pulse amplitude in the lower extremities; a difference in blood pressure between the two arms (if the dissection is beyond the left subclavian artery), usually greater than 15 to 20 mm Hg; chest pain; chest wall bruising; widened mediastinum on chest radiograph with blurring of the aortic knob; and fractures of the first and second ribs. Definitive diagnosis is made with computed tomography and transesophageal echocardiography.

Signs and Symptoms

Traumatic Aortic Rupture

- Pulse deficit or blood pressure difference from one arm to the other
- Hypotension
- Decreased level of consciousness
- Hypertension of the upper extremities
- Decrease of pulse amplitude in the lower extremities
- Decrease in blood pressure
- Chest pain
- Chest wall bruising
- Widened mediastinum on chest radiograph with blurring of the aortic knob
- Fractures of the first and second ribs

Transport Management

Traumatic Aortic Rupture

- Maintain an open airway; apply high-flow oxygen.
- Assist ventilation; be prepared to intubate.
- Initiate IV fluids.
- Administer beta-blockers (eg, esmolol or metoprolol) and withhold fluids to keep the MAP at 65 to 70 mm Hg.
- Load and go.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.
- Transport to an appropriate facility with early advance notification.

Management

Potential aortic tears are managed as follows:

1. Maintain an open airway and apply high-flow oxygen.
2. Assist ventilation and be prepared to intubate.
3. Initiate IV fluids to maintain blood pressure only at 90 mm Hg systolic or 65 mm Hg MAP.
4. Medications such as beta-blockers (eg, esmolol or metoprolol) and withholding fluids may be used to keep the MAP around 65 to 70 mm Hg.
5. Load and go.
6. Monitor oxygen saturation, vital signs, and the cardiac monitor.
7. Transport to an appropriate facility with early advance notification.

Use of a pneumatic antishock garment is contraindicated.

Myocardial Contusion

A bruise to the myocardium is primarily the result of blunt trauma to the anterior chest wall. MVCs are the obvious mechanism of injury, but other blows to the front of the chest (eg, gunshot to a bulletproof vest) may also be a cause. Estimates vary widely and put the incidence of myocardial contusion at 16% to 76%

of blunt chest trauma. Several have been reported even in low-speed (20 to 35 mph) MVCs, without external chest wall bruising. The amount of damage to myocardial cells varies widely as well. A bruise is damage to small blood vessels, causing local bleeding and direct trauma to cells. The areas downstream from damaged vessels are no longer being perfused, causing damage similar to a myocardial infarction.

Assessment

The most common signs and symptoms are similar to those of an acute myocardial infarction: chest pain, palpitations, and arrhythmias. Arrhythmias may develop as late as 12 to 72 hours after injury. Arrhythmias to watch for include premature ventricular complexes, ventricular tachycardia, and ventricular fibrillation. ST-segment elevations may be evident on the 12-lead ECG, particularly the right-sided (V_4R) leads. Bundle-branch blocks, especially on the right, and atrioventricular blocks may develop if the intraventricular septum is involved. The wider the QRS is in a bundle-branch block, the more the ventricles are out of phase, reducing the ejection fraction. Other signs include cardiac murmur, pericardial friction rub, and persistent tachycardia without other cause. Troponin I and T are very specific to cardiac injury. The creatine kinase MB is not used. If the injury is large enough, cardiogenic shock may result from reduced contractile ability of the myocardium.

Signs and Symptoms

Myocardial Contusion

- Chest pain
- Palpitations
- Arrhythmias: premature ventricular complexes, ventricular tachycardia, and ventricular fibrillation
- ST-segment elevation on the 12-lead ECG, particularly the V_4R leads
- Bundle-branch blocks, especially on the right side
- Atrioventricular blocks
- Cardiac murmur
- Pericardial friction rub
- Persistent tachycardia without other cause
- Cardiogenic shock

Transport Management

Myocardial Contusion

- Administer oxygen.
- Provide pharmacologic treatment of arrhythmias.
- Watch closely for development of hemopericardium, leading to pericardial tamponade, myocardial rupture, or ventricular aneurysm.

Management

Treatment is similar to that of a myocardial infarction—oxygen, pharmacologic treatment of arrhythmias, and a close watch for the development of hemopericardium leading to pericardial tamponade, myocardial

rupture, or ventricular aneurysm. Aspirin and fibrinolytics are contraindicated.

Diaphragmatic Rupture

Rupture of the diaphragm may come from either blunt or penetrating trauma. Penetration inferior to the nipples or scapula may lacerate the diaphragm. These lacerations are usually small and not an acute problem, but will require eventual surgical repair. A larger problem is blunt compression of the abdomen causing a large rupture and herniation of abdominal organs into the thoracic cavity. This impinges on lung function and decreases venous return, thus both ventilatory and cardiac output are reduced. Signs and symptoms include abdominal pain, acute respiratory distress, decreased breath sounds, or abdominal sounds in the chest cavity. There may be subcutaneous emphysema or obvious penetration in the area previously noted. If enough of the abdominal organs are herniated into the chest, the abdomen may be sunken and appear empty. Management includes supporting ventilation and oxygenation. Use of a pneumatic antishock garment is contraindicated. There are controversial recommendations to limit positive-pressure ventilations and to insert a nasogastric or orogastric tube to decompress the stomach.

Signs and Symptoms

Diaphragmatic Rupture

- Abdominal pain
- Acute respiratory distress
- Decreased breath sounds
- Abdominal sounds in the chest cavity
- Subcutaneous emphysema
- Obvious penetration in the abdomen
- Sunken abdomen and/or abdomen that appears empty

Transport Management

Diaphragmatic Rupture

- Assist ventilation.
- Administer oxygen.
- Insert a nasogastric or orogastric tube to decompress the stomach (controversial).

Tracheobronchial Disruption

Tracheobronchial injuries are rare, but often life threatening. These occur in less than 3% of blunt and penetrating chest injuries, but may have up to a 30% fatality rate. More commonly caused by penetrating injury, most injuries occur within an inch and a half of the carina, but can occur anywhere along the tracheobronchial tree. The leakage of air may cause a tension pneumothorax or a tension pneumomediastinum, which will act like a cardiac tamponade. Signs and symptoms include severe respiratory distress, hypoxia, tachycardia, subcutaneous emphysema, especially in the neck, hemoptysis, JVD, and tracheal deviation. An apparent tension pneumothorax that does not improve after needle thoracostomy or one with continuous flow of air from the needle or chest tube is probably a

tracheobronchial tear. Management includes judicious use of ventilatory support. If positive-pressure ventilation makes the patient worse and no relief is obtained from needle decompression, then oxygen supplementation alone may be necessary. A chest tube may be indicated, whereas rapid sequence intubation (RSI) is contraindicated. The muscle tension of the neck may be the only thing holding the trachea in place. Paralysis of the muscles may allow retraction of the trachea into the chest.

Signs and Symptoms

Tracheobronchial Disruption

- Severe respiratory distress
- Hypoxia
- Tachycardia
- Subcutaneous emphysema, especially in the neck
- Hemoptysis
- Jugular venous distention
- Tracheal deviation

Transport Management

Tracheobronchial Disruption

- Administer oxygen.
- Provide positive-pressure ventilation, unless it worsens the patient's condition.
- Perform needle decompression, if necessary.
- Insert a chest tube, if necessary and if protocols allow.

Pulmonary Contusion

Tearing and lacerations to the lung tissue can cause bleeding and leakage of plasma into alveoli and the interstitial spaces around them **Figure 10-24**. This damage occurs when the lung hits the inside of the chest wall (such as in flail chest injury) or as a result of shearing force causing stretching and tearing between fixed and movable parts of the lung. Pressure waves from explosions also stretch and tear the lung tissue. Blood in and around the alveoli interferes with gas exchange and leads to severe hypoxemia. This bleeding may be rapid, but more often develops over hours; the patient's deterioration may be 24 hours or more. Uninjured parts of the lung undergo thickening of the alveolar capillary membranes over time as well.

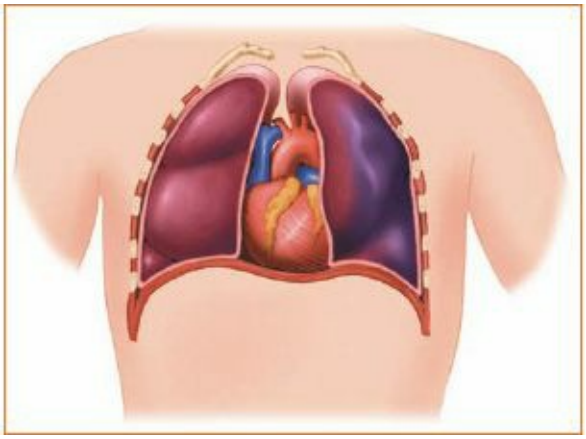


Figure 10-24 Pulmonary contusion.

Patients with flail chest or similar significant mechanisms of injury must be assumed to have a developing pulmonary contusion. Signs and symptoms may initially be absent, but over time the condition of the traumatized patient deteriorates, at which point the CCTP is called to transfer the patient to a more sophisticated medical center. The primary sign of this deterioration is increasing hypoxia. Definitive chest radiographs will show an opacity in the area of chest wall damage, but a clear chest radiograph does not rule out the pulmonary contusion. Worsening blood gases over time with a pertinent mechanism of injury are sufficient to make the diagnosis.

Signs and Symptoms

Pulmonary Contusion

- Initially absent (no signs and symptoms)
- Increasing hypoxia
- Opacity in the area of chest wall damage on chest radiograph
- Worsening blood gas levels over time

Transport Management

Pulmonary Contusion

- Provide ventilatory support: either (1) intermittent mandatory ventilation and positive end-expiratory pressure of 10 to 15 cm H_2O or (2) continuous positive airway pressure at 10 to 15 cm H_2O.
- Provide adequate analgesia.
- Give resuscitation fluids.

Treatment is to support ventilation with mechanical ventilation traditionally done with intermittent mandatory ventilation and a positive end-expiratory pressure of 10 to 15 cm H_2O. Now, more patients are successfully supported on noninvasive continuous positive airway pressure at 10 to 15 cm H_2O. In either case, especially in the presence of flail chest, adequate analgesia must be used. Some studies suggested fluid restriction for these patients, but that has largely been replaced with judicious use of necessary fluids to maintain adequate CVP or MAP. In the field, resuscitation fluids should be given as needed for

systolic pressure support.

Esophageal Perforation

Esophageal perforations are most often caused by penetrating injuries, such as from projectiles or from caustic ingestion. They can also be from a medical cause, such as cancer or gastroesophageal reflux disease erosions. Excessive vomiting may cause a Mallory-Weiss tear.

Signs and symptoms include pain, fever, dysphagia, subcutaneous air in the neck and neck stiffness, and pleuritic-type pain. Free mediastinal air or widening may be found on imaging studies. The air introduced into the mediastinum may produce a crunching sound on auscultation.

Treatment is supportive of the ABCs. A nasogastric tube insertion is usually contraindicated or should be placed with extreme care.

Signs and Symptoms

Esophageal Perforation

- Pain
- Fever
- Dysphagia
- Subcutaneous air in the neck and neck stiffness
- Pleuritic-type pain
- Free mediastinal air or widening on imaging studies
- A "crunching" sound on auscultation

Transport Management

Esophageal Perforation

- Manage the ABCs.
- If a nasogastric tube is inserted, use extreme care.

Traumatic Asphyxia

Traumatic asphyxia describes a severe, sudden crushing injury to the chest and abdomen such as a patient being caught between a truck backing up and a loading dock or a car falling off a jack. This forces blood backward out of the right side of the heart, engorging the veins of the chest, neck, and head. The deoxygenated blood makes the chest, neck, and head look blue or purple, as in extreme cyanosis, giving rise to the name asphyxia **Figure 10-25**. It is not a form of asphyxia nor is it by itself fatal, despite its undeserved reputation for high mortality. Associated injuries, if present, are far more serious. These include brain hemorrhage, possible cardiac rupture, eye injuries, flail chest, ruptured diaphragm, and pulmonary or myocardial contusions. If the patient survives the initial injury, the dramatic purple color fades after several weeks.

Specific assessment findings, besides the mechanism of injury and the purple discoloration, include JVD, conjunctival bleeding, and a sharp line of demarcation with normal skin color below. Treatment is primarily supportive with treatment for the associated injuries. If the patient is found entrapped, IVs and a

treatment plan to deal with sudden hypotension and possible release of myoglobin and potassium are necessary when the patient is released. (The patient may have crush syndrome, depending on how long he or she has been entrapped.)

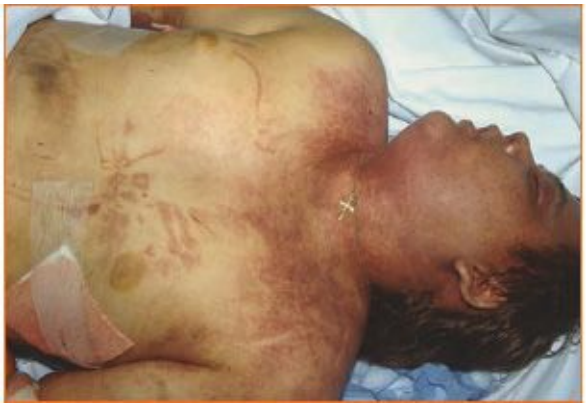


Figure 10-25 Traumatic asphyxia.

Signs and Symptoms

Traumatic Asphyxia

- Blue or purple color of the chest, neck, and head
- Associated injuries
- JVD
- Conjunctival bleeding
- Sharp line of demarcation with normal skin color below

Transport Management

Traumatic Asphyxia

- Provide supportive care.
- Treat the associated injuries.
- If the patient is found entrapped, insert IVs and develop a treatment plan to deal with sudden hypotension and possible release of myoglobin and potassium when the patient is released.

Facial Trauma

Facial trauma can be life threatening and has many implications for patients socially and regarding self-image. Facial trauma can be distracting for providers as well. Facial injuries are of special significance because the patient experiences an immediate life threat as a result of airway compromise and the ability to get sensory information through sight and hearing. The CCTP must also be aware of his or her own emotional response and reactions to these injuries.

Ear Injuries

Ear injuries are generally not considered an area of concern for CCTPs because these are not life threatening nor do they involve technology for transport. They may be present in patients who have other more serious injuries, but ear injuries usually do not need much attention on either a scene call or an interfacility transport. A punctured eardrum, however, may cause vertigo and nausea, which the CCTP may need to control with medication. The patient should be protected from aircraft noise.

External Ear Injury

External ear injuries are considered a local injury with no acute systemic implications. Long-term possibility of infection or deformity (cauliflower ear) is not usually life-threatening. Standard soft-tissue injury care applies **Figure 10-26**. Blood or fluid coming from the auditory canal indicates more serious injury. A halo test for cerebrospinal fluid mixed with blood, indicating basilar skull fracture, is easily done with a piece of filter paper or a gauze pad. Do not pack the ear canal; rather, use a loose dressing instead. Chapter 11 covers neurologic emergencies in depth.

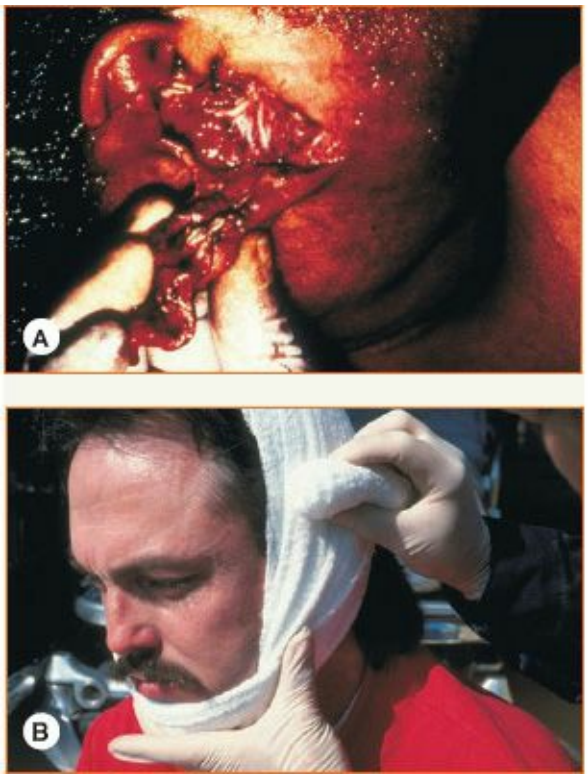


Figure 10-26 **A.** A major laceration of the ear. **B.** Place a soft, sterile pad behind the ear, between it and the scalp. Then wrap a roller gauze bandage (eg, Kling® or Kerlex®) around the head to include the entire ear.

Ruptured Tympanic Membrane

A ruptured tympanic membrane may be the result of overpressure injury, such as from an explosion or

from a direct blow to the ear. Rupture may also be the result of failure to equalize middle ear pressure during scuba diving. In the absence of infection, most will heal spontaneously and are not life threatening, but should raise the suspicion of other, much more serious overpressure injuries. Principal signs and symptoms are pain and vertigo, possibly accompanied by vomiting. There may be blood visible in the ear canal. Treatment includes external dressing to reduce the chance of infection (do not pack the ear canal), possible use of an antiemetic, and a relative contraindication to aircraft transport of the patient.

Signs and Symptoms

Ruptured Tympanic Membrane

- Pain
- Vertigo
- Vomiting
- Blood visible in the ear canal

Transport Management

Ruptured Tympanic Membrane

- Apply an external dressing (do not pack the ear canal).
- Administer an antiemetic, if necessary.
- Do not transport the patient by air, if possible.

Eye Injuries

The CCTP may transport patients with eye injuries from the field or from a local hospital to a specialty center with ophthalmologic services. Eye injuries, although often dramatic, are not life threatening. Although vision loss is serious, there is usually little for the CCTP to do other than protect the eye from further injury and transport the patient to the ophthalmology service. The major exception is chemical burns to the eye **Figure 10-27**. This is a case in which transport can actually interfere with appropriate care. Adequate washing is essential and should be performed with (preferably) sterile saline or dextrose solution for at least 10 minutes prior to transport for acid burns and at least twice as long for alkali burns. Lavage should then continue en route if possible, which may be difficult in an aircraft. Use of topical anesthesia such as tetracaine or opthane is necessary for flushing to be effective. The CCTP must be alert to the possibility of other more serious injuries that may have been overlooked and should always be treated first. There are many techniques for irrigating the eyes; the Morgan® Lens is one.

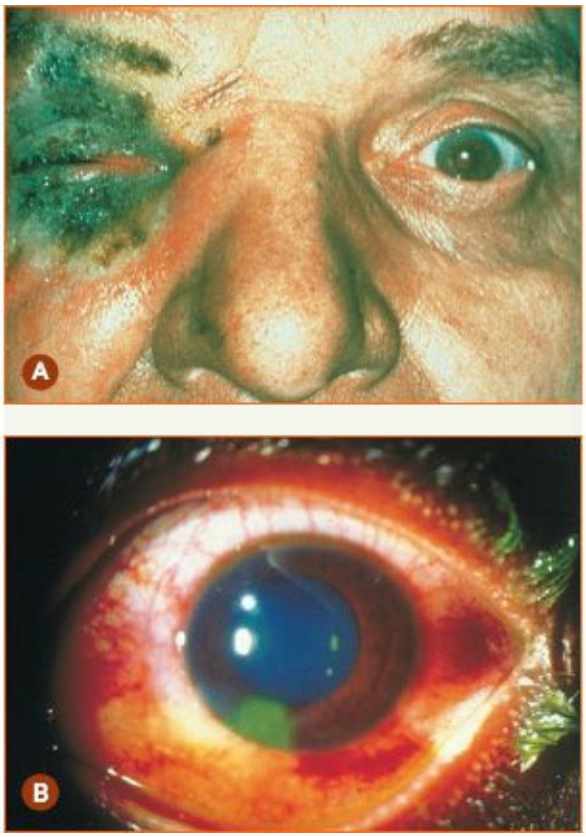


Figure 10-27 **A.** Chemical burns typically occur when an acid or alkali is splashed into the eye. **B.** A chemical burn from lye, an alkaline solution.

Eyelid Lacerations

Soft-tissue injuries to the eye and surrounding structures include lacerations and swelling. Direct pressure applied in a manner not to push on the globe (pressure directly on the globe may cause vagal stimulation) usually stops bleeding. Application of cold may reduce swelling. The CCTP should have concern for concomitant eye injuries.

Conjunctival and Corneal Injuries

The most common conjunctival and corneal injuries are abrasions and foreign bodies. More serious injuries involve shrapnel from high-speed equipment such as drills and saws. These often involve objects impaled in the eye. If possible, the eye and the object should be prevented from movement for transport. A cup or shield is generally used **Figure 10-28**. The unaffected eye may need to be patched to prevent sympathetic movement. Removal of most objects will need to be visualized with a slit lamp or ocular loupe under local anesthetic.

Hyphema

A hyphema is a collection of blood in the anterior chamber of the eye **Figure 10-29**. It may be the result of blunt trauma to the eye or a medical cause. It may be a marker of damage to other structures of the eye and, therefore, requires a full ophthalmologic examination. One of the concerns is blood clotting in the canal connecting the anterior to the posterior chamber, causing an acute rise in intraocular pressure. The patient will complain of reduced vision directly proportional to the size of the hyphema. Often the blood is visible unaided, but shining a penlight obliquely at the globe may help to visualize the blood, which will pool with gravity. If there are no other contraindications, transport should be with the patient sitting as upright as possible and both eyes patched. Pain should be managed with acetaminophen with or without codeine and aspirin; other medications with antiplatelet effects should be avoided. An anxiolytic

may facilitate transport.

Figure 10-28 An impaled object is secured with a protective barrier and a bulky dressing.

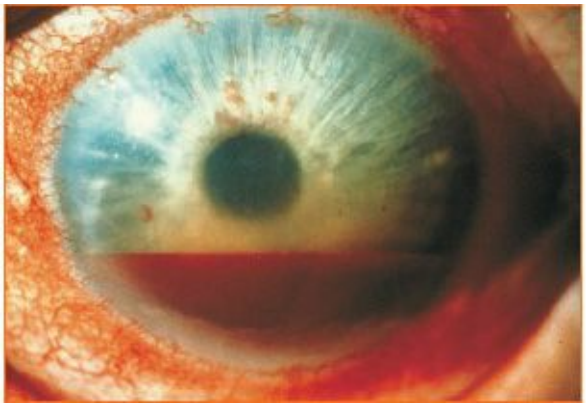


Figure 10-29 A hyphema.

Grading of the hyphema is as follows:

- **Grade 1:** Layered blood occupying less than one third of the anterior chamber
- **Grade 2:** Blood filling one third to one half of the anterior chamber
- **Grade 3:** Layered blood filling one half to less than the total anterior chamber
- **Grade 4:** Total clotted blood, often called a blackball or 8-ball hyphema

Signs and Symptoms

Hyphema

- Patient complaint of reduced vision (directly proportional to the size of the hyphema)
- Pool of blood in the eye; may be visualized either directly or by shining a penlight obliquely at the globe

Transport Management

Hyphema

- Patch both eyes.
- Transport the patient sitting as upright as possible.

- Administer analgesics; acetaminophen with or without codeine and aspirin.
- Administer an anxiolytic if necessary to facilitate transport.

Ocular Globe Rupture

Rupture of the globe with leak of vitreous humor may be the result of penetration with a foreign body or blunt trauma. Signs of an open globe include penetrating lid injury, bullous conjunctival hemorrhage, blood in the anterior chamber (hyphema), peaked or deformed pupil, lens dislocation, vitreous hemorrhage, or decreased visual acuity. Management for transport includes protecting the affected eye with a rigid eye shield or cup, not a soft patch. Antiemetics and pain medication should be given for transport. Sometimes antitussives are also used to prevent any increase in intraocular pressure as the result of coughing.

Signs and Symptoms

Ocular Globe Rupture

- Penetrating lid injury
- Bullous conjunctival hemorrhage
- Blood in the anterior chamber (hyphema)
- Peaked or deformed pupil
- Lens dislocation
- Vitreous hemorrhage
- Decreased visual acuity

Transport Management

Ocular Globe Rupture

- Protect the affected eye with a rigid eye shield or cup.
- Administer antiemetics and pain medication.
- If necessary, administer an antitussive to prevent increased intraocular pressure as the result of coughing.

Ocular Avulsion

Enucleation of the eyeball from the eye socket is possible from a trauma **Figure 10-30**. Multiple facial fractures may increase the chance of this injury. The globe may actually be hanging from the optic nerve. Despite the dramatic presentation, loss of vision is not inevitable. Proper care includes protecting the eye from further trauma in transit by using a protective cup or other rigid protective device with gauze padding.

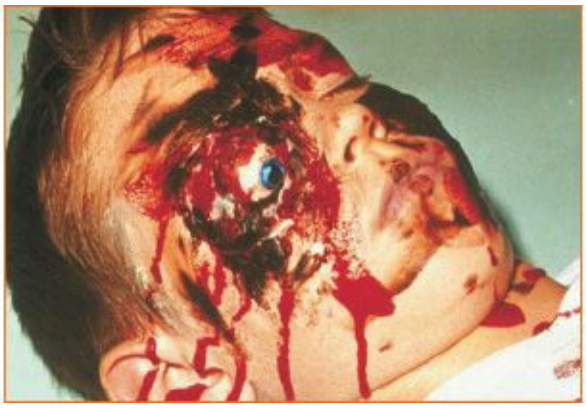


Figure 10-30 Ocular avulsion.

Transport Management

Ocular Avulsion

- Protect the eye from further trauma with a protective cup or other rigid protective device with gauze padding.

Traumatic Retinal Detachment

In the past, retinal detachment uniformly led to blindness. Now, rapid diagnosis and surgery is truly sight saving. Retinal detachment refers to separation of the inner layers of the retina from the pigmented epithelium. There are a number of causes besides trauma. Other causes include diabetic retinopathy and sickle cell disease. Initially, the patient complains of the sensation of flashing light often accompanied by a shower of floaters and vision loss. The vision loss may be described as filmy, cloudy, irregular, curtain-like, or wavy. Patients may also complain of black spots, especially in the center of the visual field, or spiderweb-like vision. It is imperative to avoid pressure to the globe. A rigid metal eye shield should be used. Do not use soft patches. Although some types of retinal detachment are treated medically, most require surgery within 24 hours.

Signs and Symptoms

Traumatic Retinal Detachment

- Sensation of flashing light, often accompanied by a shower of floaters and vision loss
- Black spots, especially in the center of the visual field
- Spiderweb-like vision

Transport Management

Traumatic Retinal Detachment

- Avoid pressure to the globe.
- Protect the eye with a rigid metal eye shield.

Signs and Symptoms

Mandibular Fracture or Dislocation

- Crepitus
- Trismus
- Swelling
- Patient complaint that the jaw "doesn't feel right" (malocclusion)

Transport Management

Mandibular Fracture or Dislocation

- If the patient's jaw is wired shut, have wire cutters available to open the jaw in case an emergency airway is needed.
- Have nasotracheal intubation skills and equipment available.
- Be prepared to make emergency surgical access.

Mandibular Fracture and Dislocation

The mandible is the only movable bone in the skull and makes up the lower third of the face. Because of its U shape, single blows can cause multiple fractures anywhere around its length. It can also become anteriorly dislocated, causing the mouth to be locked in an open position. Signs of fracture include crepitus, trismus, swelling, and patient complaints that the jaw does not feel right (malocclusion). Treatment is usually not necessary on a scene call, but transfers to rehabilitation may involve a patient with a jaw wired shut. Provision must be made for emergency airway during such transfers. This may involve having wire cutters available to open the jaw or nasotracheal intubation skills and equipment, and being prepared for emergency surgical access.

Dental Avulsion

Avulsed teeth have a good chance of reimplantation if replaced within an hour. Until that time, the tooth or teeth must be handled carefully.

Guidelines from the American Dental Association include the following:

- Never place an avulsed tooth in anything that can dry or crush the outside of the tooth.
- Do not handle the tooth roughly. Do not rinse it off or rub, scrape, or disinfect the outside of the tooth.
- Place the tooth in a soft transport device, preferably in Hank's solution (a pH-balanced, isotonic, glucose, calcium, magnesium solution). Do not use tap water. Some recommendations suggest whole milk as a second-best solution to Hank's solution. A third choice is saline, but only for less than an hour.

Neck Injuries

The critical structures running through the neck are relatively unprotected and, therefore, susceptible to many types of injuries. In addition to critical airway and vascular structures, the possibility of unstable cervical spine and cord injury must always be considered. For scene calls, selective spinal

immobilization criteria have become standard. If a patient has a cervical collar in place at a sending facility and the sending physician is unwilling to clear the cervical spine, the CCTP must consider full reimmobilization prior to transport. Patients with neck injury should always be considered difficult airway patients. Securing the airway with endotracheal intubation should be considered prior to most transports. Deterioration of the patient's airway in the back of a transport vehicle with limited space and personnel can be disastrous. Special airway considerations in the presence of neck injury include bleeding into the airway field, expanding hematomas, and tracheal disruption.

For neck trauma resulting in hoarseness accompanied by skin lacerations, ecchymosis, tenderness, subcutaneous emphysema, and/or stridor, an immediate otolaryngology consult is recommended. This may be the reason for transport to a larger hospital. Humidified oxygen, inhaled corticosteroids, and/or nebulized epinephrine may be ordered. Wounds may need to be dressed with occlusive dressings. If the wound is to the lower neck or clavicular area, vascular access may need to be achieved in the lower extremity as the result of compromised drainage from the arms.

Laryngotracheal Injuries

Laryngotracheal injuries represent less than 1% of all traumatic injuries. Most are in the area of the cervical trachea. Direct blunt trauma is the most common. Examples include a steering wheel to the extended throat, hanging, strangulation, and "clothesline" injury during sports such as mountain biking or snowmobiling. Penetrating laryngotracheal injuries are 10% of all penetrating neck injuries. Bubbling from a neck wound and subcutaneous air, along with dysphonia, dyspnea, stridor, visible wounds, and swelling, are all signs of laryngotracheal injury. These injuries can appear stable for a time, followed by rapid and catastrophic deterioration.

The most severe cases are laryngotracheal disruptions. It is vital that paralytics and muscle relaxants not be used. The only support for the trachea may be the supporting musculature. If the patient is paralyzed, the trachea may retract into the chest, making it impossible to ventilate the patient. If the trachea is visible through the neck wall, the endotracheal tube should simply be passed through the neck into the trachea.

Signs and Symptoms

Laryngotracheal Injury

- Bubbling from a neck wound
- Subcutaneous air
- Dysphonia
- Dyspnea
- Stridor
- Visible wounds
- Swelling

Transport Management

Laryngotracheal Injury

- Airway management with careful endotracheal intubation

- Occlusive dressing for open neck wounds
- Spinal precautions
- Transport to a trauma center

Thyroid Injuries

The thyroid gland is very vascular, and direct trauma to the front of the neck can cause hematomas of sufficient size to impinge on the airway. If surgical airway management is required, the location may need to be inferior to the usual site and inferior to the thyroid gland.

There are multiple case reports of **thyrotoxicosis** (thyroid storm, an excess of thyroid hormones) after trauma, including strangulation and direct blows to the anterior neck, shock (hypotension), or surgical procedures involving the thyroid. Thyroid storm has a 20% to 30% mortality rate. Thyroid storm causes a hypermetabolic crisis, including tachycardia over 140 beats/min, hyperthermia (sometimes with a temperature >103.9°F), coma with agitation, nausea, vomiting, diarrhea, and unexplained jaundice and pulmonary edema. Definitive diagnosis usually includes an elevated thyroxine level (normal level, 5.5 to 12.5 µg/dL). Treatment may include a beta-blocker such as an esmolol drip, sedation, and cooling for transport.

Signs and Symptoms

Thyrotoxicosis (Thyroid Storm)

- Tachycardia greater than 140 beats/min
- Hyperthermia (sometimes a temperature >103.9°F)
- Coma with agitation
- Nausea
- Vomiting
- Diarrhea
- Unexplained jaundice and pulmonary edema
- Elevated thyroxine level

Differential Diagnosis

Thyrotoxicosis (Thyroid Storm)

- Head injury
- Sepsis
- Toxic ingestion

Transport Management

Thyrotoxicosis (Thyroid Storm)

- Administer a beta-blocker (eg, an esmolol drip).
- Administer a sedative.

- Implement cooling measures.

Vascular Neck Injuries

Injury to the carotid, subclavian, and vertebral arteries and external and internal jugular veins can produce rapid exsanguination, hematoma formation, or embolization of air. Vascular neck injury accounts for one fourth of all penetrating neck trauma. Mortality may be as high as half of these patients. Many of the complications of these injuries do not occur for days to weeks and involve thromboemboli causing neurologic problems. Because of the possibility of venous air embolism, direct pressure with an occlusive dressing should be applied while the patient is kept in the Trendelenburg position. Keep in mind that the Trendelenburg position may lead to respiratory compromise and that ventilatory assistance may be needed.

Transport Management

Vascular Neck Injury

- Apply direct pressure to the injury with an occlusive dressing.
- Keep the patient in the Trendelenburg position.

Abdominal Trauma

Traumatic injuries to the abdomen will be some of the most difficult injuries for the CCTP to recognize. Whether providing care at a trauma scene or transferring a critical patient from the emergency department to a trauma center, it is important to document an accurate history and physical exam for later comparison. At a sending facility, it is usually worthwhile to interview the crew that brought the patient from the field. With interfacility critical care transport calls, always try to review all studies that have been done, including labs, radiographs, ultrasound (FAST exam), computed tomography scans, and, less frequently performed, diagnostic peritoneal lavage.

The abdominal cavity is an extremely large space that is home to many different vital organs. In many instances, your patient will experience blunt or penetrating trauma to the abdominal cavity, and signs or symptoms of injury may be very difficult to observe until shock becomes obvious. Because of the size of the abdominal cavity, a significant amount of blood can collect within the cavity and go unnoticed. Unlike other vital organs in the body, those in the abdominal cavity have very little protection, which makes them that much more susceptible to injury.

The following organs, structures, and vessels are located within the abdomen, and, if injured, can cause potential harm to your patient:

- Spleen
- Liver
- Kidneys
- Aorta
- Bladder
- Gallbladder
- Small and large intestines
- Pancreas

- Stomach

Blunt or penetrating trauma to many of these organs can cause a life-threatening hemorrhage and serious organ damage. It is important for the CCTP to focus attention on the mechanism of injury, instead of the outward signs of trauma found on assessment. Injuries to any of the previously listed organs may not readily present with outward signs and symptoms. This makes it that much more important for the CCTP to perform a detailed assessment and a thorough ongoing assessment during the transport, looking for subtle changes that might indicate a progressing condition. If a significant injury is suspected or confirmed, the most definitive treatment that the CCTP can provide is rapid transport to the trauma center, so that surgical intervention can be performed if needed. Once the CCTP recognizes the need for immediate transport to the trauma center, his or her job is to stabilize and maintain the patient's hemodynamic status as efficiently as possible until arrival at the trauma center. This task will be difficult and may require the administration of a crystalloid solution, blood products, or even vaso-active medications in the more severe cases. Grey-Turner's sign **Figure 10-31** and Cullen's sign **Figure 10-32** are indications of internal abdominal bleeding.

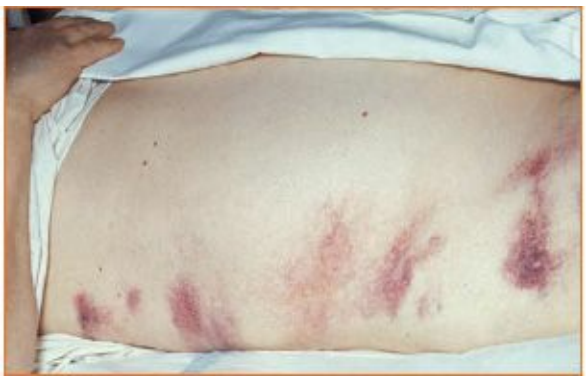


Figure 10-31 Grey-Turner's sign.

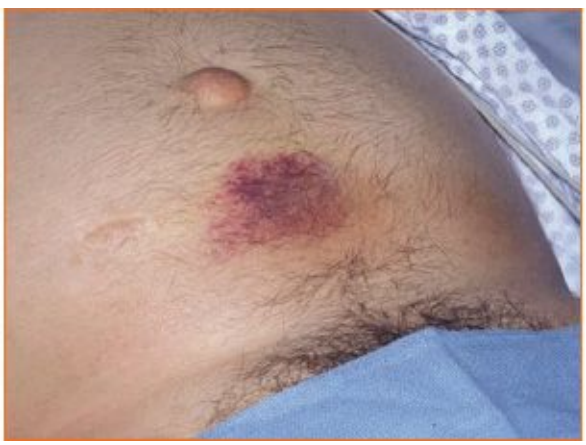


Figure 10-32 Cullen's sign.

Abdominal trauma can be classified into two different categories: blunt and penetrating trauma. Blunt trauma occurs when any type of external force is placed on the abdominal cavity, such as a fall, automobile crash, or motorcycle crash. Penetrating trauma can involve many different objects, such as knives, guns, tree limbs, or metal rods. One of the least suspected culprits in the blunt trauma classification is the seatbelt. The seatbelt has significantly decreased the incidence of morbidity and mortality related to automobile crashes, but continues to cause less noticeable abdominal injuries. Penetrating injuries will be very difficult for the CCTP to assess because the necessary information is often not available (eg, the size of the knife, the length of the blade, and whether it was jagged or straight-

edged). In many instances, even if you know this information, it will still be very difficult to rapidly identify what structures inside the abdominal cavity were injured without the assistance of imaging studies.

When beginning the physical assessment of the patient, the CCTP should try to ascertain as much about the events leading up to the injury as possible. Gathering more information will make diagnosing and treating the problem that much quicker and oftentimes simpler. The physical exam performed to rapidly identify blood loss is the most important task. When completing the physical exam, remember to always inspect, auscultate, and palpate, in that order. The primary assessment needs to be completed at the bedside, before beginning transport. It will be very difficult to obtain an accurate assessment in the back of a moving helicopter or ambulance if you choose to wait that long. This assessment must be repeated frequently during the transport, watching for changes that require immediate intervention. The CCTP will need to monitor the patient's level of consciousness, heart rate, and blood pressure, watching for changes that would indicate a progressively worsening condition.

Patients who experience massive amounts of blood loss may compensate initially, but they will eventually begin to present with signs of shock. Signs and symptoms to be constantly looking for include:

- Altered mental status
- Tachycardia
- Absence of palpable pulses
- Pale, moist, and mottled skin
- Poor peripheral perfusion
- Hypotension

The assessment should not be limited to these symptoms; however, they are some of the most common occurrences related to hypovolemic shock. Regardless of your findings during the initial assessment, all patients suspected of having abdominal injuries should be transported on high-flow oxygen with fluid administration. A minimum of two lines (as with most critical care transport patients) should be achieved, and central lines may need to be considered. CVP and arterial pressure lines are a very good idea because of the inaccuracy of noninvasive blood pressure monitoring in transit, especially in low blood pressure states. However, permissive hypotension should also be considered, especially if an aneurysm of a great vessel is suspected. The patient's blood pressure should be maintained at 90 mm Hg systolic (±10 mm Hg) or 65 mm Hg MAP. A bolus of crystalloid should be given in 250-mL to 500-mL amounts titrated to blood pressure, MAP, or CVP. The more severe cases may need aggressive airway management and blood administration; most transport programs carry packed red blood cells (O negative) for emergency situations. To prevent hemodilution, protocols should be written that allow the administration of human blood if a large volume of fluids is deemed necessary. Many new fluids with oxygen-carrying capacity are in the final stage of research and will begin to show up on the market. Except in cases of suspected urethral injury, all patients with serious abdominal trauma will, in addition to the vascular access, need to have a Foley placed prior to transport. Completing this step prior to transport will help the CCTP evaluate shock by evaluating the amount of urine output. In addition, the presence of frank blood will indicate renal system damage. Patients should be kept warm, and open abdominal wounds should be covered with sterile dressings and then covered with an occlusive dressing to prevent evaporative cooling.

Signs and Symptoms

Abdominal Injury

- Altered mental status
- Tachycardia
- Absence of palpable pulses
- Pale, moist, and mottled skin
- Poor peripheral perfusion
- Hypotension

Transport Management

Abdominal Injury

- Administer high-flow oxygen and fluids.
- Establish at least two IV lines; consider placement of central lines.
- Keep the patient's blood pressure at 90 mm Hg systolic or 65 mm Hg MAP.
- Administer a bolus of crystalloid in 250- to 500-mL amounts titrated to blood pressure, MAP, or CVP.
- If necessary, secure the airway.
- If necessary, administer blood products.
- Except in cases of suspected urethral injury, insert an indwelling catheter prior to transport.
- Keep the patient warm.
- Cover open abdominal wounds with sterile dressings than cover with an occlusive dressing to prevent evaporative cooling.

Hollow vs Solid Organ Injury

Hollow organs primarily leak their contents when injured, whereas solid organs bleed. Treatment should be oriented toward treating shock. Solid organs include the liver in the upper right quadrant, the spleen in the upper left quadrant, and the pancreas and the kidneys in the retroperitoneal space. The hollow organs include the stomach in the upper left quadrant, the intestines in all quadrants, and the gallbladder under the liver in the upper right quadrant. Hollow organs in the midline include the urinary bladder and ureters, and the uterus and great vessels of the descending (abdominal) aorta and the inferior vena cava. The primary concern with hollow organ injury is peritonitis.

Spleen

About 5% of circulating blood filters through the spleen every minute. This organ gets direct vascular supply from the aorta, and its drainage goes directly to the inferior vena cava. The spleen is the most commonly injured organ in the abdominal cavity. It can even be injured by what seems to be minor trauma. Young adults and patients with sickle cell disease are at particular risk, as a result of the spleen being relatively larger. Located in the upper left quadrant of the abdomen, the spleen is partially protected by the lower rib cage, but lacks a strong exterior capsule. Injuries to the spleen that do not cause a disruption to the organ itself will likely only cause minimal amounts of bleeding. More significant bleeding may occur if the exterior capsule is damaged. As mentioned previously, the presence of normal vital signs does not indicate the absence of an injury. Especially with injuries involving the spleen, the patient may only complain of pain on primary assessment, and symptoms of shock may develop later. In

some cases, symptoms arise as late as 2 to 3 weeks after the original trauma. A helpful tool for recognizing a splenic injury through referred pain is called **Kehr's sign**. This sign presents as pain in the shoulder as a result of the presence of blood or other irritants in the peritoneal cavity. Pain in the left shoulder (Kehr's sign) is considered a classic symptom of a ruptured spleen, but it also may result from any free blood irritating the diaphragm. The same phenomenon also occurs on the right side with irritation to the liver or gallbladder. Failure to recognize, understand, and treat the signs and symptoms of hypovolemic shock related to internal hemorrhage will rapidly lead to death. However, many spleen injuries are often able to be managed through careful observation, because the spleen sometimes heals over time and invasive interventions are not needed.

Signs and Symptoms

Spleen Injury

- Pain in the area of the spleen
- Kehr's sign: pain in the shoulder

Transport Management

Spleen Injury

- Treat the patient for signs and symptoms of hypovolemic shock.

Liver

The largest organ in the abdominal cavity, the liver is also the most vascular organ, and receives 25% of the cardiac output. The liver does have a little more protection than the spleen, because it is surrounded by many different structures of the thorax, including ligaments that stabilize the organ and keep it from being jostled around during impact. However, the ligament in front of the liver (ligamentum teres) can slice the liver in sudden deceleration. Additionally, the liver's close proximity to the lower rib cage makes it susceptible to injury after a rib fracture occurs. Liver injuries will cause pain in the right upper quadrant, along with Kehr's sign in the right shoulder as blood accumulates around the diaphragm.

In the past, the majority of liver injuries were surgically repaired. Studies have shown that many of the less severe liver injuries can endure 1 or 2 days of observation before making a decision to surgically repair the organ. In most instances, simple observation is the only intervention that is needed, because the liver heals over time and invasive interventions are not needed. This is not the case for severe liver injuries, because surgery is likely the only intervention that will prevent death.

Large and Small Intestines

The intestines are hollow organs that occupy much of the space within the abdominal cavity. Penetrating trauma is the most common cause of injury to the intestines, because they lie very close to the surface of the abdomen. When objects such as knives or bullets penetrate the abdominal wall, injury to the hollow intestine is likely. Although penetrating trauma is the most common, blunt trauma to the abdominal wall can also cause injury to the small bowel. In this particular situation, the most common culprit is the seatbelt, because the lapbelt lies along the lower quadrant of the abdominal cavity **Figure 10-33**. The patient would likely present with an ecchymotic area across the lower portion of the abdomen (seatbelt

sign), which should necessitate assessment for internal injuries.

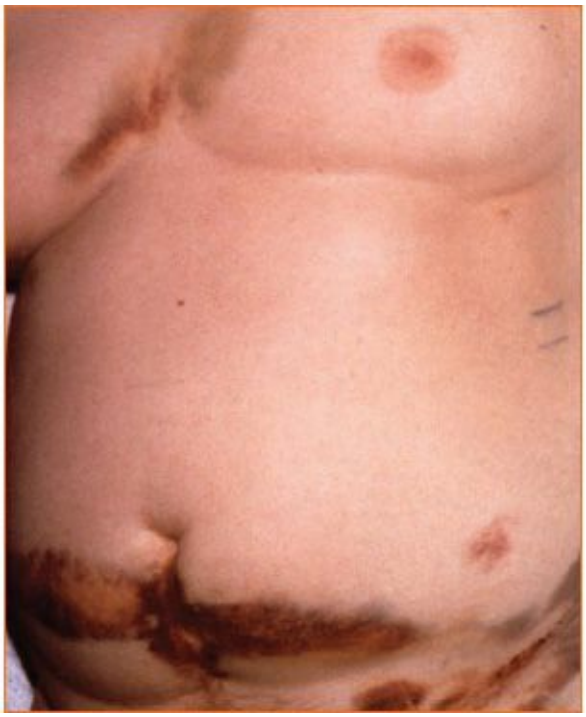


Figure 10-33 Seatbelt sign.

Signs and Symptoms

Intestinal Injury

- Ecchymotic area across the lower portion of the abdomen (seatbelt sign)
- Presence of pain with palpation to the abdomen
- Guarding
- Abdominal distention
- Rebound tenderness
- Vomiting

Transport Management

Intestinal Injury

- Maintain a high index of suspicion for occult injury.
- Establish two large-bore IVs.
- Consider other injuries, such as chest, neck, and lumbar spine injuries.
- Provide rapid transport.

The lack of visible injury should not be used to exclude internal injury. Once again, the CCTP will rely on his or her assessment skills to detect abnormalities. In some cases, the presence of pain, with palpation to the abdomen, might be the only indicator of injury. In addition to the pain with palpation, other indicators, such as guarding, abdominal distention, rebound tenderness, and vomiting, may be

present.

The large intestine or colon is the most frequent area to be injured by penetrating trauma. Injury to the large intestine or colon was found in 96% of penetrating traumas in one study, of which gunshots accounted for more than 90%. Ruptures of the colon or rectum are particularly prone to sepsis and peritonitis.

Stomach

Damage to the stomach from trauma is rare. The most common blunt trauma occurs when the stomach is caught against the steering wheel when full. The stomach can also be damaged in penetrating injuries.

Duodenum

The duodenum is a retroperitoneal organ and, therefore, is well protected. If ruptured because of its retroperitoneal location, it may not produce symptoms, but as a result of its close proximity to multiple organs, it is never injured by itself. This injury should be suspected in children who are thrown off a bicycle and strike their abdomen on the handlebars.

Jejunum and Ileum

The jejunum and ileum are generally victims of penetrating trauma. Approximately 80% of gunshot wounds to the abdomen and 30% of stab wounds lacerate these organs. Because the jejunum and ileum are anchored to the abdominal wall by the ligament of Trietz, the mesentery and its vasculature often is torn and causes hemorrhage.

Vascular Injuries

Unfortunately, injuries that occur to the vessels of the abdomen are usually life threatening. The abdominal aorta is a large vessel that carries blood away from the heart, supplies all the abdominal organs, and continues its distribution of blood and oxygen to the toes. Severe blunt trauma may cause disruption of these vessels, but the most common cause of vascular injury is, once again, penetrating trauma. In instances of blunt trauma, the shearing force at impact causes the vessels to break away from their branches, thus allowing blood to freely flow into the abdominal cavity. The major blood vessels in the abdomen that have the potential for being injured are:

- Aorta
- Inferior vena cava
- Renal artery
- Mesenteric artery
- Iliac artery

Management

Bleeding from an injury to any of the major abdominal vessels may occur rapidly, and signs of shock may not even be noticed. Aggressive management of these patients is required, but may not be very helpful, because arterial injuries will cause exsanguination unless immediate surgery is available. Therefore, rapid transport to a hospital capable of immediate surgery may be the best possible treatment. The abdominal cavity is also home to several large veins that will cause the body to lose large amounts of blood, but not at the rapid pace of an artery. Bleeding into the abdominal cavity may slowly occur without obvious signs or symptoms.

The treatment is similar to the other abdominal injuries. Airway patency is the key, because an altered mental status is likely with patients who are experiencing massive blood loss. Immediate steps should be taken to endotracheally intubate to secure the airway if necessary. Attention should be focused

on treating the hypovolemic shock and transporting to a center capable of trauma surgery. In the initial treatment of this patient, aggressive fluid resuscitation is the key to maintain a systolic blood pressure of at least 80 mm Hg but not higher than 90 mm Hg, with progression toward blood products as soon as possible. If the patient does survive the transport, the surgical team needs to intervene as soon as possible so they can attempt to surgically repair the disturbed vessel. Even if the patient makes it to surgery, several serious complications can occur after the surgery, such as disseminated intravascular coagulation, continued bleeding, and thrombosis formation. All of these conditions are life threatening, and will be treated as such by the team caring for the patient.

Signs and Symptoms

Abdominal Vascular Injury

- Potentially none
- Altered mental status
- Hypovolemic shock

Transport Management

Abdominal Vascular Injury

- Maintain airway patency; insert an endotracheal tube if necessary.
- Administer aggressive fluid resuscitation; maintain a systolic blood pressure of at least 80 mm Hg but not higher than 90 mm Hg.
- Administer blood products, if available.
- Transport the patient immediately to a center capable of trauma surgery.

Pelvic Trauma

Pelvic Fracture

Pelvic fractures are the result of motor vehicle trauma (approximately 60% to 70% of the time), pedestrian vs motor vehicle (12% to 18%), motorcycle crashes (5% to 10%), and falls (up to 10%). A pelvic fracture results from significant force. Because of the force involved, the presence of a pelvic fracture should alert the CCTP to the possible existence of other injuries. Mortality in one study of blunt trauma pelvic fractures was as high as 50%. Most of the threat to life comes from internal hemorrhage. The pelvis is made up of three paired bones: the ileum, ischium, and pubis. Together, they are called the innominate bones and are connected posteriorly by the sacrum and coccyx. The very rich blood supply comes from the left and right iliac arteries. The veins in the pelvis are without valves and are adhered to the pelvic wall. Because they are thin, are easily torn, and can flow in either direction, fractures can result in catastrophic hemorrhage. Subjective assessment includes a good history of the mechanism of injury, location, and radiation of pain, as well as the presence of hematuria and the possibility of pregnancy. Objective examination should include observing for the rotation or uneven height of the iliac crests and uneven length of the legs. Palpation of the posterior aspect of the pelvic ring should not elicit tenderness. Gentle lateral compression and inward compression should not result in pain, crepitus, or movement. This test should be done carefully and stopped if any instability is felt and not repeated.

The pelvis also houses the female reproductive organs and the bladder in both sexes. When full, the bladder can rise above the pelvic rim and become victim to seatbelt injury causing rupture. The uterus, ovaries, and fallopian tubes are well protected in the nonpregnant patient. Injuries to these organs are usually found later using computed tomography.

Management of pelvic injury depends on the type of fracture. Field treatment consists of splinting and treatment for shock. Soft-tissue damage to the genitalia can be managed like any other soft-tissue injury. Packaging for transport of suspected or confirmed pelvic fracture should involve immobilization on a scoop stretcher or backboard or use of a pelvic binder **Figure 10-34**.

Signs and Symptoms

Pelvic or Open-Book Fracture

- Rotation or uneven height of the iliac crests
- Uneven length of the legs
- Tenderness upon palpation of the posterior aspect of the pelvic ring
- Pain, crepitus, or movement upon gentle lateral compression and inward compression

Figure 10-34 A pelvic binder.

Transport Management

Pelvic or Open-Book Fracture

- Splint the fracture.
- Treat the patient for shock.
- Manage any soft-tissue damage to the genitalia.
- Package the patient for transport using immobilization on a scoop stretcher or backboard; alternatively, use a pelvic binder.

Open-Book Fracture

Open-book fractures, a subset of pelvic fractures, account for about 15% of pelvic fractures and involve the loss of stability of the pelvic ring. They result from anterior-posterior compressive forces separating the pelvis at the symphysis pubis. The greatest danger is from hemorrhage. Some of these fractures have gone undiscovered until the pneumatic antishock garment was deflated in the emergency department. Reduction of these fractures decreases the bleeding. Assessment and treatment are the same as for any pelvic fracture, and patients should be transported on a well-padded backboard or scoop. Pelvic binders

or even pneumatic antishock garments have also been successfully used.

Extremity Trauma

Musculoskeletal Injuries

Musculoskeletal injuries are rarely life threatening. They include fractures, sprains, strains, dislocations, muscular contusions, and compartment and crush syndromes. Often dramatic, they may be an indicator of the amount of energy transferred to the body and the likelihood of additional injury. However, the CCTP must not let dramatic fractures distract from a proper primary and secondary assessment. Initial immobilization on the scene can be accomplished by using a backboard as a whole-body splint to accelerate time off the scene for multiple trauma patients. On interfacility transports, there is usually time to make sure individual fractures are properly splinted. In both cases, adequate pain control should be used if blood pressure allows. All musculoskeletal injuries should be assessed for the six Ps—pain, pallor, pulselessness, paresthesia, paralysis, and pressure—before and after any manipulation. This should be documented on the patient's chart, and pulse locations should be marked with a felt-tip or skin marker on the patient to speed rechecking during transport.

Fractures

A break in the continuity of a bone is a fracture, and may be open or closed. Regardless of whether a fracture is open or closed, the CCTP must make prevention of further injury due to movement with pain assessment and management a priority. The CCTP should use appropriate analgesic medications as permitted in system protocols to reduce the pain and suffering of the patient.

In a closed fracture, the skin over the bone remains intact, thus reducing the probability of infection. However, closed fractures can be as dangerous as open fractures due to involvement of internal bleeding into the tissues. Depending on the anatomic area of the body where a closed fracture occurs, it could result in a large amount of blood loss before enough pressure is internally achieved to tamponade the bleeding. For example, a closed fracture of one femur could easily result in the loss of 1 L of blood, whereas bilateral femur fractures could result in the loss of 2 L of blood **Table 10-5**. Multiple fractures of trauma patients can cause massive internal hemorrhage and even death, with no external bleeding noted.

In an open fracture, the skin over the bone is broken, thus adding the potential danger of infection, which could lead to sepsis and death. Open fractures typically result from high-energy injuries, and thus have the potential for more blood loss than closed fractures.

Clinical signs and symptoms of fractures include pain, swelling, deformity, rigidity, shortening, ecchymosis, guarding, and crepitus. The patient could have one or more of the presented signs and symptoms. Management of isolated fractures is as follows:

- Maintain an open airway and apply high-flow oxygen if necessary.
- Assess pulses and motor and sensory function distal to the fracture site.
- Control bleeding with direct pressure and pressure dressings; if that is not effective after adequate time, then a tourniquet should be used. Apply a sterile dressing to any exposed bone or tissue.
- Initiate IV fluids to maintain peripheral perfusion.
- Consider the administration of analgesic medication titrated to pain relief prior to manipulating the fracture if the patient is hemodynamically stable.
- Splint in a normal anatomic position with an appropriate splint while maintaining circulation.

TABLE 10-5 Estimated Blood Loss by Fracture Type

Estimated Potential Blood Loss (mL)	Fracture Type
125 each	Rib
250–500	Radius or ulna
500–750	Humerus
500–1,000	Tibia or fibula
1,000–2,000	Femur
1,000 to massive	Pelvis

Signs and Symptoms

Extremity Fractures

- Pain
- Swelling
- Deformity
- Rigidity
- Shortening
- Ecchymosis
- Guarding
- Crepitus

Differential Diagnosis

Extremity Fractures

- Fracture
- Sprain
- Contusion
- Compartment syndrome

Transport Management

Extremity Fractures

- Maintain an open airway; apply high-flow oxygen if necessary.
- Assess pulses and motor and sensory function distal to the fracture site.
- Control bleeding with direct pressure and pressure dressings; if that is not effective after adequate time, apply a tourniquet.
- Apply a sterile dressing to any exposed bone or tissue.
- Initiate IV fluids.
- Consider administration of analgesic medication.
- Splint the extremity in its normal anatomic position with an appropriate splint while maintaining

circulation.
- Reassess pulses and motor and sensory function distal to the fracture site after application of the splint.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.
- Splint femur fractures with a traction splint.

- Reassess pulses and motor and sensory function distal to the fracture site after application of the splint.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.
- Femur fractures should be splinted using a traction splint, because this reduces the muscle spasms and pain and bleeding.

Femur Fracture

The femur is the largest bone in the body, surrounded by some of the strongest muscles. When the femur is broken, the muscles contract, causing sharp bone ends to override and damage soft tissue. More importantly, the slack muscle can provide a large space for the collection of blood. Generally, femur fractures should be considered to account for loss of at least 1 L of blood from circulation.

The traction splint pulls the slack muscles taut again, tamponading the bleeding. When first invented during the Crimean war, the traction splint reduced mortality from femur fracture secondary to gunshot from 80% to 20%. However, there are a number of contraindications and dangers with use of traction splints. The contraindications include hip and pelvic fractures, knee injury, and ankle damage. The dangers include damage or worsening damage to the neurovascular bundle of the thigh; this includes the sciatic and femoral nerves and the vasculature of the area.

Vertebral Fracture

Trauma is the leading cause of injury to the vertebral column. About 20,000 new spinal cord injuries occur in a year in the United States, with nearly half as a result of motor vehicle trauma, followed by intentional violence, falls, and sports injuries. Vertebral fractures can be stable or unstable. Unstable injuries can result in permanent devastating neurologic deficits. Any movement during transport can transform an unstable fracture without neurologic deficit into a life-long disability. The current practice is to fully immobilize the spine for transport. This often means placing the patient in the sending facility on a backboard again. The use of traditional rigid backboards for immobilization has been associated with the development of pain and skin breakdown when patients are confined for more than 30 minutes. Adequate padding or use of vacuum mattresses may reduce this problem. Acute spinal injury is classified as stable or unstable and by the mechanism of injury. Flexion, flexion with rotation, extension, and compression are all mechanisms of injury. Some fractures also have specific names, such as clay shoveler's fracture or hangman's fracture. Secondary injury can be the result of hypoxia, hypoglycemia, hypotension, or hyperthermia, as well as mishandling during transport. All of these conditions must be assessed for and treated if found so they do not exacerbate a cord injury secondary to vertebral fracture.

Cervical Fracture

The most devastating vertebral fractures occur in the seven cervical vertebrae. Control of the entire body, including cardiorespiratory functions, descends through the cervical vertebra. The phrenic nerve controlling the diaphragm arises from C3, C4, and C5. Damage to vertebrae in this area may compromise respiratory effort. Patients being transported from one facility to another who have not definitively had their cervical spine cleared should be completely immobilized. Advance planning for airway management

and prevention of aspiration, especially in small aircraft, must be undertaken prior to transport.

Thoracic Fracture

Thoracic fractures can lead to a loss of innervation of the intercostal muscles, leading to respiratory insufficiency. Sympathetic nervous control arises from the thoracic and high lumbar spinal cord. Although rare, a condition called "spinal shock" can affect the thoracic area or higher. In this type of shock, hypotension is accompanied by bradycardia instead of tachycardia. Pulse pressure will be normal or widened, instead of narrowed as in hypovolemia. The area above the fracture will be pale and cool, but the area below the fracture will be warm. Treatment should be fluids and pressors to maintain MAP in a range of 80 to 90 mm Hg. Transport considerations should match the stabilization precautions for cervical vertebra.

Lumbar Spine Fracture

The lumbar vertebrae are the largest vertebrae. However, they are not supported as the thoracic vertebrae are by the ribs and are, therefore, prone to injury. Jackknifing injuries, such as over a seatbelt, are frequently the mechanism of injury. Immobilization for transport is the appropriate treatment.

Humerus Fracture

The humerus, like the femur, is a vascular long bone. Blood loss from a humeral fracture may be significant. As much as 750 mL of blood may be lost from a single fracture. In addition, radial nerve injury is possible. During on-scene calls for multiple trauma patients, the humerus may simply be splinted to the body on a long board to facilitate rapid evacuation. For interfacility transports, it should be splinted carefully with attention to distal circulation and nerve function. It is a good practice to mark the location of pulses that are found prior to transport with a skin marker. This will allow for quick repeat assessment during transport. The absence of distal pulses or nerve function should receive immediate attention, which usually involves traction in anatomic alignment and manipulation.

Rib Fracture

Single rib fractures are rarely life threatening. They may cause pneumothorax, and torn intercostal arteries can lead to significant blood loss or hemothorax. Multiple rib fractures will negatively impact respiratory excursion even if there is no flail segment. To treat rib fractures, provide sufficient pain medication so that self-splinting does not interfere with respiration. Multiple rib fractures may require ventilatory assistance.

Nasal Fracture

Nasal fractures are important to the CCTP because of the concern for patency of the airway. Swelling as a result of nasal fractures can interfere with the nose as an air passage and foreclose use of this route for an artificial airway, such as nasopharyngeal or nasotracheal airways.

Dislocations

When a bone displaces from a joint, the condition is called a dislocation. When a body part exceeds its normal range of motion, a dislocation can occur. Until proven otherwise, all dislocations should be assumed to be coupled with a fracture. Although joint dislocations are not life threatening, they are true emergencies as a result of the potential for neurovascular compromise that could lead to amputation if not appropriately treated. The CCTP should use appropriate analgesic mediations as permitted in protocols in order to reduce the pain and suffering of the patient.

Clinical signs and symptoms of a dislocation include pain, swelling, distorted anatomy, shortening, ecchymosis, guarding or being "locked," and being unable to move. The patient could have one or more of

the presented signs and symptoms. Management of a dislocation is as follows:

Signs and Symptoms

Dislocations

- Pain
- Swelling
- Distorted anatomy
- Shortening
- Ecchymosis
- Guarding or being "locked"
- Inability to move

Differential Diagnosis

Dislocations

- Fracture
- Contusion

Transport Management

Dislocations

- Maintain an open airway; apply high-flow oxygen as needed.
- Initiate IV fluids.
- Assess pulses and motor and sensory function distal to the dislocation.
- Splint the dislocation in the position of comfort with an appropriate splint while maintaining circulation.
- Reassess pulses and motor and sensory function distal to the injured site after application of the splint.
- Administer analgesic medication titrated to pain relief and blood pressure.
- Monitor oxygen saturation, vital signs, and the cardiac monitor.

- Maintain an open airway and apply high-flow oxygen as needed.
- Initiate IV fluids to maintain peripheral perfusion.
- Assess pulses and motor and sensory function distal to the dislocation.
- Splint in the position of comfort with an appropriate splint while maintaining circulation.
- Reassess pulses and motor and sensory function distal to the fracture site after application of the splint.
- Administer analgesic medication titrated to pain relief and blood pressure. This may need to be done prior to splinting to facilitate placement of the splint.

- Monitor oxygen saturation, vital signs, and the cardiac monitor.

Subluxations

A subluxation is a partial dislocation of a joint. Sometimes also called sprains, subluxations can be graded from I to IV. A grade I sprain has minimal abnormal movement, and a grade IV is a dislocation.

Amputation

An amputation is the separation of a body part from the remainder of the body. Amputations can be life threatening and are often disabling. There is potential for hemorrhage, but usually bleeding can be controlled with direct pressure to the stump. If not a tourniquet, then a band at least 2″ wide, such as a blood pressure cuff, should be used. The CCTP should make every attempt to locate the part and transport it with the patient for possible reattachment. If located, the amputated part should be put into a plastic bag and then placed in ice water and transported with the patient.

Management

General management of fractures and trauma depends on the overall state of the patient. If the patient needs immediate resuscitation, airway, ventilation, control of blood loss, and fluid resuscitation must take priority. In cases of on-scene trauma calls, the trauma patient requires immediate resuscitation about 10% of the time. The priority in these cases is resuscitation and transport to surgical capability to stop internal bleeding. In such situations, the care of fractures is limited to stopping external bleeding and stabilization by splinting on a backboard to facilitate rapid transport. For the calls that do not require critical resuscitation, the priority shifts to pain control, assurance of distal neurovascular function, and careful splinting of each fracture. On interfacility critical care transports, similar priorities apply, although for stable patients the type of immobilization may be much more sophisticated.

Splinting

Splinting a fracture is done primarily to prevent further harm from movement of the bone ends, which could cause further soft-tissue injury, bleeding, and pain. The principles of splinting involve holding the bones in an anatomically correct position and preventing any movement. To get the bones into the anatomically correct position, tension needs to be applied while gently returning the limb to alignment. This should be stopped if there is any resistance or a significant change in pain level. Traditional rigid splints must be well padded to prevent skin breakdown and pain at pressure points. Alternatively, vacuum splints have been found to be effective and comfortable, but, like older air splints, are affected by altitude changes. No matter what type of fixation splint is used, the entire bone with the adjacent joints must be held immobile in order for the splint to be effective. Distal neurovascular function should be documented before application and frequently during transport. Any reduction in function requires readjustment of the splint.

Casting

Casts made of plaster, or now more commonly fiberglass, are applied by an orthopaedic surgeon after acute swelling has gone down. This is usually at least 24 hours after the injury occurs. Swelling under a cast can cause compartment syndrome. Any patient with a cast who is transported by a CCTP should be evaluated for neurovascular function distal to the cast prior to transport and periodically during transport. It is recommended that the location where pulses are found be marked on the skin and documented in the patient care record.

Reduction/Realignment

Typically, reduction and realignment is not a priority of care in the critical patient. Resuscitation of cardiovascular status must always take priority. Usually, reductions are done by an orthopaedist as part of the resuscitation team or soon thereafter. Realignment becomes a priority if the neurovascular function distal to the injury is compromised. This is accomplished by applying gentle traction in the anatomic direction of the bone distal to the fracture while stabilizing the bone proximal to the break. Then the bone is brought into alignment or to a position in which maximal pulse is felt and then splinted in that direction.

External Fixation

External fixation of fractures has become common. Placed by an orthopaedic surgeon, external fixation uses external rods held to the bone with plates and screws. The apparatus is placed surgically. The external joints are adjusted over time as needed by the orthopaedist. The CCTP will from time to time transport patients with this device in place. Adequate padding and positioning is required for transport.

Internal Fixation

Internal fixation is the placement of rods such as Harrington rods with plates and screws in the operating room to hold fractures in place for healing. Later the rods may be removed or left in place permanently. Occasionally the rods work themselves out of position and may need surgical revision. Generally, this equipment is not of concern during transport.

Compartment Syndrome

In compartment syndrome, bleeding or swelling occurs within the nonstretchable fascia that divides each extremity into compartments and exceeds the amount of space available, thus impairing circulation and causing pain. The most common anatomic locations for compartment syndrome are the forearms and legs. However, any extremity and the buttocks can develop compartment syndrome. In one study, 69% of compartment syndrome cases were associated with fracture (half of the fractures were to the tibia), but burns, external compression from splints or bandages, and crushing injury can all lead to compartment syndrome. In the same study, 31% of patients had soft-tissue injuries without a fracture. Compartment syndrome usually develops over a period of hours.

Clinical signs and symptoms of compartment syndrome initially present as pain out of proportion for the evident injury, sometimes described as burning and paresthesia over the compartment site. Additional signs include tension of a muscle when not in use, loss of distal sensation or pulses, and extreme pain on extension or voluntary movement. The presence of pulses or Doppler blood flow does not rule out compartment syndrome. The signs are commonly referred to as the six Ps—pain, pallor, pulselessness, paresthesia, paralysis, and pressure. Sometimes a seventh P is included, poikilothermia (ie, affected extremities may be cold).

Management of compartment syndrome is as follows:

1. Maintain an open airway and apply high-flow oxygen as necessary.
2. Assess pulses and motor and sensory function.
3. Elevate the extremity above the heart.
4. Place ice packs over the extremity.
5. Initiate IV fluids to maintain peripheral perfusion.
6. Consider loosening the constrictive splint material and/or clothing.
7. Administer pain medication.
8. Monitor carefully for signs of, and be prepared to treat, rhabdomyolysis, hyperkalemia, and myoglobinuria.

For interfacility transport, there are several methods of monitoring actual compartment pressures invasively. In noncontracting muscle, the normal mean interstitial tissue pressure is close to 0 mm Hg. If this pressure becomes elevated to 30 mm Hg or more, small vessels in the tissue can become compressed, which results in reduced blood flow, ischemia, and pain. Pay close attention to compartment pressure and diastolic blood pressure **Figure 10-35**. It is considered an emergency if diastolic blood pressure drops below 30 mm Hg over compartment pressure. When compartment pressure exceeds diastolic pressure, the blood flow in the extremity is stopped. Untreated compartment syndrome–mediated ischemia of the muscles and nerves leads to eventual irreversible damage and death of the tissues within the compartment.

Figure 10-35 Compartment pressure monitoring devices.

Signs and Symptoms

Compartment Syndrome

- Pain
- Pallor
- Pulselessness
- Paresthesia
- Paralysis
- Pressure
- Poikilothermia (affected extremities may be cold)

Transport Management

Compartment Syndrome

- Maintain an open airway; apply high-flow oxygen as necessary.
- Assess pulses and motor and sensory function.
- Elevate the extremity above the heart.
- Place ice packs over the extremity.
- Initiate IV fluids to maintain peripheral perfusion.
- Consider loosening constrictive splint material and/or clothing.
- Administer pain medication.
- Monitor carefully for signs of, and be prepared to treat, rhabdomyolysis, hyperkalemia, and myoglobinuria.

Crush Injury and Rhabdomyolysis

Rhabdomyolysis is damage to the sarcolemma (muscle membrane) from any cause. This damage allows an influx of calcium and sodium, followed by water, to the cells and a release of myoglobin, aspartate transaminase, lactate, creatine kinase, potassium (K^+), uric acid, and phosphorus. The membrane damage also results in extracellular hypocalcemia and hyperkalemia. Hyperkalemia leads to cardiac arrhythmias. The release of myoglobin with hypovolemia and acidosis leads to the blockage of renal tubules, causing acute renal failure.

There are multiple etiologic causes of rhabdomyolysis, both traumatic and medically induced. The leading causes are exercise, alcohol, drugs, infections, trauma, compression, and seizures. The list of causes also includes metabolic myopathies, drugs and toxins (including cocaine and hallucinogens such as PCP and MDMA), infections, electrolyte abnormalities (including hypokalemia, hypophosphatemia, and both hypernatremia and hyponatremia), electrical current injuries, hypoxia (including carbon monoxide poisoning and sickle cell crisis), hyperthermia (including from heat stroke, neuroleptic malignant syndrome, and malignant hyperthermia), and idiopathic causes. Assessment must start with a high index of suspicion because of the many causes. Any patient who lies in one position without being able to move for several hours is at risk. Physical examination may reveal motor weakness, obvious evidence of trauma or compression, and sensory loss. All of these symptoms may be accompanied by evidence of dehydration and dark or tea-colored urine. The urine on a dipstick will show blood with only a few red blood cells on microscopic urine analysis, and a blood test will show significant creatine kinase levels of 5 times normal or higher. The upper normal level is 150 U/L. Sometimes, several hundred thousand units per liter may be present in rhabdomyolysis. Electrolytes will show hyperkalemia (> 5.5 mEq/L), with the expected ECG changes of tall pointed T waves with a narrow base that widens as K^+ increases, and then a flattening P wave. Hypocalcemia (< 4.3 mEq/L) may also be present, which will show on an ECG as a prolonged QT interval.

Signs and Symptoms

Rhabdomyolysis

- Motor weakness
- Obvious evidence of trauma or compression
- Sensory loss
- Dehydration
- Dark or tea-colored urine
- Blood with only a few red blood cells on microscopic urine analysis
- Significant elevations in creatine kinase (5 times normal or higher)
- Hyperkalemia (>5.5 mEq/L), plus ECG changes of tall, pointed T waves with a narrow base that widens as K^+ increases, and then a flattening P wave
- Hypocalcemia (>4.3 mEq/L), plus a prolonged QT interval on ECG

Transport Management

Rhabdomyolysis

- Administer 2 L (or more) of saline.
- Administer 1 mEq/kg of sodium bicarbonate.

- Place the patient on a heart monitor.
- If signs of elevated K^+ are seen, give calcium as often as every 4 minutes as a slow IV push.
- If IV or intraosseous access cannot be obtained, apply a tourniquet proximal to the entrapment.
- Insert an indwelling catheter, and give enough fluids to maintain output of 200 to 300 mL/h.
- Give enough bicarbonate to keep urine pH greater than 6.5, by adding 1 to 2 amps per 1 L saline given.
- Administer mannitol.
- On interfacility transfers, monitor electrolytes.

Management

When a CCTP is called to a scene for prolonged (longer than 4 hours) entrapment of a large muscle area, it is imperative that evaluation and treatment be started prior to the release of the entrapped patient. Crush syndrome is known as the smiling death. This is because extricated patients are shown happy and smiling until the released K^+ reaches the heart and sudden death ensues. Immediately prior to rescue, 2 L or more of saline (not potassium-containing lactated Ringer's solution) should be infused and 1 mEq/kg of sodium bicarbonate should be given. The patient should be on a heart monitor when released and if signs of elevated K^+ are seen, then calcium should also be given as often as every 5 minutes as a slow IV push. If IV or intraosseous access cannot be obtained, a tourniquet should be applied proximal to the entrapment to prevent the release of myoglobin and K^+ and the entry of fluid into the muscle mass. Once an indwelling catheter is placed, fluids should be given to maintain output of 200 to 300 mL/hr. Bicarbonate should be given to keep urine pH greater than 6.5. This can be done by adding 3 amps to a liter of 5% dextrose in water to maintain an isotonic solution. Mannitol is the diuretic of choice. It is both an osmotic diuretic, reducing swelling of the damaged muscle, while serving as an intravascular volume expander. It also dilates and flushes the renal tubules. Mannitol also is a free radical scavenger that reduces direct damage. Loop diuretics (furosemide) should be avoided because they acidify the urine. On interfacility transfers, the previously mentioned therapies should be guided by following electrolytes carefully. This includes a set immediately prior to transfer and, if available, use of bedside electrolyte monitoring during transport, such as with an I-STAT device **Figure 10-36**.

Figure 10-36 An I-STAT bedside electrolyte monitoring device for use during transport.

Pulseless Extremity

Although a traumatized pulseless extremity may be repositioned successfully to reestablish circulation, there are inherent dangers to this maneuver. It should be done with manual traction in anatomic alignment with the long bone. Traction should reduce and not cause pain if it is done in the correct anatomic direction. If the injury is a dislocation rather than a fracture, analgesia and benzodiazepines may help

relax the muscles to facilitate the repositioning. Position the extremity to maximize the strength of the pulse and splint in position. Mark the skin where pulses are obtained to make reassessment faster.

Pharmacologic Management

Nonsteroidal Anti-Inflammatory Drugs

Although nonsteroidal anti-inflammatory drugs are used in stable patients to control pain and reduce inflammation, during the acute phase of resuscitation and transport of critical patients, opiates such as fentanyl should be used. Ketorolac can be used for moderate pain if there is no bleeding and if hypovolemia is not an issue.

Opiate Analgesics

During critical care transport, opiates are the mainstay of pain control. Proper amelioration of pain should be a primary concern of the CCTP. All opiates have side effects. The most serious in the short term can be respiratory depression. Hypotension and nausea or vomiting can also be side effects of opiates as a result of the release of histamine. Fentanyl is rapidly becoming the most widely used opiate for critical care use because it does not release histamine. These side effects can be controlled even with use of traditional morphine if it is given slowly and with enough fluids.

Antibiotics

Early use of antibiotics is common practice with trauma. Especially in patients with open fractures and body cavity wounds, it has become common practice to start IV antibiotics during the resuscitation phase of care. The CCTP will often transport patients from one facility to another with an antibiotic hanging. In most cases, the CCTP will simply need to be aware of any drug interactions and be alert for signs of an allergic reaction. If the patient is intubated, watch the capnography wave form for signs of bronchospasm.

Muscle Relaxants

The muscles around a fracture will undergo spasms to hold the bone ends in place. This self-splinting causes pain. Muscle relaxants can be used to control this pain in more stable patients. These medications are usually not used in the transport of the critical care patient. Occasionally, benzodiazepines are used both as anxiolytics and as muscle relaxants for critical care transport. Other muscle relaxants include cyclobenzaprine (Flexeril) and carisoprodol (Soma). These drugs potentiate opiates.

Anticoagulants

Anticoagulants represent another group of medications that the CCTP must be very comfortable with. The CCTP will transport many patients who will be on anticoagulants; most will be cardiac patients. During the critical care phase of the trauma, use of anticoagulants must be very carefully balanced against the risk of further bleeding. Anticoagulants are used in trauma to prevent deep venous thrombosis (DVT) and disseminated intravascular coagulation (DIC). DVT can result from damage to the blood vessel walls due to tissue debris or stasis of blood during low-pressure states and patient immobilization. Heparin has been the main preventive measure against DVT but is being replaced by low-molecular-weight heparin as a result of lower risk and less need to follow coagulation studies as closely. DIC can result from multiple blood transfusions, hypothermia, and damage to various organs, including the liver. There is a high risk in pregnant patients in particular. Treatment is complex and varied, but may include use of anticoagulants along with transfusion of various blood products.

Sedatives/Hypnotics

Anxiety and agitation impact negatively on patient care. In addition to the psychologic and physiologic impact, the agitated patient can endanger himself or herself as well as the transport crew. Adequate control of anxiety and agitation is an important part of safe and humane critical care transport, second only to cardiorespiratory stability and pain control. Benzodiazepines such as lorazepam and sedatives such as propofol are examples of medications used by many critical care transport units. It may be necessary to use etomidate and a long-acting paralytic such as vecuronium to provide for safe transport of the patient and to minimize his or her pain.

Trauma to Special Populations

Geriatric trauma and trauma to pregnant patients are covered here. Pediatric trauma is covered in Chapter 23.

Geriatric Trauma

Some 34 million Americans (12%) are older than 65 years, the age we call geriatric; but when it comes to trauma, mortality rates start to climb after the age of 30 years. The incidence of complicating comorbid conditions also increases after 30 years of age. These coexisting medical conditions cause lesser traumas, which then have a higher mortality. By the year 2030, 20% of the US population will be older than 65 years. Although patients older than 65 years account for only 11% of pedestrian accidents, they account for 25% of the fatalities. A driver older than 65 years is five times more likely to be fatally injured in an MVC than a younger driver. The 1-year mortality rate for hip fractures in the over-65 population is 20% and the 2-year rate is 33%. One in five burn center admissions is a patient older than 65 years. This is about 1,500 people a year, with a fatality rate seven times that of younger patients.

Assessment of the airway needs to include the presence of dentures or other dental devices that can become dislodged. Removing dentures may make intubation easier, but without having them in place, getting a mask seal is more difficult. Broken teeth are at an increased risk of becoming a foreign body in older patients. Kyphosis can cause problems for both spinal immobilization and airway management. Extra padding to fill the void under the head and additional personnel to assist with immobilization may be necessary. Because of a reduced cough reflex and an increased risk of aspiration, additional attention to suctioning may be necessary. The nasal tissues are more fragile and, if the patient is on anticoagulants, the risk of bleeding may render the use of both basic and advanced nasal airways problematic. Respiratory assessment must take into account reduced vital capacity and decreased ability for chest excursion, even prior to injury. Reduced tidal volume and lower minute volume can lead to significant reduction in oxygen and carbon dioxide exchange as a baseline. Progressive hypoxia can result from even minor chest injury. Increased PIP may be necessary as a result of chest wall stiffness. Older patients are often more dependent on diaphragmatic excursion, and any restriction of the abdomen may interfere with this respiratory movement. Assessment for shock and perfusion may be difficult in the elderly. Capillary refill may be normally delayed. Furthermore, a cardiovascular assessment of the geriatric patient may be complicated by medications such as beta-blockers or other antihypertensives. These medications may interfere with compensatory mechanisms of shock, holding the heart rate artificially low in the face of hypovolemia. Patients with pacemakers also will be unable to compensate by raising their heart rate. Preexisting hypertension means that decompensated shock may present as what appears to be a normal blood pressure to the observer. A slow or absent vasoactive response means that the patient is more dependent on preload. Although fluid resuscitation is still the mainstay of traumatic shock resuscitation for the elderly, caution must be exercised to prevent fluid overload from the inability of the

cardiovascular system to adjust to rapid changes in fluid volumes. Slow responses to questions or a lack of orientation to a location or date may not be a sign of head trauma but part of a retired and elderly person's lack of concern with dates and the slower processing of a normal older patient. Therefore, if the patient is conscious, history taking may require more patience when waiting for answers. The use of open-ended questions is recommended. Management considerations, in addition to those previously listed, include extra care to maintain temperature control and extra padding between splints or backboards as a result of the reduced subcutaneous fat.

Trauma During Pregnancy

Abdominal Injuries to Pregnant Patients

Injuries to pregnant patients are complicated by the multiple physiologic changes to the patient and the fact that the CCTP is now responsible for two lives. The primary cause of fetal demise in the traumatized mother is maternal shock and death. Therefore, the best treatment for the fetus is to resuscitate the mother. Trauma occurs in 8% of pregnancies but is the leading cause of death in pregnancy, accounting for 45% of maternal deaths. The most common cause of trauma during pregnancy is MVCs, followed by assault. About 8% of women between 15 and 40 years of age presenting to trauma centers do not even know they are pregnant. For the physiologic changes in pregnancy, see Chapter 21.

Some of the changes in pregnancy can mimic shock. In the first trimester, blood pressure drops about 5 mm Hg systolic and 15 mm Hg diastolic. The pregnant patient's heart rate increases as much as 20 beats/min. Other changes may hide shock as well. Blood volume increases by about 50% in mid-pregnancy, and a relative anemia results from hemodilution. The traumatized patient may lose as much as 40% of blood volume before the signs of shock are obvious. Because of the increase of coagulation factors and fibrinogen, the risk of DVT, pulmonary embolus, and DIC increases. This is especially true in patients with an ISS greater than 8.

Because of the increase in blood flow to the uterus to over 0.5 L/min with venous congestion of the pelvic vessels, the risk of massive blood loss is considerably increased with trauma to the bony pelvis.

Any pregnant patient who suffers from a pelvic injury most certainly has a bladder and/or uterine injury. At term, the uterus/placenta is perfused with approximately 600 to 800 mL of blood *per minute*! There should be a high rate of suspicion for these injuries, because these patients may exsanguinate rapidly.

Abruptio Placenta

In blunt trauma, as much as 70% of fetal demise is the result of abruptio placenta. Sudden deformation of the uterine wall can shear the placenta from the site of implantation. There may be no external signs of this occurrence. Signs of abruption include vaginal bleeding (only about 37% of the time), abdominal cramping, and symptoms of maternal hypovolemia. The most important sign is fetal distress. Ultrasounds are less than 50% accurate in finding an abruption. Women with an abruption are at an extremely high (54 times) risk for DIC as a result of release of thromboplastin from the placenta.

Signs and Symptoms

Abruptio Placenta

- Vaginal bleeding
- Abdominal pain
- Back pain

- Uterine tenderness
- Signs of shock
- Lack of fetal heart sounds

Differential Diagnosis

Abruptio Placenta

- Ectopic pregnancy
- Placenta previa
- Preterm labor
- Spontaneous abortion

Transport Management

Abruptio Placenta

- If the patient is beyond 20 weeks' gestation, tilt her left laterally at least 15° to prevent vena cava syndrome.
- Provide bleeding control.
- Maintain the patient's airway; provide maximum oxygenation.
- Initiate early IV access and implement aggressive fluid resuscitation (eg, with warmed lactated Ringer's solution).
- Provide rapid transport.

Management

Fetal resuscitation focuses on maternal resuscitation, and maternal resuscitation starts with the ABCs. With a higher risk of aspiration and the increase in gastric acidity, isolating the patient's airway is of vital importance. Because of increased oxygen consumption and reduced reserve, all mothers should receive maximum oxygenation. Hypoxia alone can cause a 30% reduction in uterine blood flow. Increased tidal volume and respiratory alkalosis as a baseline must be taken into account with ventilators and end-tidal carbon dioxide ($ETCO_2$) monitoring. The target for $ETCO_2$ should be 30, not 40 mm Hg. Early IV access with aggressive fluid resuscitation should be initiated before signs of shock. Warmed lactated Ringer's solution has been shown to restore fetal oxygenation better than other crystalloids. Beyond 20 weeks of gestation, the patient should be tilted left laterally at least 15° to prevent vena cava syndrome. CVP is helpful in guiding fluids, but remember that CVP normally trends down to as low as 3 mm Hg as pregnancy progresses.

Flight Considerations

The principles presented in this chapter apply to both ground and air critical care transport; however, as a result of the critical nature of their injuries, patients with severe trauma are more likely to be transported by air, especially in situations in which ground transport time could take considerably longer than air.

Summary

Caring for patients with traumatic injuries requires the CCTP to have a solid understanding of the trauma system in the United States. Knowledge and application of commonly used trauma scoring systems and triage are essential parts of daily work as a CCTP. The CCTP must apply this knowledge and the appropriate assessment skills to assess, triage, treat, and transport patients with traumatic injuries to the most appropriate facility. It is imperative that the CCTP is competent in clinical skills, understands why certain injuries occur, and has knowledge of which emergency procedures need to be performed to decrease morbidity and mortality. In mastering these skills, the CCTP becomes an integral part of the overall trauma care and management process, which in turn maximizes the patient's acute care, rehabilitation, and recovery.

Case Study

YOU ARE WORKING THE DAY SHIFT with a nurse-medic–staffed helicopter. You have been dispatched to a scene. You receive a report from your communication center that you are going to a farm site where an older man has been kicked by a bull. The patient is currently alert and his vital signs are stable, as reported by the paramedics on the scene. The closest Level 1 trauma center from the scene is a 30-minute flight.

Upon landing, you and your partner approach the scene, which is in the pasture of a beef cattle farm. You survey the scene to ensure your safety. You receive a report from the local paramedics. The patient, a 68-year-old man, was assisting in feeding cattle when one of the bulls attacked him, first striking him in the chest with his head (horns removed) and then kicking him in the chest. The medic states that the patient is complaining of shortness of breath and severe chest pain, but otherwise he is alert and oriented and his vital signs remain stable.

Your assessment reveals a well-developed man who appears to be extremely short of breath. He is wearing a nonrebreathing mask and is receiving oxygen at 15 L/min; his oxygen saturation is 90%. In looking at his chest wall, you see a significant defect on the left side of the chest, with paradoxical chest wall motion. The patient is alert and oriented, with a GCS score of 15. He states his breaths are becoming progressively shorter. The patient has two large-bore IVs in place and has already received 1,000 mL of normal saline. His vital signs include a blood pressure of 110/70 mm Hg, a pulse rate of 118 beats/min, a respiratory rate of 32 breaths/min, and an oxygen saturation of 90%.

You and your partner discuss the patient's need for pain medication. You administer 1 μg/kg IV of fentanyl. His heart rate slows down to approximately 100 beats/min, but his respiratory status does not improve. You and your partner discuss managing this patient's airway/ventilator status and elect to intubate the patient orally before leaving the scene. Using your local protocol, you administer etomidate, 0.3 mg/kg IV, and succinylcholine, 2.0 mg/kg IV. Cricoid pressure is applied and c-spine stabilization is maintained. The intubation is performed without difficulty. After intubation, you hear bilateral breath sounds but crackles in the left base. You apply capnography and monitor his $ETCO_2$. His initial reading after intubation was 60 mm Hg. You finish packaging the patient and begin transport.

In flight, you administer long-term paralytics and, additional sedation agents, and continue to treat the patient's pain. His vital signs remain stable, with a blood pressure of 98/70 mm Hg, a pulse rate of 90 beats/min, and ventilations at 16 breaths/min with 97% saturation. His $ETCO_2$ reading is 40 mm Hg, which shows that he is adequately ventilated. You and your partner discuss IV flow rates and elect to keep open both his IV lines. The patient is admitted to the Level 1 trauma center and a report is given to the attending staff.

1. Why did you elect to intubate this person when the standard of care for flail chest is to keep the

patient off mechanical ventilation if it all possible?

2. Why did you decide to continue the patient's fluids in-flight?

Analysis

For cases of flail chest the patient's pain is treated aggressively. With the pain under control, many times the patient is then able to ventilate adequately on his or her own. In this patient there were two concerns that needed to be considered. First, the patient was dyspneic and was not breathing adequately, as shown by his poor SaO_2, despite high-flow oxygen. Even after his pain was treated and his heart rate slowed down, his oxygen saturations were borderline at best. Second, the patient has a 30-minute flight, and the chance that his condition would continue to deteriorate seems high. In the treatment of flail chest, mechanical ventilation should be reserved for patients who have persistent respiratory insufficiency or failure, after adequate pain control. This patient met that standard; therefore, intubation is appropriate, and RSI should be performed. Follow local protocol for appropriate medication use. In this patient, it is imperative that you continue to treat his pain after administration of long-term paralytics and sedation. C-spine stabilization also needs to be maintained during intubation and the flight. Patient monitoring after intubation should include continuous capnography. A capnography reading is an excellent indicator of adequate ventilation in flight.

Many services are carrying continuous positive airway pressure devices on the helicopter. This patient would have been a candidate for continuous positive airway pressure in flight, thus avoiding mechanical ventilation. The CCTP would have to be astute in ongoing pulmonary assessment to realize when the patient's condition is failing and would need to provide mechanical ventilation.

With chest trauma that is significant enough to break multiple ribs, there is a higher associated risk of underlying parenchymal injury to the lung. Aggressive fluid resuscitation only worsens the interstitial fluid leak, which leads to ventilation-perfusion mismatch and, thus, worsening pulmonary contusion. This patient had already received 1,000 mL of fluid, which probably worsened his lung condition.

The patient's mean arterial blood pressure should be maintained at or above 65 mm Hg. His current reading is approximately 79 mm Hg, which is adequate for end-organ perfusion. This patient's IV fluids should be restricted. If his blood pressure dictates, titrate the patient's fluids according to his MAP.

Prep Kit

Ready for Review

- A paradigm shift in the care of critically injured trauma patients has resulted in an emphasis on rapidly identifying and treating those patients who need immediate transport to an appropriate trauma center.
- The ability of the CCTP to rapidly identify life-threatening injuries while recognizing the need for transport to a trauma center will help to decrease the incidence of morbidity and mortality related to these traumatic injuries.
- CCTPs may encounter trauma patients during a scene call, when they are called to back up field BLS or ALS providers. In these cases, the CCTP will be working in more austere and less controlled conditions with less sophisticated assessment tools than in an interfacility transport.
- CCTPs may also aid in transferring critical patients with trauma, usually from smaller facilities to trauma centers. In this situation, the CCTP may have access to the results of any assessment findings identified prior to transport and may be able to enlist the aid of the sending physician or other house staff to help with stabilization efforts prior to transport.

- The balance of treatment before transport vs what can be done en route should always be a top priority in the CCTP's decision-making process.
- Trauma is a major cause of death in persons younger than 40 years, and is the leading cause of death in children.
- Many people survive a motor vehicle crash, only to suffer a debilitating injury that leaves them incapacitated. Providers must appreciate the importance of avoiding secondary injury caused by improper handling of these patients.
- Trauma scoring systems are used in the critical care transport environment to provide assessment information and indicators of patient survivability.
- The Glasgow Coma Scale (GCS) is a neurologic assessment tool that assigns a numerical value to the patient's condition based on three body functions: best eye-opening response, best verbal response, and best motor response.
- The GCS should be used regardless of the cause of central nervous system alteration. It is predominantly used and reported during the care of critically ill and injured patients when their current level of consciousness is pertinent to their acuity level.
- Patients who present with GCS scores of less than 8 require intubation or another form of advanced airway intervention.
- The trauma score takes into account the GCS score, respiratory rate, respiratory expansion, systolic blood pressure, and capillary refill rate. The score, which ranges from 1 to 16 (the best possible score), reflects the likelihood of patient survival.
- The revised trauma score measures three physiologic parameters: respiratory rate, systolic blood pressure, and GCS score. The total score ranges from 0 (the most severely debilitated patient) to 13 or, when a weighted system is used, from 1.0 to 7.8408.
- The revised trauma score is considered flawed because it measures the body's physiologic response to the injury and will not identify a small percentage of severely injured trauma patients.
- The AIS is an anatomic scoring system that provides a reasonably accurate means of ranking the severity of the injury by categorizing injuries into six body regions. Scores for each region range from 1 (minor injury) to 6 (unsurvivable), but do not take into account multisystem injuries.
- The ISS is an anatomic scoring system that provides an overall score for patients with multiple injuries. Scores range from 1 (minor injury) to 75 (high mortality).
- A patient with an ISS of greater than 15 is frequently considered a major trauma patient who requires immediate attention and, in most cases, transfer to a Level I facility.
- The trauma injury severity score is used to determine the survival probability of the critically ill and/or injured patient. It takes into account the ISS, the revised trauma score, and the patient's age, which collectively summarize the patient's physiologic and anatomic indicators.
- Understanding Newton's first law of motion—"A body in motion remains in motion in a straight line unless acted upon by an outside force"—enables the CCTP to predict patterns of injuries in trauma incidents and deliver the appropriate standard of care.
- Traumatic injuries are generally classified into one of two categories: blunt or penetrating.
- Blunt injuries result from energy exchange between an object and the body, without intrusion through the skin—for example, rapid forward decelerations (collisions), rapid vertical decelerations (falls), and energy transferred from blunt instruments.

- Penetrating injuries refer to injuries caused by external forces in which the tissue is penetrated by an object—for example, injuries caused by projectiles such as bullets, knives, fragments from explosions, and falls upon fixed objects.
- Deceleration injuries are caused by a sudden stop of the body's forward motion. Because they may not be visible to the CCTP, an excellent understanding of the types of mechanisms of injury and effective assessment skills are imperative for detecting them.
- External force injuries are caused by forces that violate the tissues of the body. Their severity depends on the anatomic area involved, the mass, and the velocity of the foreign object that enters the body.
- The CCTP must not develop tunnel vision; that is, he or she must not focus on a visible injury, which may or may not be life threatening, while paying little or no attention to other, life-threatening injuries.
- Maintaining a calm, professional demeanor allows the CCTP to maintain a high index of suspicion and attention to detail.
- Trauma assessment begins with scene size-up, which encompasses scene safety, body substance isolation, number of patients, determination of whether additional resources are needed, and mechanism of injury (ie, what happened or how the patient was injured).
- Trauma assessment encompasses both a physical examination and a subjective interview (when possible); the latter information often has to come from someone other than the patient.
- The primary assessment should take no longer than 1 minute and includes the general impression of the patient, level of consciousness, and assessment of the ABCs.
- If the initial trauma primary assessment reveals no major injury, the CCTP should move on to either a rapid trauma exam or a focused exam. Trauma patients who have an isolated injury require a focused examination; a rapid trauma assessment is used for patients with multisystem injuries.
- A rapid trauma assessment allows the CCTP to rapidly assess the head, neck, chest, abdomen, pelvis, and extremities to find and correct any life threats and should be completed in less than 1 minute.
- When conducting a focused exam, the questions to ask will vary depending on the patient's specific injury.
- A more comprehensive secondary assessment is generally done during transport to a trauma center. The secondary assessment is a more comprehensive examination to determine additional injuries that may have been overlooked in the primary assessment; its findings serve as a baseline for future treatment decisions in the continuity of care.
- Critical patients who fall into the "load-and-go" category should have the secondary assessment performed during transport; noncritical patients may be assessed on the scene.
- Once the primary and secondary assessments are completed, the CCTP must determine the criticality of the patient and make a transport decision. Patients who meet critical care criteria should immediately be loaded into the critical care transport unit and taken to the appropriate trauma center.
- Issues related to airway, breathing, and external bleeding are the only reasons to delay transport, and it should only be delayed long enough to intervene with these problems.
- Continual reassessment of the patient and interventions performed should occur during the transport. If the patient's condition deteriorates, the CCTP should immediately reassess the primary assessment and address any negative findings, if possible.
- Triage is a system of sorting patients based on injury severity, with the goal being to use the limited resources for those patients who need them the most ("doing the greatest good for the greatest number").

- Immediate (red) triage tags denote the highest-priority cases. Patients in this category need immediate care and transport to definitive treatment centers.
- Delayed (yellow) triage tags are reserved for second- or delayed-priority patients. Patients in this category need treatment and transport, but those measures can be delayed.
- Minimal (green) triage tags are given to third-priority patients—the "walking wounded," who have minor soft-tissue injuries.
- Expectant (black) triage tags are used for patients for whom no treatment is indicated—those who are dead, those who are alive but nonsalvageable, and those who are not injured.
- The triage officer has ultimate control of the counting and prioritizing of patients; the treatment officer oversees the treatment area; the transportation officer oversees transport of patients; and the staging officer oversees and directs the staging area to deploy needed resources to the appropriate treatment sectors.
- START (simple triage and rapid treatment) triage is one of the easiest methods of triage and entails a limited assessment of the patient's ability to walk, respiratory status, hemodynamic status, and neurologic status.
- The JumpSTART triage system is used for prioritizing care of children younger than 8 years or who appear to weigh less than 100 lb. It includes the same parameters as the START system, albeit tailored to pediatric patients.
- Trauma centers are classified into Levels I through IV, with Level I (regional centers that provide every aspect of trauma care) having the most resources, followed by Level II (facilities that provide initial definitive care), Level III (facilities that provide assessment, resuscitation, emergency care, and stabilization, and then transfer patients to higher-level centers), and Level IV (clinics that provided advanced trauma life support prior to patient transfer).
- Patients with severe trauma (Level I patients) are more likely to be transported by air, especially in situations in which ground transport time could take longer than air transport.
- Of all trauma deaths, 25% are directly associated with thoracic injuries.
- For trauma patients, computed tomography scanning is typically used for head injury identification, identification of bleeding, and complex fractures. Typical studies will scan the head, chest, and abdominal-pelvic regions.
- Magnetic resonance imaging provides much greater contrast that is useful for identification of abnormalities in trauma.
- Focused Assessment with Sonography for Trauma (FAST) ultrasound is used to identify the presence of free intraperitoneal or pericardial fluids, and has replaced diagnostic peritoneal lavage and laparotomy for this indication.
- Intra-abdominal pressure (IAP) is the static pressure inside the abdominal compartment. Deleterious effects of intra-abdominal hypertension (IAH) can lead to abdominal compartment syndrome (ACS) and death if untreated. Definitive treatment is surgical decompression, but there are many interventions that can help prevent IAH and ACS. Also, the CCTP may be asked to obtain IAP measurements during transport.
- When transporting trauma patients, CCTPs should always ask for and ensure that all copies of any imaging studies are included as part of the patient record.
- The primary function of the thorax is to allow for adequate oxygenation and circulation. Therefore, injuries to the chest must be promptly found and managed.

- Care must be taken in traumatized patients to ensure adequate positive-pressure ventilations without allowing interthoracic pressure to rise too high; this may become an issue during flight.
- Pneumothorax injuries cause air to be drawn into the thoracic cavity with each breath. A collapse as small as 10% can be life threatening if there are other comorbid issues.
- Complications of sealing a sucking chest wound can cause a tension pneumothorax if air is still able to enter the pleural cavity from the lung side of the wound and builds up or if the seal is ineffective.
- A simple pneumothorax is most often associated with a closed chest injury, such as from a fractured rib being driven inward into the lung or from a medical cause of a collapse of the lung.
- A tension pneumothorax is a life-threatening injury that results from a continual influx of air into the pleural space. It may occur secondary to a closed simple pneumothorax or after sealing an open pneumothorax.
- Management of a tension pneumothorax is performed by immediate needle decompression.
- Hemothorax can be caused by blunt or penetrating trauma, as well as a tumor eroding through the great vessels. It may potentially lead to hypovolemic shock or, if a large amount of blood accumulates in the pleural cavity, it can interfere with lung function.
- Patients with a pneumothorax, hemothorax, hemopneumothorax, or empyema ultimately may need a chest tube insertion. In addition, CCTPs routinely care for and transport patients with chest tubes in place.
- Because of the potential for massive hemorrhage from thoracic trauma, the CCTP must be familiar with the administration of blood.
- A flail chest (flail segment) consists of a fracture in two or more places to two or more adjacent ribs. Bruises to the underlying tissue causing a pulmonary or myocardial contusion are the most dangerous sequelae of a flail chest.
- Flail segments pose a threat to the patient's ability to breathe, increase the risk for developing a hemothorax or pneumothorax, and require immediate treatment.
- The hallmark treatment for flail chest is mechanical ventilation with a sedate, paralyzed patient who has been adequately treated for pain. Some patients may be managed conservatively with pain medications and oxygen supplementation alone.
- Pericardial tamponade is a life-threatening condition that requires immediate treatment. It occurs in 2% of penetrating injuries to the chest and upper abdomen, and is more common in stab wounds to the heart than in gunshot wounds.
- Most often, prehospital management of pericardial tamponade is only supportive; sometimes, however, the CCTP may need to perform or assist in an emergency pericardiocentesis.
- Traumatic aortic rupture is the most common cause of immediate death in motor vehicle collisions.
- Tears in the ascending aorta are almost uniformly immediately fatal, but patients with tears on the descending side have a 15% chance of making it to the operating room. However, there is a 30% mortality rate among those patients who make it to the hospital.
- Myocardial contusion (bruise to the myocardium) is primarily the result of blunt trauma to the anterior chest wall, such as trauma that occurs during motor vehicle collisions or with other blows to the front of the chest (eg, gunshot to a bulletproof vest).
- With myocardial contusion, damage to small blood vessels may cause local bleeding and direct trauma to cells. Because the areas downstream from the damaged vessels are no longer perfused, the damage

may resemble that associated with myocardial infarction.

- If blunt compression of the abdomen occurs (eg, from blunt or penetrating trauma), it may cause diaphragmatic rupture and herniation of abdominal organs into the thoracic cavity. This impinges on lung function and decreases venous return, resulting in decreased ventilatory and cardiac output.
- Tracheobronchial injuries are rare, but often life threatening. The leakage of air with such damage may cause tension pneumothorax or tension pneumomediastinum, which will act like a cardiac tamponade.
- Management of tracheobronchial disruption includes judicious use of ventilatory support. CCTPs must be aware that paralysis of the muscles may allow retraction of the trachea into the chest.
- Tearing and lacerations to the lung tissue (pulmonary contusion) can cause bleeding and leakage of plasma into alveoli and the interstitial spaces around them. This damage interferes with gas exchange and leads to severe hypoxemia.
- Patients with pulmonary contusion may be managed with noninvasive, continuous positive airway pressure and resuscitation fluids.
- Esophageal perforations are most often caused by penetrating injuries, such as from projectiles or from caustic ingestion, but can also be caused by a medical issue, such as cancer, gastroesophageal reflux disease erosions, or excessive vomiting.
- Traumatic asphyxia (a severe sudden crushing injury to the chest and abdomen) is not a form of asphyxia nor is it by itself fatal. Associated injuries, if present, tend to be far more serious.
- Facial trauma can be life threatening as a result of airway compromise and the threat to the patient's ability to get sensory information through sight and hearing; it also has many social and self-image implications for the patient and can be distracting for caregivers.
- A punctured eardrum may cause vertigo and nausea, which the CCTP may need to control with medication. The patient should be protected from aircraft noise.
- Blood or fluid coming from the auditory canal indicates a more serious injury and should be assessed with the halo test.
- A ruptured tympanic membrane is not life threatening, but should raise the suspicion of other, much more serious overpressure injuries.
- Eye injuries, although often dramatic, are not life threatening. Care typically consists of protecting the eye from further injury and transporting the patient to the ophthalmology service.
- In chemical burns to the eye, adequate washing is essential and should be performed for at least 10 minutes prior to transport for acid burns and at least twice as long for alkali burns.
- If an object is impaled in the eye, both the eye and the object should be prevented from movement, if possible, for transport.
- Hyphema (a collection of blood in the anterior chamber of the eye) may be a marker of damage to other structures of the eye and requires a full ophthalmologic examination.
- Rupture of the ocular globe with leak of vitreous humor may be the result of penetration by a foreign body or blunt trauma.
- Despite the dramatic presentation of enucleation of the eyeball from the eye socket, loss of vision is not inevitable. Proper care includes protecting the eye from further trauma in transit by using a protective cup or other rigid protective device with gauze padding.
- Retinal detachment (separation of the inner layers of the retina from the pigmented epithelium) may be caused by trauma, diabetic retinopathy, and sickle cell disease. With rapid diagnosis and surgery,

blindness may sometimes be avoided in such cases.

- Treatment is usually not necessary on a scene call for mandibular fracture or dislocation, but provision must be made for an emergency airway during the transfer of any patient whose jaw is wired shut.
- Avulsed teeth have a good chance of reimplantation if replaced within an hour.
- The critical airway and vascular structures running through the neck are relatively unprotected during trauma events. In addition, the possibility of unstable cervical spine and cord injury must always be considered and protected against.
- Securing the airway with endotracheal intubation should be considered prior to transporting any patient with a neck injury. Deterioration of the patient's airway in the back of a transport vehicle with limited space and personnel can be disastrous.
- Most laryngotracheal injuries occur in the area of the cervical trachea and are most frequently caused by direct blunt trauma. These injuries can appear stable for a time, but then be followed by rapid and catastrophic deterioration.
- In case of laryngotracheal disruptions, paralytics and muscle relaxants are absolutely contraindicated.
- Direct trauma to the front of the neck (including the thyroid gland) can cause hematomas of sufficient size to impinge on the airway. Thyrotoxicosis (thyroid storm) has a 20% to 30% mortality rate.
- Injury to the carotid, subclavian, and vertebral arteries, and to the external and internal jugular veins, can produce rapid exsanguination, hematoma formation, or embolization of air, with lethal outcomes.
- If the patient has experienced blunt or penetrating trauma to the abdominal cavity, signs or symptoms of injury may be very difficult to identify until shock becomes obvious. Because of the size of the abdominal cavity, a significant amount of blood can collect within the cavity and go unnoticed.
- With abdominal injuries, it is important for the CCTP to focus attention on the mechanism of injury, instead of the outward signs of trauma found on assessment.
- If a significant abdominal injury is suspected or confirmed, the most definitive treatment that the CCTP can provide is rapid transport to the trauma center, while stabilizing and maintaining the patient's hemodynamic status as effectively as possible, so that surgical intervention can be performed if needed.
- The seatbelt has significantly decreased the incidence of morbidity and mortality related to automobile crashes, but continues to cause less noticeable abdominal injuries.
- Penetrating injuries may be very difficult for the CCTP to assess because the necessary information about the penetrating object is often not available. Even when this information is available, it is difficult to rapidly identify which structures inside the abdominal cavity were injured without the assistance of imaging studies.
- In case of abdominal injury, the physical exam performed by the CCTP to rapidly identify the presence of blood loss is the most important task.
- The primary assessment of a patient with abdominal injury should be completed before beginning transport, and repeated frequently during transport, watching for changes that require immediate intervention.
- All patients suspected of having abdominal injuries should be transported on high-flow oxygen with fluid administration. The more severe cases may need aggressive airway management and blood administration.
- The primary concern with hollow-organ injury is the potential for peritonitis to develop.

- The spleen is the most commonly injured organ in the abdominal cavity. Young adults and patients with sickle cell disease are at particular risk for spleen injury.
- Failure to recognize, understand, and treat the signs and symptoms of hypovolemic shock related to internal hemorrhage will rapidly lead to the patient's death.
- The ligament in front of the liver can slice the liver in sudden deceleration. The liver's close proximity to the lower rib cage also makes it susceptible to injury after a rib fracture occurs.
- Although penetrating trauma is the most common source of injury to the intestines, blunt trauma to the abdominal wall—often caused by a seatbelt—can also damage the small bowel.
- Approximately 80% of gunshot wounds to the abdomen and 30% of stab wounds lacerate the jejunum and ileum.
- Injuries that occur to the vessels of the abdomen are usually life threatening. Severe blunt trauma may cause disruption of these vessels, but the most common cause of vascular injury is penetrating trauma.
- Rapid transport of patients with an injury to any of the major abdominal vessels to a hospital capable of immediate surgery is the best possible treatment.
- The presence of a pelvic fracture should alert the CCTP to the possible existence of other injuries, given the force that must be exerted to cause pelvic fracture; the potential for (possibly fatal) internal hemorrhage is high.
- Subjective assessment for pelvic fracture includes identification of the mechanism of injury, location and radiation of pain, presence of hematuria, and possibility of pregnancy. Objective examination should include observing for the rotation or uneven height of the iliac crests and uneven length of the legs, palpating the posterior aspect of the pelvic ring, and exerting gentle lateral compression and inward compression.
- Musculoskeletal injuries, although often dramatic, may distract the CCTP from more serious injuries. Nevertheless, their presence may be an indicator of the amount of energy transferred to the body and may suggest the likelihood of additional injury.
- All musculoskeletal injuries should be assessed for the six Ps (pain, pallor, pulselessness, paresthesia, paralysis, and pressure) before and after any manipulation.
- Regardless of whether a fracture is open or closed, the CCTP must make prevention of further injury as a result of movement, with pain assessment and management, a priority.
- Closed fractures can be as dangerous as open fractures because of the internal bleeding into the tissues involved.
- In an open fracture, the skin over the bone is broken, adding the potential danger of infection, which could lead to sepsis and death.
- Femur fractures should be considered to account for the loss of at least 1 L of blood from circulation.
- Any movement during transport of a patient with a vertebral fracture can transform an unstable fracture without neurologic deficit into a life-long disability. Accordingly, the current practice is to fully immobilize the spine for transport.
- The most devastating vertebral fractures can occur in the seven cervical vertebrae. Planning for airway management and prevention of aspiration (especially in small aircraft) must be made prior to transport of any patient with such an injury.
- Thoracic fractures can lead to a loss of innervation of the intercostal muscles, leading to respiratory insufficiency. Spinal shock, in which hypotension is accompanied by bradycardia instead of

tachycardia, can affect the thoracic area or higher.

- Humerus fractures may result in significant blood loss. Absence of distal pulses or nerve function should receive immediate attention, usually in the form of traction in anatomic alignment and manipulation.
- Until proven otherwise, all dislocations should be assumed to be coupled with a fracture. Joint dislocations are true emergencies as a result of the potential for neurovascular compromise that could lead to amputation if the injury is not treated appropriately.
- Amputations can be life threatening and are often disabling. There is potential for hemorrhage, although bleeding can usually be controlled with direct pressure to the stump.
- General management of fractures and trauma depends on the overall state of the patient. If the patient needs immediate resuscitation, then airway, ventilation, stopping blood loss, and fluid resuscitation must take priority.
- For fracture and trauma scene calls that do not require critical resuscitation, the priority for care is pain control, assurance of distal neurovascular function, and careful splinting of each fracture.
- No matter which type of fixation splint is used, the entire bone with the adjacent joints must be held immobile for the splint to be effective.
- Swelling under a cast can cause compartment syndrome. The most common anatomic locations for compartment syndrome are the forearms and legs.
- If diastolic blood pressure drops below 30 mm Hg over compartment pressure, it is considered an emergency. When compartment pressure exceeds diastolic pressure, the blood flow in the extremity is stopped.
- Rhabdomyolysis (damage to the sarcolemma) initiates a cascade of events that ultimately results in cardiac arrhythmias and acute renal failure. The leading causes of rhabdomyolysis are exercise, alcohol, drugs, infections, trauma, compression, and seizures.
- When a CCTP is called to a scene involving a patient with prolonged entrapment of a large muscle area, it is imperative that evaluation and treatment be started prior to the release of the entrapped patient to avoid the mortality associated with crush syndrome.
- Although a traumatized, pulseless extremity may be repositioned successfully to reestablish circulation, there are dangers in doing so. If attempted, repositioning should be done with manual traction in anatomic alignment with the long bone.
- A primary concern of the CCTP is to properly ameliorate pain by using nonsteroidal anti-inflammatory drugs in stable patients and opiates in acute-care and critical patients.
- The most serious side effect of opiates in the short term is respiratory depression. Hypotension and nausea or vomiting can also be side effects of these drugs.
- Especially in patients with open fractures and body cavity wounds, it has become common practice to start IV antibiotics during the resuscitation phase of care. Before doing so, be aware of any drug interactions and stay alert for signs of allergic reaction.
- Many patients the CCTP will transport will be on anticoagulants, and most will be cardiac patients. In trauma patients, use of anticoagulants in the critical care phase must be very carefully balanced against the risk of further bleeding.
- The agitated patient can endanger himself or herself as well as the transport crew. For this reason, adequate control of anxiety and agitation is an important part of safe and humane critical care transport.

- Considerations when treating geriatric patients with trauma include several aging-related issues: presence of dentures or other dental devices, kyphosis, reduced cough reflex and increased risk of aspiration, fragility of the nasal tissues, increased risk for bleeding, reduced vital capacity and decreased ability for chest excursion, chest wall stiffness, difficulty of assessment for shock and perfusion, preexisting cardiovascular abnormalities or use of cardiovascular medications, tendency toward fluid overload, slower mental processing, and difficulties in thermoregulation.
- The primary cause of fetal demise in the traumatized pregnant patient is maternal shock and death. Therefore, the best treatment for the fetus is to resuscitate the mother.
- Some of the changes in pregnancy can mimic shock, which makes it difficult to diagnose true shock in a pregnant trauma patient.
- The risk of massive blood loss is considerably increased with trauma to the bony pelvis of a pregnant patient. Any pregnant patient who suffers from a pelvic injury most certainly has a bladder and/or uterine injury.
- Fetal resuscitation centers on maternal resuscitation, and maternal resuscitation starts with the ABCs.

Vital Vocabulary

Abbreviated Injury Scale (AIS) A trauma scoring system that ranks injury severity by assigning an individual injury score of 1 to 6 to six body regions, with 1 being a minor injury and 6 being an injury with a high mortality rate; does not account for multisystem injuries.

abdominal compartment syndrome (ACS) A condition that can result from intra-abdominal hypertension, and that includes a range of deleterious effects, including decreased end-organ perfusion; if untreated, it can lead to death.

Beck's triad The combination of a narrowed pulse pressure, muffled heart tones, and jugular vein distention associated with cardiac tamponade; usually resulting from penetrating chest trauma.

FAST An ultrasound examination directed at identifying the presence of free intraperitoneal or pericardial fluids, performed by transducing four distinct areas of the abdomen; stands for Focused Assessment with Sonography for Trauma.

Glasgow Coma Scale (GCS) An evaluation tool used to determine level of consciousness, which evaluates and assigns point values (scores) for eye opening, verbal response, and motor response, which are then totaled; effective in helping predict patient outcomes.

Injury Severity Score (ISS) A trauma scoring system that adds the squares of the three highest abbreviated injury scale scores to create a score between 1 and 75 that accounts for multiple injuries, with 1 being a minor injury and 75 being an injury with a high mortality rate.

JumpSTART triage A sorting system for pediatric patients younger than 8 years or weighing less than 100 lb. There is a minor adaptation for infants because they cannot ambulate on their own.

Kehr's sign Left shoulder pain that may indicate a ruptured spleen. Right shoulder pain may indicate trauma to the liver.

morbidity The number of nonfatally injured or disabled people. Usually expressed as a rate, meaning the number of nonfatal injuries in a certain population in a given time period divided by the size of the population.

mortality Deaths caused by injury and disease. Usually expressed as a rate, meaning the number of deaths in a certain population in a given time period divided by the size of the population.

pericardial tamponade Impairment of diastolic filling of the right ventricle as a result of significant amounts of fluid in the pericardial sac surrounding the heart, leading to a decrease in the cardiac output.

pericardiocentesis A procedure in which a needle or angiocath is introduced into the pericardial sac to relieve cardiac tamponade.

revised trauma score A trauma scoring system that rates injury severity by comparing the Glasgow Coma Scale score, the systolic blood pressure, and the respiratory rate and assigning a score between 1 and 13 based on these three values; in some cases the parameters are weighted, resulting in a score of between 1.0 and 7.8408.

rhabdomyolysis Damage to the sarcolemma (muscle membrane) from any cause, leading to a release of potassium and myoglobin.

START triage A patient sorting process that stands for simple triage and rapid treatment and uses a limited assessment of the patient's ability to walk, respiratory status, hemodynamic status, and neurologic status.

thyrotoxicosis An excess of thyroid hormones resulting in a hypermetabolic crisis, including tachycardia over 140 beats/min; hyperthermia (sometimes greater than 103.9°F); coma with agitation, nausea, vomiting, diarrhea, and unexplained jaundice; and pulmonary edema; marked by an elevated thyroxine level; also called thyroid storm.

tracheal deviation The late sign of a tension pneumothorax in which the trachea is tugged to one side of the neck, usually opposite the side of the pneumothorax.

trauma injury severity score A calculated scoring system that uses the results of the injury severity score, the revised trauma score, and the patient's age to determine survivability rate; rarely used in the transport setting.

trauma score A score from 1 to 16 that takes into account the Glasgow Coma Scale score, respiratory rate, respiratory expansion, systolic blood pressure, and capillary refill, and relates to the likelihood of patient survival; not accurate for patients with severe head injuries.

traumatic asphyxia A condition resulting from severe, sudden crushing injury to the chest and abdomen, forcing blood backward out of the right side of the heart; engorging the veins of the chest, neck, and head; and giving the chest, neck, and head an extremely cyanotic appearance.

Bibliography

American Geriatrics Society, National Council of State Training Coordinators. *Geriatric Education for Emergency Medical Services*. Sudbury, MA: Jones and Bartlett Publishers; 2003.

Aspesi M, Gamberoni C, Severgnini P, et al. The abdominal compartment syndrome: clinical relevance. *Minerva Anesth*. 2002; 68(4):138–146.

Association of Air Medical Services (AAMS). *Appropriate Utilization of Critical Care Ground Transport Services: Position Statement of the Association of Air Medical Services (AAMS)*. Available at: http://www.aams.org/Content/NavigationMenu/PublicationsProducts/ResourceDocuments/PostCCGAppropriateUsePaper.doc. Accessed February 4, 2008.

Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. *Arch Surg*. 2003;138(6):637–643.

Brohi K. *Revised Trauma Score and Desktop Calculator*. Trauma. org: Care of the Injured. London,

United Kingdom. March 10, 2007. Available at: http://www.trauma.org/index.php/main/article/386/. Accessed January 21, 2009.

Cheatham ML, Malbrain ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome, II: recommendations. *Intensive Care Med*. 2007;33(6):951–962.

Gabbe BJ, Cameron PA, Finch CF. Is the revised trauma score still useful? PubMed: National Center for Biotechnology Information and US National Library of Medicine. Available at: http://www.ncbi.nlm.nih.gov/pubmed/14616576. Accessed May 5, 2009.

Gentilello LM, Jurkovich GJ, Stark MS, et al. Is hypothermia in the victim of major trauma protective or harmful? a randomized, prospective study. *Ann Surg*. 1997(October);226(4):439–449.

Helm M, Lampl L, Hauke J, Bock KH. Accidental hypothermia in trauma patients: is it relevant to preclinical emergency treatment? *Anaesthetist*. 1995;44:101–107.

Martin SR, Kilgo PD, Miller PR, et al. Injury-associated hypothermia: an analysis of the 2004 National Trauma Data Bank. *Shock*. 2005;24(2):114–118.

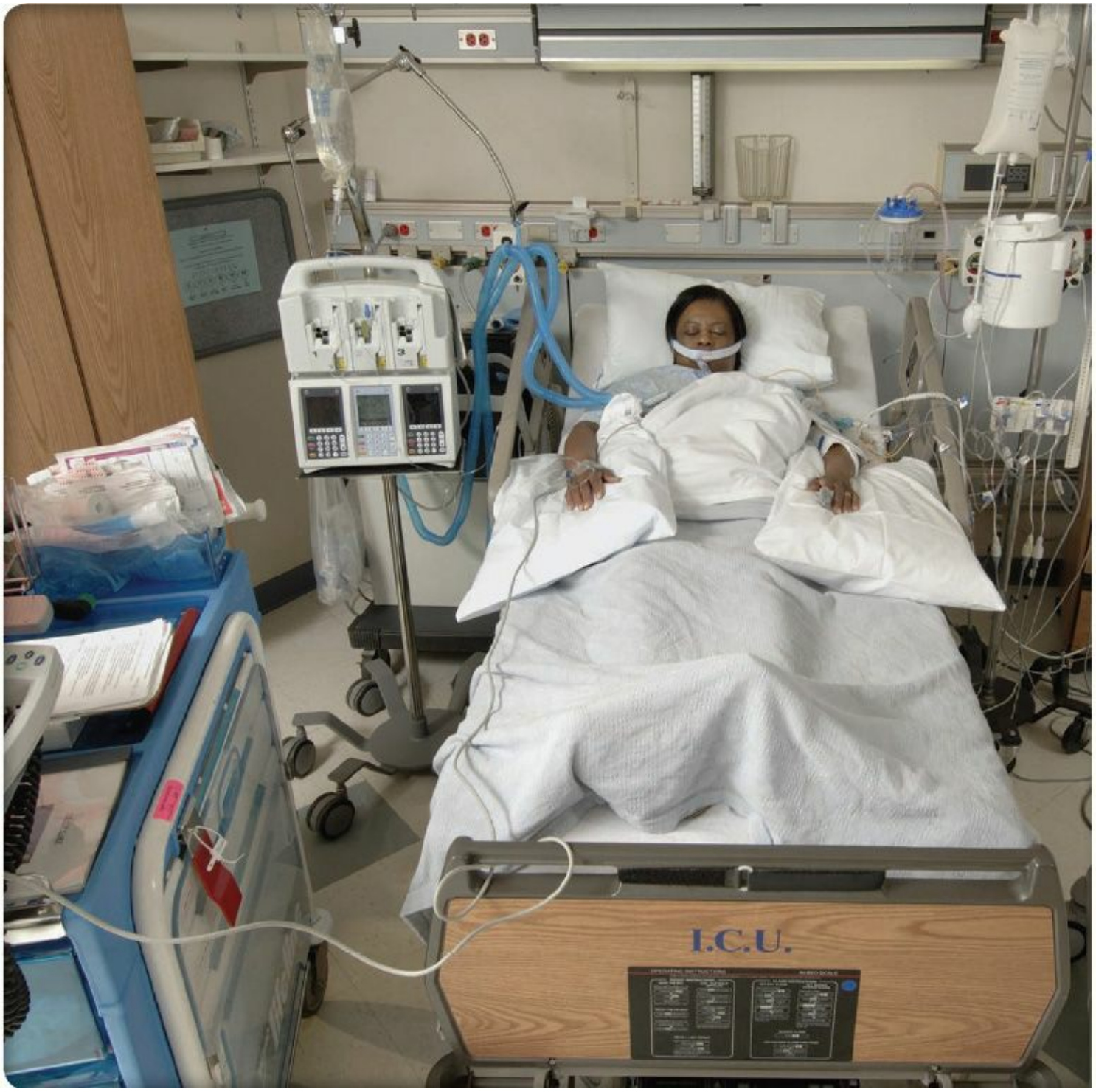
Marx JA, Hockberger RS, Walls RM. *Rosen's Emergency Medicine: Concepts and Clinical Practice* , 6th ed. St Louis, MO: Mosby; 2006.

McNelis J, Soffer S, Marini CP, et al. Abdominal compartment syndrome in the surgical intensive care unit. *Am Surg*. 2002;68(1):18–23.

PHTLS Committee, National Association of Emergency Medical Technicians. *Prehospital Trauma Life Support*, 6th ed. St Louis, MO: Mosby; 2007.

Smith CE. Prevention and treatment of hypothermia in trauma patients. TraumaCare International ITACCS. Available at: http://www.itaccs.com/traumacare/archive/04_01_Spring_2004/prevention.pdf. Accessed April 23, 2009.

Wang HE, Callaway CW, Peitzman AB, et al. Admission hypothermia and outcome after major trauma. *Crit Care Med*. 2005;33(6):1296–1301.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Neurologic Emergencies

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Objectives

1. Describe the major anatomic structures of the nervous system and their physiology as is pertinent to the CCTP (p 366–380).
2. Describe how to perform a neurologic assessment, including the following specific assessments: mini-mental exam; assessment of speech abnormalities, cranial nerves, eyes, motor function, and sensory function, reflex testing, and evaluation of meningeal irritation (p 381–395).
3. Discuss the pathophysiology of traumatic brain injury, including primary and secondary brain injury (p 395–396).
4. Explain the significance of cerebral perfusion pressure and mean arterial pressure (p 396).
5. Discuss the pathophysiology of specific neurologic injuries, including scalp injuries, skull fractures, facial fractures, epidural hematoma, subdural hematoma, and diffuse axonal injury (p 396–404).
6. Discuss lab values relevant to neurologic emergencies and their interpretation (p 424).
7. Define intracranial pressure (ICP) and explain its pathophysiology (p 405).
8. Discuss clinical manifestations of increased ICP, including brain herniation (p 407).
9. Discuss the concept of ICP monitoring, including indications, contraindications, methods, devices, waveforms, complications, procedures, adverse reactions and interventions, and post-procedure care (p 408).
10. Discuss management of ICP during transport (p 412).
11. Describe spinal injuries, including primary spinal cord injury and secondary spinal cord injury, and their assessment, management, and complications (p 416–421).
12. Discuss types of stroke, their assessment, transport management, and thrombolytic and fibrinolytic therapy (p 421).
13. Describe the pathophysiology of subarachnoid hemorrhage, its assessment, and its management, including neurologic diagnostics (p 423).
14. Discuss seizures and epilepsy, including transport management (p 424).
15. Discuss transport considerations for patients with neurologic injuries (p 425).
16. Discuss considerations for managing neurologic emergencies in flight (p 426).

Introduction

Critical care patients with neurologic complications present a variety of assessment and management challenges during transport for the critical care professional. The CCTP will find that an understanding of

the underlying principles of neurologic structure, function, and dysfunction will provide the basis for critical care management. Patients with neurologic illness and injury require skilled care that includes a thorough history and physical examination as well as a variety of specialized interventions.

A majority of neurologic patients who require transport will have traumatic brain injury (TBI). An in-depth understanding of the types of injuries, their pathophysiology, and current (evidenced-based) management trends is fundamental to managing these patients in a way that ensures the best possible outcome.

Anatomy and Physiology

The human nervous system is a truly remarkable arrangement of fibers running throughout the body that allows the body to interact with and adapt to differing environments by regulating the activities of virtually every other body system. An intimate understanding of the anatomy and physiology of the nervous system is essential to the delivery of competent medical care—and especially assessment—by the CCTP. This chapter reviews the structures and functions of certain components of the nervous system that are most pertinent.

Nervous System Organization

The nervous system is perhaps the most diverse and highly organized organ system in the human body. Its various components are classified in terms of location or function.

When classifying the parts in terms of their location, the nervous system is divided into the central nervous system (CNS), which consists of the brain and spinal cord, and the peripheral nervous system (PNS), which consists of the spinal and **cranial nerves**.

The parts of the PNS are also differentiated on the basis of the information that is transmitted on certain fibers. Most activities mediated by the nervous system occur as a result of sensory stimulation sensed by special receptors, such as visual, auditory, or tactile receptors. **Afferent pathways** (ascending pathways) carry sensory impulses toward the CNS. **Efferent pathways** (descending pathways) carry impulses away from the CNS to effector organs, such as muscles (smooth and skeletal) or glands **Figure 11-1**.

Physiologically, the nervous system consists of voluntary and involuntary divisions. The **voluntary (somatic) nervous system** is composed of nervous system fibers that connect the structures of the CNS with skeletal muscles and the integument. The **involuntary (autonomic) nervous system** is divided into the sympathetic and parasympathetic branches. Both of these branches are composed of nervous system fibers that connect the structures of the CNS with smooth muscle, cardiac muscle, and glands, which allow the system to regulate the body's internal environment by controlling organ systems. For example, the sympathetic nervous system is responsible for sweating, pupil dilation, ejaculation, and temperature regulation, as well as the shunting of blood from the periphery to the core—the "flight or fight" responses.

Central Nervous System

The CNS consists of the brain and the spinal cord, both of which are encased in and protected by bone. The brain, located within the cranial cavity, is the largest component of the CNS. It contains billions of neurons that serve a variety of vital functions.

Cranial Anatomy and Physiology

The brain is a very delicate, gelatinous-like substance that requires the protection afforded by the skull. Although primarily protective, excessive forces can cause a fracture to the adult skull and force bone

fragments into vulnerable brain tissue. The skull, or cranium, consists of the **neurocranium** (brain box) and the **viscerocranium** (the 14 bones that make up the facial skeleton).

The neurocranium is the part of the skull that encloses the brain and provides a protective vault for this vital organ **Figure 11-2**. It has a domelike roof—the calvaria (skullcap)—and a cranial base. The neurocranium includes eight bones: the frontal bone, paired parietal bones, paired temporal bones, an occipital bone, a sphenoid bone, and an ethmoid bone.

When the skull is observed from the inside, the superior surfaces form a smooth inner wall. In contrast, the basilar skull contains many ridges and folds with sharp edges that normally provide structure for the support of many parts of the brain **Figure 11-3**. When the head comes into contact with significant forces, the brain may shift over this base and become lacerated or contused by the sharp edges. This mechanism is a common culprit in many of the brain injuries encountered by the CCTP.

The base of the skull is also the location for openings, or foramina, which allow cranial nerves and blood vessels to enter and exit the cranial cavity. These openings also weaken the area, leaving it susceptible to fracture.

Special Populations

Children have extremely pliable skull bones and often have not healed their suture lines or closed their fontanelle prior to the age of 8 years. Excessive forces can cause a fracture in the adult skull, but in a child they are more likely to result in brain injury without skull fracture.

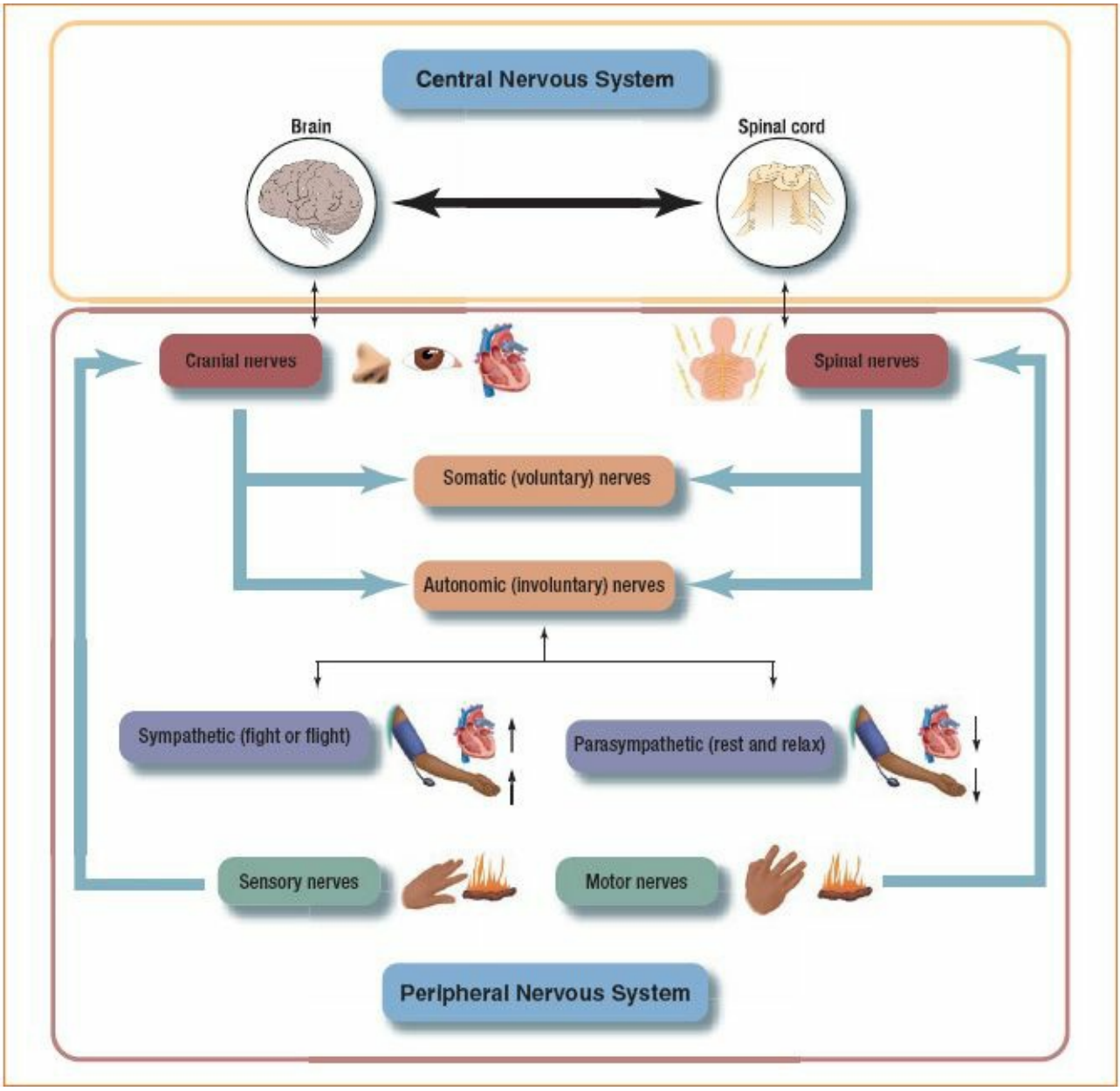


Figure 11-1 Organization of the nervous system.

Meningeal Anatomy and Physiology

The entire CNS is enclosed by a set of three membranes collectively known as the meninges **Figure 11-4**. The cranial meninges—internal to the neurocranium—protect the brain and form the supporting framework for arteries, veins, and venous sinuses. The cranial meninges consist of three layers: (1) the **dura mater**, an external thick, dense fibrous membrane; (2) the **arachnoid mater**, an intermediate, delicate membrane; and (3) the **pia mater**, an internal delicate, vascular membrane.

The dura mater is adherent to the internal surface of the cranium and is actually a two-layered membrane. The outer layer comprises the periosteum for the cranial bones and is the layer that actually adheres to the internal surface of the calvaria. The inner meningeal layer of the dura extends into the cranial space and is continuous at the foramen magnum with the dura covering the spinal cord. Except for those sites in which the two dural layers separate to form a dural venous sinus, the two layers are fused. The four extensions or folds of the dura mater are the falx cerebri, the tentorium cerebelli, the falx cerebelli, and the diaphragma sellae.

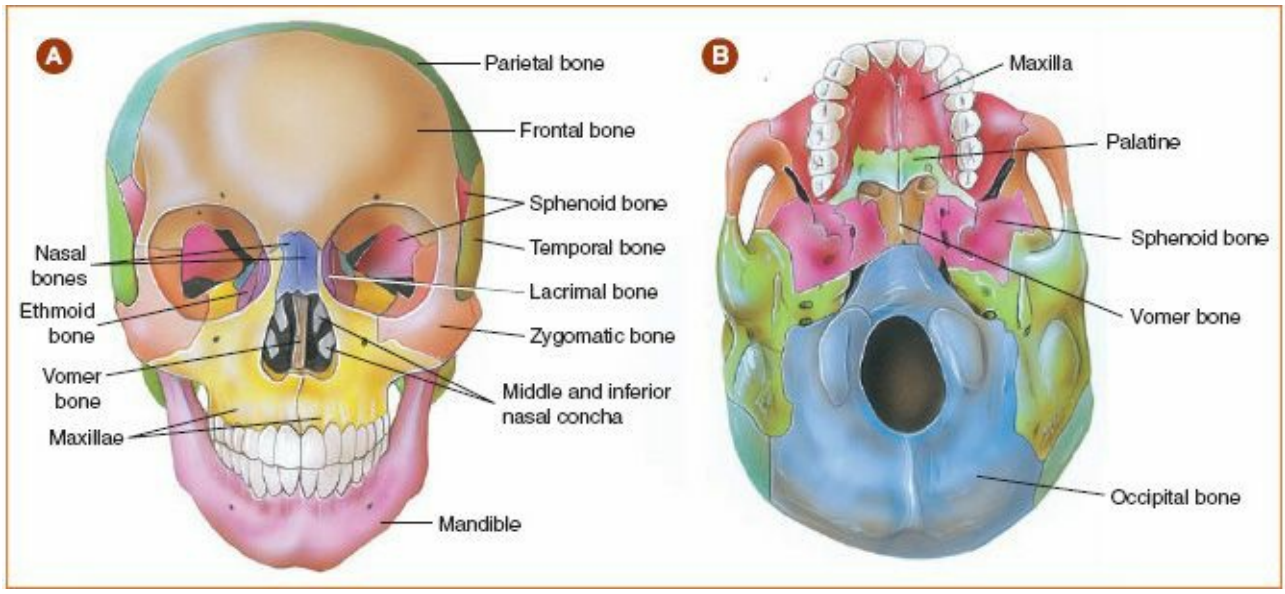


Figure 11-2 The skull and its components. **A.** Front view. **B.** Bottom view.

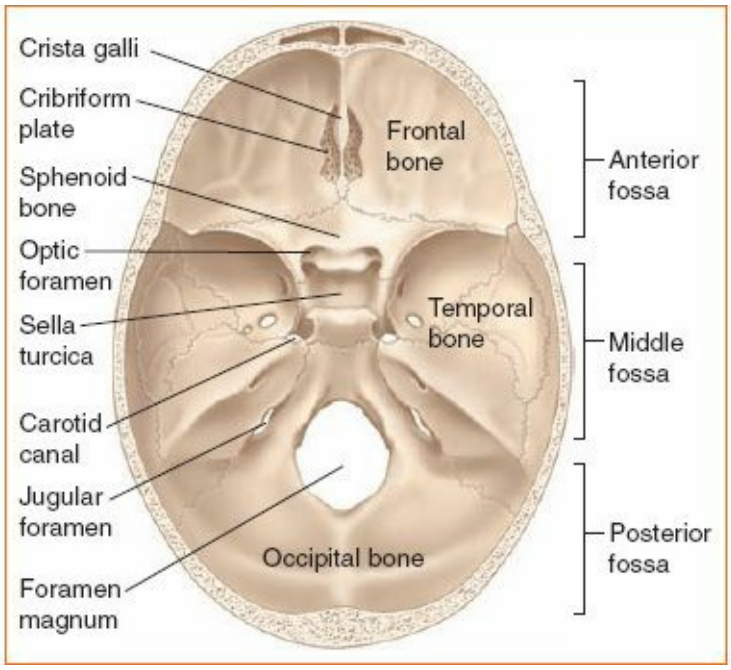


Figure 11-3 The floor of the cranial vault and its anatomy.

The **falx cerebri** is a double fold of dura mater that divides the cerebrum into right and left hemispheres by descending vertically into the longitudinal fissure that extends from the frontal lobe to the occipital lobe. The falx cerebri is the structure that is observed to *shift* on computed tomographic (CT) scan during times of brain swelling. It extends posteriorly by becoming continuous with the tentorium cerebelli.

The falx cerebri attaches to the **tentorium cerebelli** in the midline and holds it up, giving it a tent-like appearance. The tentorium cerebelli separates the occipital lobes of the cerebrum from the cerebellum and brain stem. This fold of the dura mater divides the brain into an upper compartment (supratentorial) and a lower compartment (infratentorial). The tentorium cerebelli is noted for its role in brain swelling, as it is the location where the **uncus** (the medially curved anterior part of the hippocampal gyrus) of the temporal lobe slips off of the tentorium and compresses cranial nerve (CN) III, which lies underneath the dural structure. This process causes a unilateral fixed and dilated pupil, characteristic of uncal herniation.

The **falx cerebelli** forms the division between the two lateral lobes of the cerebellum. The final extension of the dura mater, the **diaphragma sellae**, forms a roof over the sella turica, which contains the pituitary gland.

Further dural separations form the **dural venous sinuses**, which are endothelial-lined spaces between the periosteal and meningeal layers of the dura mater. Large veins from the surface of the brain empty into these sinuses, and all blood from the brain ultimately drains through them into the internal jugular veins. It is in these dural sinuses that arachnoid granulations—tufted prolongations of the arachnoid mater that protrude through the meningeal layer of the dura into the venous sinuses and transfer cerebrospinal fluid (CSF) to the venous system—are found.

The arachnoid mater is an extremely thin and delicate layer that loosely encloses the brain. It is closely applied to the meningeal layer of the dura mater by the pressure of the circulating CSF beneath the arachnoid layer. Web-like arachnoid trabeculae (small protrusions of connective tissue) pass between the arachnoid and pia, connecting the two layers and forming the subarachnoid space, which contains CSF and a variety of cerebral arteries and veins. Although it is commonly stated that the brain *floats* in CSF, the brain is actually suspended in the CSF-filled subarachnoid space by the arachnoid trabeculae. In essence, the meninges and CSF form a fluid-filled cushion that protects the brain and spinal cord.

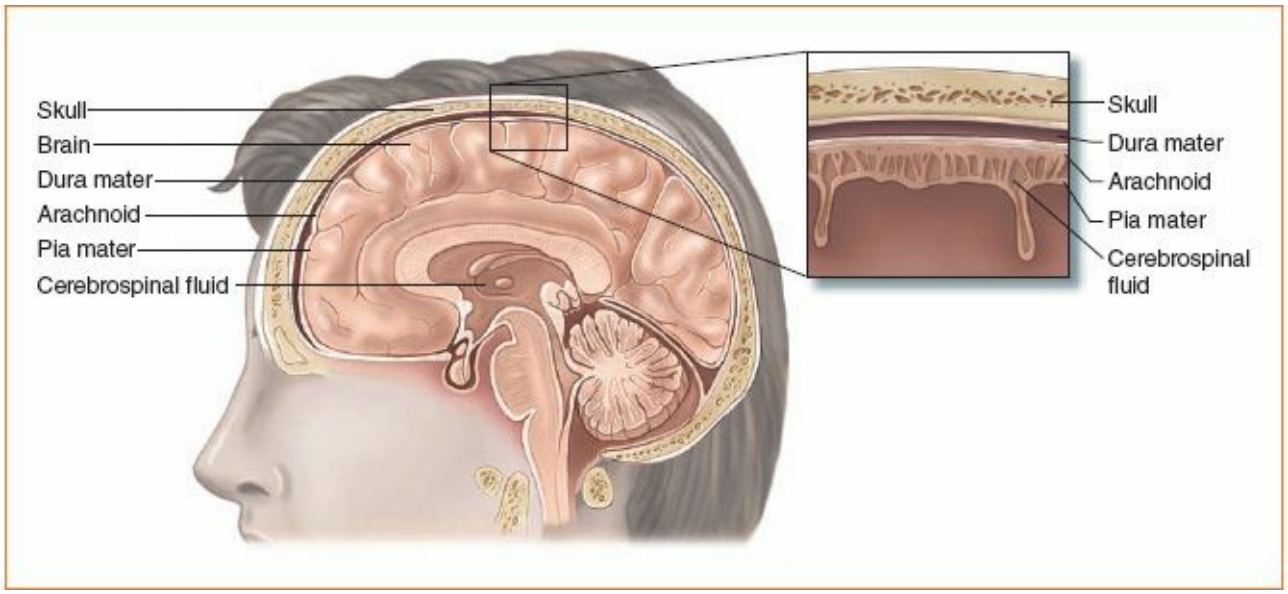


Figure 11-4 The meninges.

The pia mater is a vascular membrane that adheres to the surface of the brain and follows its contours. It is rich in small blood vessels that supply a large volume of arterial blood to cerebral tissues. In fact, when the cerebral arteries penetrate the cerebral cortex, the pia follows them for a short distance, forming a pial coat and creating a periarterial space. Tufts or folds of the pia mater in the lateral, third, and fourth ventricles form a portion of the choroid plexus, which is responsible for the production of CSF.

Three meningeal spaces are related to the cranial meninges. The **extradural space** or epidural space is normally not an actual space but only a potential one between the cranial bones and the periosteal layer of the dura. It becomes a real space only when blood from torn vessels pushes the periosteum from the cranium and accumulates. The dura–arachnoid junction, also called the **subdural space**, is normally only a potential space but may develop into a real one after a blow to the head causes a loss of blood into the area. The subarachnoid space between the arachnoid and the pia is actually a space that contains CSF, trabeculae, arteries, and veins.

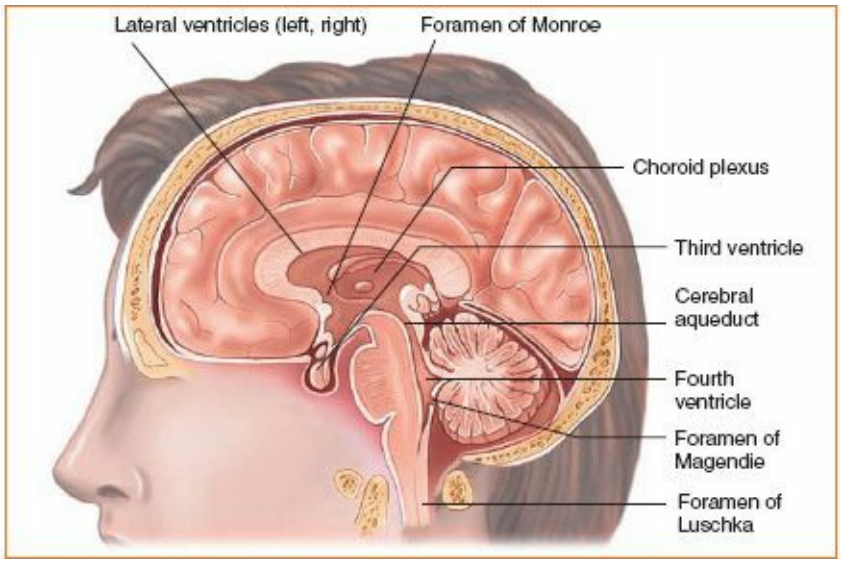


Figure 11-5 The ventricles of the brain and the cerebral aqueduct.

Ventricular System and CSF

The ventricular system is the central CSF-filled core of the brain. It consists mainly of two lateral ventricles (the largest ventricles) and midline third and fourth ventricles connected by the cerebral aqueduct **Figure 11-5**.

One lateral ventricle lies in each of the cerebral hemispheres. The lateral ventricles extend from the frontal lobe to the occipital lobe and have horns that extend from the main ventricular chamber. They are named for the respective area of the cerebrum in which they are contained. The lateral ventricles are important structures for **intracranial pressure (ICP)** monitoring, CSF drainage, or placement of a CSF shunt.

The **foramen of Monro** connects the two lateral ventricles with the third ventricle, a central cavity. Situated directly above the midbrain, the third ventricle lies between the structures of the thalamus in the diencephalon.

The **cerebral aqueduct**, the narrowest portion of the ventricular system, provides communication with the fourth ventricle, which lies between the brain stem and the cerebellum. Because of its small size, it is vulnerable to obstruction. At the base of the fourth ventricle, two openings—the **foramen of Luschka** and the **foramen of Magendie**—open the ventricular system into the subarachnoid space and are essential for the normal flow of CSF.

The **choroid plexus** of the lateral and the third and fourth ventricles produces more than 70% (approximately 500 mL/d) of the CSF by active transport and diffusion. Microscopically, the choroid plexus has the appearance of a delicate sponge with a collection of blood vessels covered by a thin coating of cells. CSF is constantly secreted from these surfaces. The remaining 30% of the CSF is produced from other sites within the ventricular system, including the ependymal cells lining the ventricles and blood vessels of the meninges and the blood vessels of the brain and spinal cord.

CSF fills the ventricular system and surrounds the brain and spinal cord in the subarachnoid space. This fluid provides a protective mechanism for the CNS by acting as a *shock absorber* for minor acceleration and deceleration and participates in the removal of waste products from cerebral tissue. CSF is normally clear, colorless, and odorless with a total volume of approximately 150 mL, of which only 23 mL is in the ventricles. The remaining 127 mL fills the subarachnoid space surrounding the brain and spinal cord. Under normal conditions, CSF is virtually cell free and low in proteins, glucose, potassium ions, and calcium ions relative to plasma. It is high, however, in magnesium, sodium, and chloride ions as compared to plasma. The normal composition of CSF is detailed in **Table 11-1**.

The CSF also has a slightly lower osmolality (289 mOsm/L) compared with serum (290 to 300 mOsm/L), which explains the transport of small molecules, metabolic products, and drugs from the surrounding brain tissue into the CSF.

No feedback mechanisms exist for the regulation of the rate or volume of CSF production. That is, the choroid plexus continues to produce CSF even if the ventricles and subarachnoid spaces are filled and the pressure is high. Consequently, any obstruction to the normal flow—either in the ventricular system or at the level of the arachnoid granulations—may produce high CSF pressure and lead to brain damage.

Once the CSF is formed in the ventricles, it flows within the closed system. Fluid formed in the two lateral ventricles passes into the third ventricle by way of the two foramina of Monro. The fluid then flows through the cerebral aqueduct into the fourth ventricle. The CSF exits the fourth ventricle by taking one of two paths. In the first path, fluid exits medially through the foramen of Magendie, where it is then directed to the subarachnoid space of the spinal cord. In the second path, fluid exits the fourth ventricle laterally through the two foramina of Luschka, where it is directed to the subarachnoid space surrounding the brain.

TABLE 11-1 CSF Composition

Property (Substance)	Value (Normal Range)
pH	7.35-7.45
Specific gravity	1.007
Glucose	50-75 mg/dL
Protein	5-25 g/dL
Pressure	70-200 mm H_2O (lumbar puncture) 3-15 mm Hg (brain ventricle)
Potassium	2.8-3.2 mEq/L
Sodium	147-151 mEq/L
Chloride	118-132 mEq/L
Calcium	1.05-1.35 mg/dL
Magnesium	0.78-1.26 mEq/L
Carbon dioxide	22.9 mEq/L

On entering the subarachnoid space, CSF moves over the surface of the spinal cord and brain to ultimately leave the subarachnoid space and enter the dural venous sinuses via the arachnoid villi. The arachnoid villi are very permeable oneway valves that allow CSF to exit easily from the subarachnoid space into the venous sinuses, but do not allow blood to enter the subarachnoid space.

Blockage of the movement of CSF into the dural venous sinuses leads to a condition known as communicating hydrocephalus. If CSF cannot leave the ventricular system, the condition is called noncommunicating hydrocephalus.

Anatomy and Physiology of the Brain

The brain gives members of the human species the ability to reason, function intellectually, express

personality and mood, and interact with their environment. The brain weighs approximately three pounds —a meager 2% of body weight—yet consumes 20% of the total cardiac output. The three major areas of the brain are the cerebrum, the brain stem, and the cerebellum.

Cerebrum

The cerebrum is, by mass, the largest part of the brain, accounting for 80% of its weight. It consists of two cerebral hemispheres that are incompletely separated by the longitudinal cerebral fissure. The cerebral hemispheres are connected at the base of the longitudinal cerebral fissure by the corpus callosum. The **corpus callosum** is a large tract of transverse fibers that provides a communication link between the two cerebral hemispheres. The hemispheres are further separated from the underlying brain stem and cerebellum by the transverse cerebral fissure. Both fissures contain meninges and thick folds of dura—specifically, the falx cerebri and the tentorium cerebelli.

The surface of the cerebrum is characterized by numerous convolutions called **gyri**. The gyri functionally increase the cortical surface area. Grooves between adjacent gyri are called **sulci**. Deeper grooves are referred to as **fissures**.

The **cerebral cortex** is the outermost layer of the cerebrum. This layer is 2- to 5-mm thick and contains billions of unmyelinated cell bodies of dendrites and neurons, which are often referred to as gray matter. Underneath the cerebral cortex are the white matter (myelinated) tracts, which communicate impulses from the cerebral cortex to other areas of the brain. Three types of fibers—commissural (transverse), projection, and association—are found in the white matter and are named for the role they play in communication of information:

- Commissural fibers are tracts that communicate between corresponding parts of the two hemispheres. The corpus callosum is the largest of these fiber tracts.
- Projection fibers communicate between the cerebral cortex and lower portions of the brain and spinal cord.
- Association fibers allow communication between various regions of the same hemisphere.

The **cerebral hemispheres** are divided into four paired lobes, based on the divisions demarcated by the fissures: the frontal lobes, the parietal lobes, the temporal lobes, and the occipital lobes **Figure 11-6**. Another area found deeper inside the cerebrum can also be classified as a lobe and is called the limbic lobe.

Frontal Lobe

The largest of the four lobes of the cerebral hemispheres is the **frontal lobe**, which accounts for approximately one third of the total cortical tissue. The frontal lobe lies underneath the frontal bone of the skull and is separated posteriorly from the parietal lobe by the central fissure and inferiorly from the temporal lobe by the lateral fissure. It is further divided into different areas based on the functions for which they are responsible **Figure 11-7**.

The **prefrontal area** of the frontal lobe is responsible for a variety of higher functions. This area provides control of thought, concentration, depth perception, the ability to think abstractly, memory, and autonomic nervous system response, concomitant to emotional change, with help from the thalamus and hypothalamus.

The **premotor area** is adjacent to the motor area. A connection between these areas exists with cranial nerves III, IV, VI, IX, X, and XII, allowing coordination of certain movements. For example, stimulation of the lateral portion results in gross generalized movements, such as turning of the eyes and head, turning of the trunk with the head, and coordinated eye movements.

The **motor area** contains pyramidal cells that control voluntary motor function on the opposite side of the body. Body parts that require a great deal of dexterity, such as the thumb and tongue, are allotted a larger motor area than body parts that do not require much dexterity, such as the shoulder and trunk.

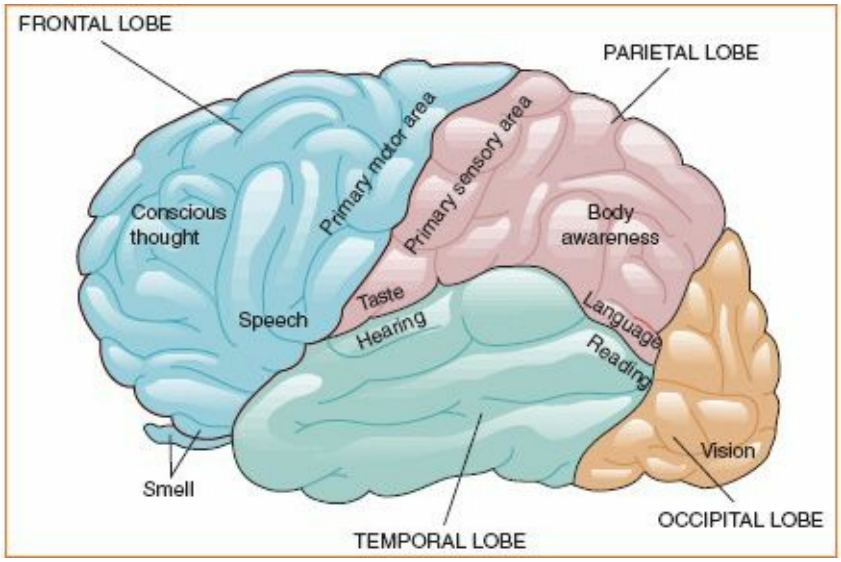


Figure 11-6 The lobes of the cerebrum.

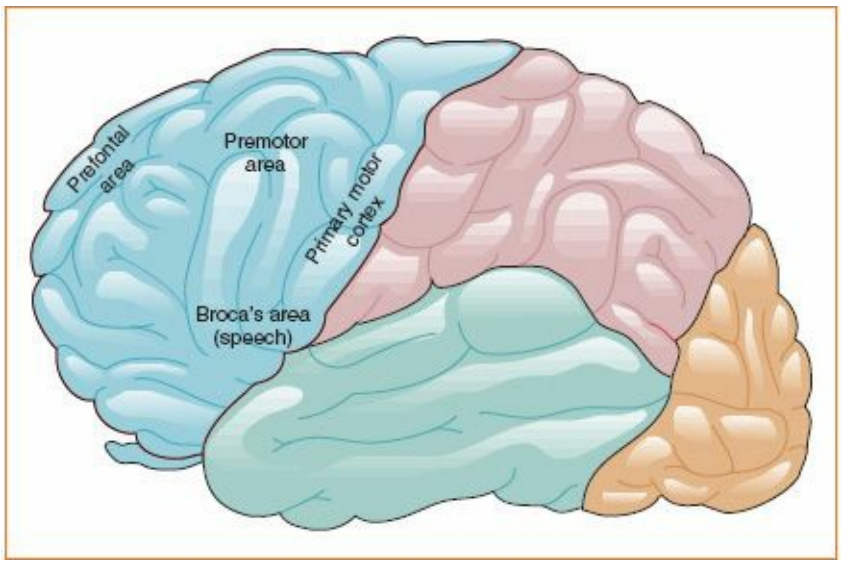


Figure 11-7 The cerebrum, highlighting the frontal lobe and its specific areas.

Broca's area is located at the inferior frontal gyrus. This association area participates in the formulation of words. Any damage to this area either from primary injury (direct trauma) or secondary injury (ischemia) results in expressive or nonfluent aphasia.

Parietal Lobe

The **parietal lobe** is situated directly posterior to the frontal lobe lateral to the central fissure. This lobe is largely responsible for sensory functions, including the integration of sensory information; awareness of body parts; interpretation of touch, pressure, and pain; and recognition of object size, shape, and texture. The parietal lobe consists of a sensory strip that lies adjacent to the motor strip of the frontal lobe. Sensory areas for certain parts of the body lie close to the motor areas for the same body part. Fibers going to the sensory strip bring stimuli associated with cutaneous and deep sensibility sensations, as well as cutaneous sensation of touch, pressure, position, and vibration. Input from the thalamus also reaches the

sensory strip.

Associative areas of the parietal lobe interpret sensory input in terms of size, shape, texture, and weight. They enable individuals to localize sensations and define them in terms of pressure, temperature, or vibration. Interpretive aspects of the parietal lobe's response to stimuli include awareness of body parts, orientation in space, and recognition of environmental spatial relationships.

Occipital Lobe

Occupying the most posterior portion of the cerebrum, the **occipital lobe** is separated from the cerebellum by the tentorium cerebelli. This area of the brain is the primary receptive area for vision, specifically the interpretation of visual stimuli. The primary visual cortex receives impulses from projections of the optic tract; these impulses are then referred to the visual associative areas for interpretation and integration. If a person sustains an injury to the visual associative areas, he or she will be able to see objects but will not be able to recognize or identify objects (visual agnosia).

Temporal Lobe

Located beneath the temporal bone of the cranium, the **temporal lobe** lies in the lateral portion of the cerebrum. It is separated from the frontal and parietal lobes by the lateral fissure. The primary functions of this region relate to hearing, speech, behavior, and memory.

Within the temporal lobe, the primary auditory receptive areas receive sound impulses and assist in determining the source of the sound and interpreting the meaning of the sound. The auditory association area is part of the superior temporal gyrus and is known as **Wernicke's area**. Usually the largest part of the dominant hemisphere, it is responsible for comprehension of both written and spoken words. If Wernicke's area is damaged, a person may hear sounds, but they will be meaningless (receptive aphasia).

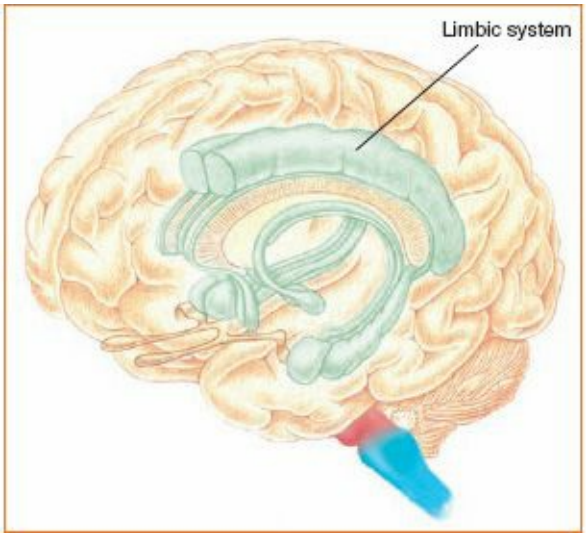


Figure 11-8 The limbic system is the seat of emotions, instincts, and other functions.

In the superior portion of the temporal lobe, at the junction of the frontal, parietal, and temporal lobes, lies the interpretative area. This area is responsible for integrating auditory, visual, and somatic association areas into complex thoughts and memory. It plays a significant role, along with the frontal lobe, in cerebration.

Limbic Lobe

The **limbic lobe** (also called the rhinencephalon) is properly an anatomic part of the temporal lobe,

although its function is sometimes discussed separately from the temporal lobe. This area forms the border of the lateral ventricles and contains the hippocampus, the uncus, the primary olfactory cortex, and the amygdaloid nucleus. Functions of the limbic lobe include self-preservation, primitive behavior, moods, the visceral processes associated with emotion, short-term memory, and the interpretation of smell **Figure 11-8**.

Diencephalon

The **diencephalon** is a major division of the cerebrum and is divided into four regions: the thalamus, the hypothalamus, the subthalamus, and the epithalamus. It is the lowest structure of the cerebrum and actually lies on top of the brain stem surrounding the third ventricle. Also found in this region is the pituitary gland and the internal capsule.

The **thalamus**, the largest portion of the diencephalon, consists of a pair of egg-shaped masses of gray matter that form the lateral walls of the third ventricle and are connected to the midbrain. It acts as a relay station for motor and sensory activity; basic neuronal activity, such as the processing of brain activity; and memory, thought, emotion, and complex behavior. The relay function of the thalamus is a complex function coordinated with the parietal lobe of the cerebrum. All sensory pathways, except for olfactory, communicate with an area of the thalamus. Once a sensory impulse reaches the thalamus, it sorts and sends the impulse to the appropriate area of the cerebral cortex for final processing. The role of the thalamus in motor activity is to coordinate the cerebrum and cerebellum to produce a smooth, integrated motor response.

TABLE 11-2 Functions of the Hypothalamus

Function	Mechanism
Temperature regulation	Temperature of blood flowing through the hypothalamus is monitored. Impulses are sent to the sweat glands, peripheral vessels, and muscles (shivering).
Regulation of food intake	The hunger center produces the sensation of hunger when stimulated. The satiety center decreases the desire for food when the stomach is full or blood glucose is high.
Regulation of water intake	Serum osmotic pressure is monitored and antidiuretic hormone is released when osmotic pressure is high, is inhibited when osmotic pressure is low.
Control of pituitary gland secretion	Releasing or inhibiting factors are sent from the hypothalamus to the pituitary gland.
Control of behavioral responses	Mediation of visible physical expressions in response to emotions, such as blushing, dryness of mouth, and clammy hands
Control of autonomic nervous system responses	The parasympathetic system response is elicited by stimulation of the anterior region of the hypothalamus. The sympathetic system response is mediated by the posterior region of the hypothalamus.

Located below the thalamus, the **hypothalamus** forms the floor and the anterior walls of the third

ventricle. The pituitary gland is situated below the hypothalamus in the sella turcica and is connected to the hypothalamus by the pituitary stalk. The hypothalamus has several functions related to regulating and maintaining the internal body environment and interacting with the limbic system to generate physical responses to emotions. Hypothalamic function falls into two major areas: (1) the maintenance of homeostasis (ie, internal balance) and (2) the implementation of behavioral patterns. The hypothalamus exerts its influence through the endocrine system and neuronal pathways **Table 11-2**.

The **epithalamus** is located in the **dorsal** portion of the diencephalon and contains the pineal gland, which is thought to play a role in physical growth and sexual development. The **subthalamus** is located below the thalamus and is closely related to the basal ganglia in function.

Basal Ganglia

The **basal ganglia** are several masses of nuclei located deep in the cerebral hemispheres. The main role of the structures is associated with fine motor function, particularly of the hands and lower extremities. They provide a pathway and assist in processing information from the cerebral motor cortex and the thalamus. Much of the basal ganglia's function is conducted through the extrapyramidal (involuntary) pathways.

Internal Capsule

As radiation and projection fibers coming from various parts of the cerebral cortex converge at the brain stem, they form the corona radiata. As the fibers enter the thalamus–hypothalamus region, they are collectively called the **internal capsule**. The capsule is a massive bundle of efferent and afferent fibers connecting the various subdivisions of the brain and spinal cord.

Brain Stem

The brain stem acts as a bridge between the cerebral hemispheres and the spinal cord; it is the only structure connecting the cerebellum with the cerebral cortex. All motor and sensory fibers travel through the brain stem—both those ascending to the cerebral hemispheres and those descending to the spinal cord. The brain stem consists of three parts: the midbrain, the pons, and the medulla oblongata.

The **midbrain** is a small area extending between the diencephalon, the pons, and the third ventricle **Figure 11-9**. Several important structures are found in the midbrain, including the cerebral aqueduct (Sylvian, which connects the third and fourth ventricles), the superior colliculi (which controls visual reflex and the center for upward gaze) and inferior colliculi (auditory system), cranial nerves III (oculomotor) and IV (trochlear), and the reticular activating system (RAS).

The major function of the midbrain is to relay stimuli involved in voluntary motor movement of the body. Also found in the region are the tectospinal and rubrospinal tracts of the extrapyramidal (involuntary) motor system. The **tectospinal tract** controls reflex motor movements in response to visual and auditory stimuli, and the **rubrospinal tract** controls the tone of flexor muscles. The **Edinger-Westphal nucleus** is located in the midbrain and is responsible for mediating the autonomic reflex centers for pupillary accommodation to light.

Located between the midbrain and the medulla oblongata **Figure 11-10**, the **pons** relays information to and from the brain and spinal cord along fiber tracts. Motor tracts for motor movement descend through the pons, and sensory tracts necessary for touch, pressure, proprioception, pain, and temperature ascend through the pons. The **ventral** surface of the pons contains fibers that connect to the cerebellum, which allows for efficient and smooth transmission of influences from the cerebellum to the cerebral cortex. Two respiratory control centers are found in the pons: the apneustic center and the pneumotaxic center. The apneustic center is responsible for stimulating and producing sustained respirations. The pneumotaxic

center antagonizes the apneustic center, clinically inhibiting inspiration. Both centers of control communicate with the medullary respiratory center. Cranial nerves V (trigeminal), VI (abducens), VII (facial), and VIII (acoustic) originate in the pons.

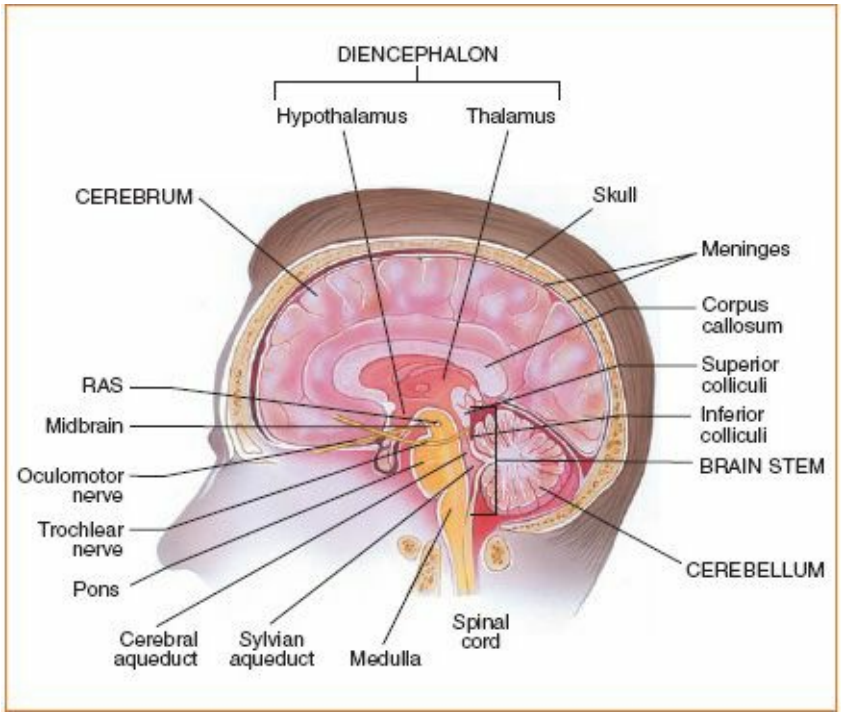


Figure 11-9 The midbrain.

The **medulla oblongata**, the lowermost portion of the brain stem, connects with the spinal cord at the foramen magnum. In the caudal aspect of the medulla, decussation (crossing) of the motor fibers occurs. Inferior to the point of decussation, stimuli from the left side of the brain control movement on the right side of the body, and vice versa.

The medulla also contains groups of neurons that control involuntary functions such as swallowing, vomiting, coughing, vasoconstriction, and respiration. The respiratory center of the medulla works in conjunction with the apneustic and pneumotaxic centers of the pons to produce controlled respirations. The medullary respiratory control center is responsible for the basic involuntary rhythm of respiration, but must be stimulated from higher centers to maintain a life-sustaining respiratory pattern. The reticular formation also has its beginnings in the medulla, with cranial nerves IX (glossopharyngeal), X (vagus), XI (spinal accessory), and XII (hypoglossal) exiting from the medulla.

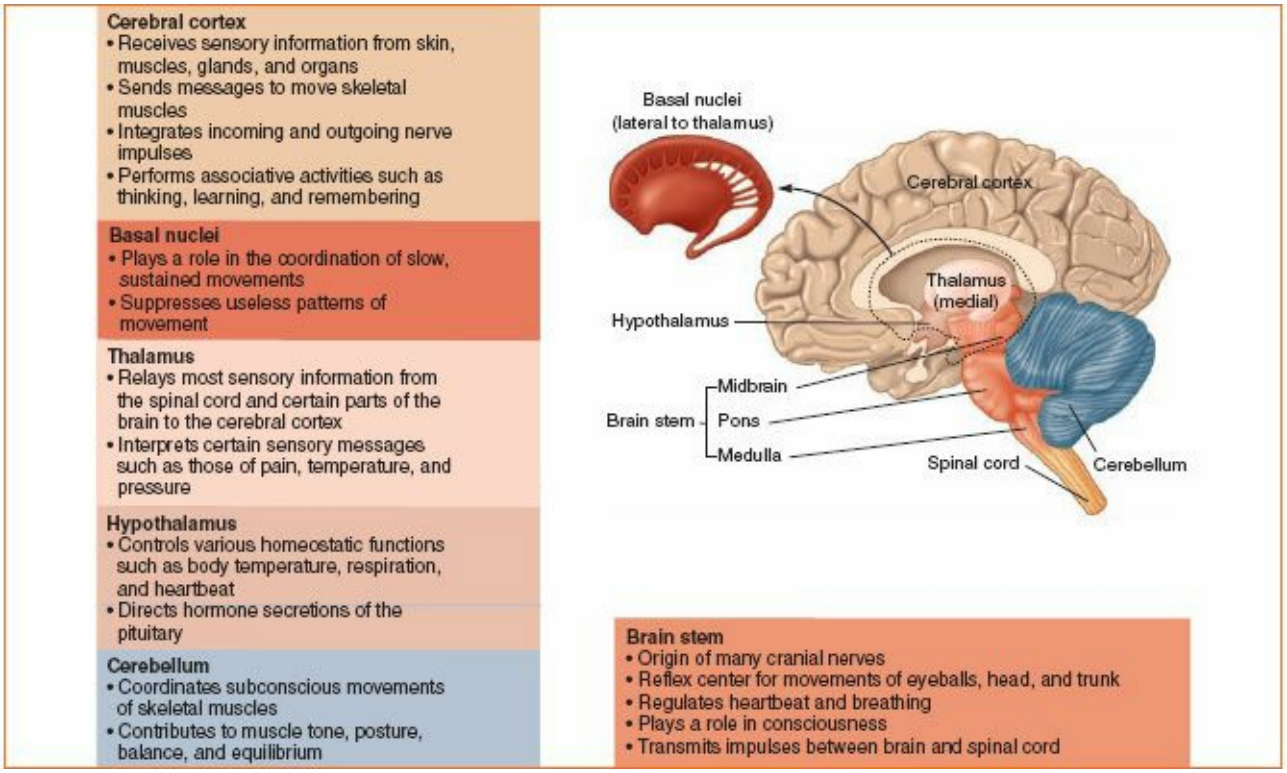


Figure 11-10 The pons.

Cerebellum

The cerebellum is located just superior and posterior to the medulla oblongata and consists of three major parts: (1) the cortex, or the gray outer covering; (2) the white matter, which connects the cerebellum to other parts of the CNS; and (3) four pairs of deep cerebellar nuclei, located deep within the white matter. The gross structure of the cerebellum includes two distinct hemispheres and the vermis, or central portion **Figure 11-11**.

The primary function of the cerebellum is coordination of voluntary movement. The vermis is responsible for trunk control. The three lobes of the hemispheres are responsible for upper and lower extremity control, including control of anti-gravity muscles, proprioception, tactile impulses, motor tone facilitation, volitional breaks in movement, synergy of movement, and equilibrium. Input is received from sensory pathways of the spinal cord, the brain stem, and the cerebrum. Output is transmitted through descending motor pathways. Cerebellar influences work through continual excitatory and inhibiting stimuli, resulting in smooth motor movements instead of rapid, erratic movements.

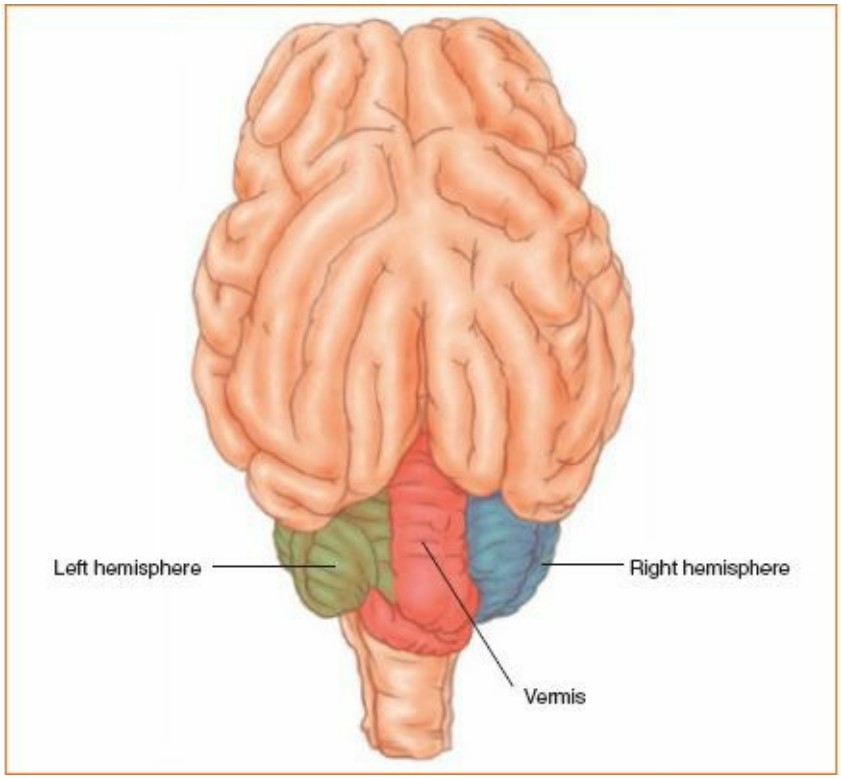


Figure 11-11 The vermis and the hemispheres of the cerebellum.

Reticular Formation

The **reticular formation (RF)** is a set of neurons that extends from the upper level of the spinal cord, through the medulla, pons, and midbrain, and into the thalamus and cerebral cortex. Composed of both motor and sensory tracts, the RF is closely tied to functions of the basal ganglia, thalamus, cerebellum, and cerebral cortex. This bundle of neural fibers has many excitatory and some inhibitory capabilities, and can enhance, suppress, or modify impulse transmission. The main role of the descending RF is to provide a balance between the excitatory and inhibitory stimuli so as to maintain normal muscle tone, which supports the body against gravity. Also located in the RF are centers for blood pressure, respiration, and heart rate function. The ascending RF is essential for arousal, attention, and perceptual association.

Reticular Activating System

The **reticular activating system (RAS)** is a diffuse system, not an actual anatomic structure, that extends from the lower brain stem to the cerebral cortex. Input from multiple sensory pathways is received, and signals are transmitted to dispersed areas of the cerebral cortex, providing multiple opportunities for stimulation of the RAS. The lower portion of the RAS, which is rooted in the brain stem, assists with control of the sleep–wakefulness cycles and consciousness; the upper portion, which is in the thalamus, allows the ability to focus attention on a specific task. When damage occurs to the upper portion of the RAS, the person enters a vegetative state, exhibiting sleep-wake cycles and other brain stem functions but no upper levels of cerebration.

Cerebral Circulation

The brain has huge demands—15% to 20% of total cardiac output and 40% of the oxygen in the available blood to meet normal cerebral metabolic needs. The predominant metabolic process that occurs is the oxidation of glucose to provide energy. Therefore, the overall goal of cerebral circulation is to provide enough blood to supply oxygen, glucose, and nutrients for this process.

The total amount of blood received by the brain is distributed carefully among areas where metabolic demands are high. Carbon dioxide serves as the primary regulator for blood flow in the CNS. A potent vasodilator, its presence causes more blood to flow to certain areas of the brain. The brain is supplied blood by two pairs of arteries: the two vertebral arteries and the two internal carotid arteries **Figure 11-12**.

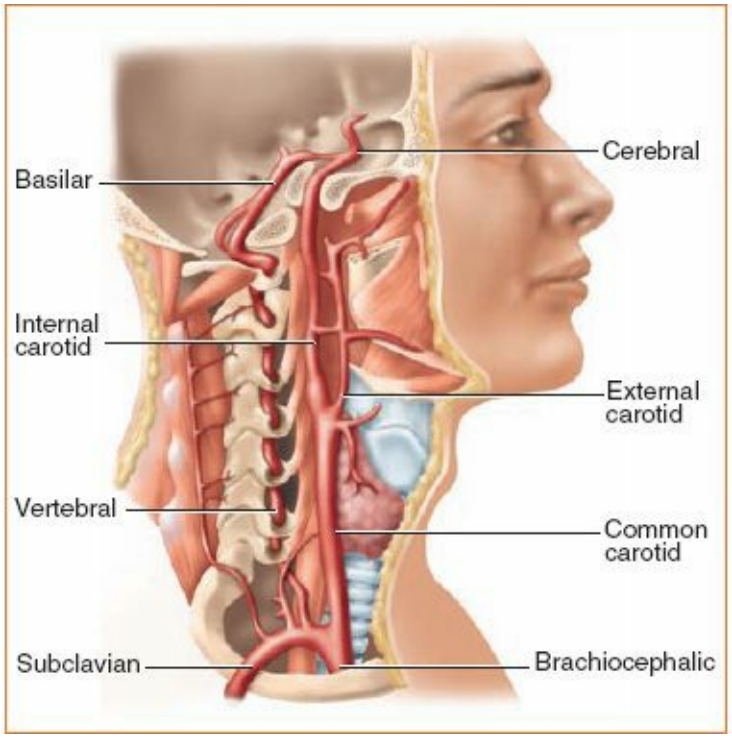


Figure 11-12 Blood supply to the brain via arteries.

Circle of Willis

The **circle of Willis** is a system of arteries located at the base of the skull that is divided into anterior and posterior circulation **Figure 11-13**. It is physiologically significant in that this structure may be able compensate for reduced blood flow from any one of the major contributors to cerebral circulation. Some individuals, however, may have hypoplastic or absent communicating vessels that may inhibit this compensatory mechanism.

The circle of Willis is fed by the internal carotid and basilar arteries. The three cerebral arteries (anterior, middle, and posterior) supplying each hemisphere are connected by communicating arteries to form a complete circle. **Table 11-3** lists the areas of the brain supplied by the cerebral arteries.

Vertebral Arteries

The vertebral arteries, or posterior circulation, arise from the subclavian arteries and enter the skull through the foramen magnum, ventrolateral to the spinal cord. At the level of the pons, the two vertebral arteries join to form the basilar artery. The basilar artery then divides at the level of the midbrain to form paired posterior cerebral arteries. The vertebral arteries and their branches predominantly supply blood to the cerebellum, brain stem, and spinal cord, as well as to some parts of the cerebrum. The basilar artery continues upward into the posterior portion of the circle of Willis and into the two posterior cerebral arteries.

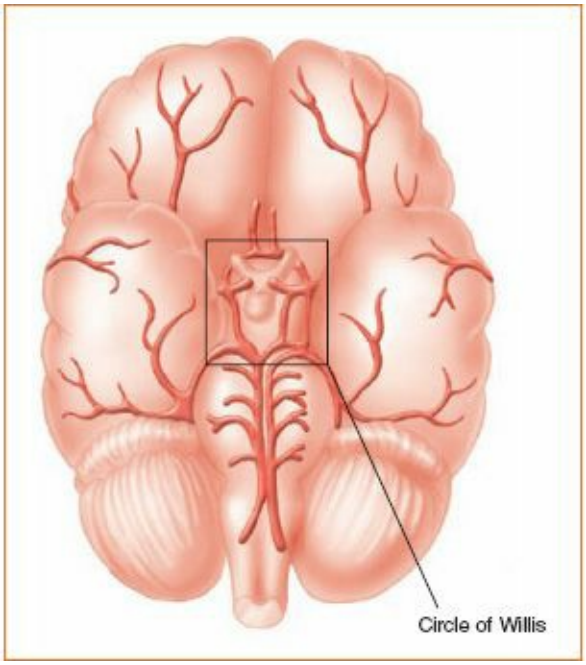


Figure 11-13 The circle of Willis.

TABLE 11-3 Major Cerebral Arteries	
Cerebral Artery Origin	**Structures Supplied**
Anterior	Basal ganglia, corpus callosum, medial surface of cerebral hemispheres, and superior surface of frontal and parietal lobes
Middle	Frontal lobe, parietal lobe, and cortical surface of temporal lobe
Posterior	Occipital lobes and the medial and lateral aspects of the temporal lobe

Internal Carotid Arteries

The internal carotid arteries anteriorly supply a proportionately greater amount of blood flow. They originate from the common carotid arteries and enter the cranium through the base of the skull. At the base of the brain, the internal carotid arteries connect to the circle of Willis and then branch into the anterior and middle cerebral arteries, which provide the majority of anterior circulation.

Venous Drainage

The superficial and deep veins of the brain enter the dural venous sinuses, which subsequently drain into the internal jugular veins. Cerebral veins on the superolateral surfaces of the brain drain into the superior sagittal sinus. Cerebral veins on the postero-inferior aspect drain into the straight, transverse, and superior petrosal sinuses **Figure 11-14**.

Blood-Brain Barrier

The internal environment of the brain is kept in delicate balance by several protective mechanisms. Particularly noteworthy in this respect is the **blood-brain barrier**, the network of endothelial cells and astrocytes (neuroglia) that envelop delicate cerebral capillaries. The blood-brain barrier is found throughout the brain except in small areas of the fourth ventricle, hypothalamus, and pineal gland, where chemoreceptors and osmoreceptors sample circulating plasma. The barrier regulates the transport of nutrients, ions, water, drugs, and waste products through the process of selective permeability.

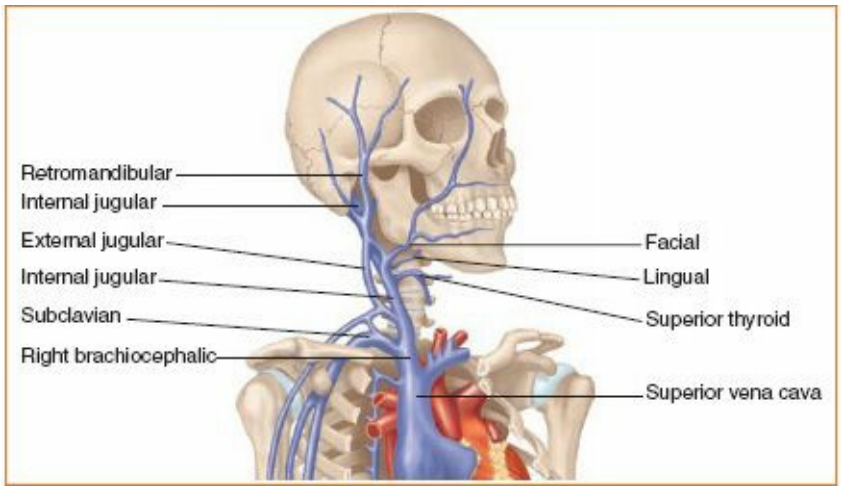


Figure 11-14 The veins of the neck.

Functionally, the blood-brain barrier has tight junctions between adjacent capillary endothelial cells, as opposed to the capillaries found elsewhere in the body, which have pores between adjacent endothelial cells. Molecules are transported through the endothelial cells in order to keep out toxic or harmful compounds and protect the fragile neurons.

Passage of substances across the blood-brain barrier depends on the particles' size, lipid solubility, chemical breakdown, and protein-binding potential. The barrier is readily permeable to water, oxygen, carbon dioxide, and glucose. Most drugs or compounds that are lipid soluble and stable at a physiologic pH rapidly cross the barrier as well. Uptake of sodium and potassium is slow, however, as is uptake of dyes and other organic and inorganic anions and cations.

Anatomy and Physiology of the Spinal Cord

An understanding of the form and function of spinal anatomy coupled with a high level of suspicion for spinal cord injury (SCI) is required to decipher the often subtle findings associated with a possible SCI.

Spine

The spine usually consists of 33 irregular bones (vertebrae) articulating to form the vertebral column, which is the major structural component of the axial skeleton **Figure 11-15**. Some patients may have 32 or 34 vertebrae. These skeletal components are stabilized by both ligaments and muscles. Together these components support and protect neural elements while allowing for fluid movement and erect stature.

Vertebrae are identified according to their location as cervical, thoracic, lumbar, sacral, or coccyx. The **vertebral body**, the anterior weight-bearing structure, is made of bone that provides support and stability. Components of the vertebra include the lamina, pedicles, and spinous processes **Figure 11-16**. Each vertebra is unique in appearance and, with the exception of the atlas and axis (C1 and C2, respectively) **Figure 11-17**, shares basic structural characteristics.

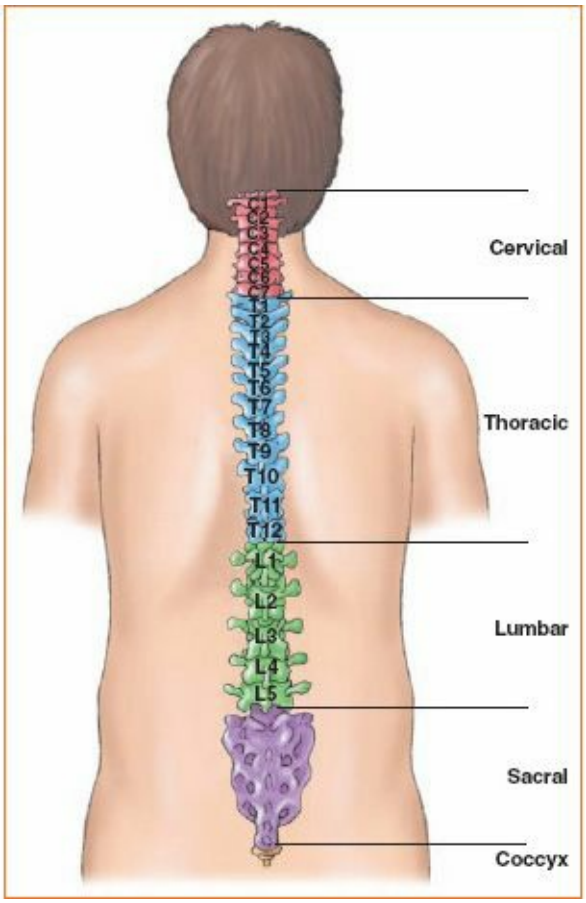


Figure 11-15 The sections of the spinal column, with the letter/number of each vertebra labeled.

The inferior border of each pedicle contains a notch forming the **intervertebral foramen**. This space in the middle of the vertebra allows the exit of a peripheral nerve root and spinal vein as well as the entrance of a spinal artery on both sides at each vertebral junction.

The transverse spinous processes comprise the junction of each pedicle and lamina on each side of a vertebra. They project laterally and posteriorly and form points of attachments for muscles and ligaments. The posterior spinous process is formed by the fusion of the posterior lamina and serves as an attachment site for muscles and ligaments.

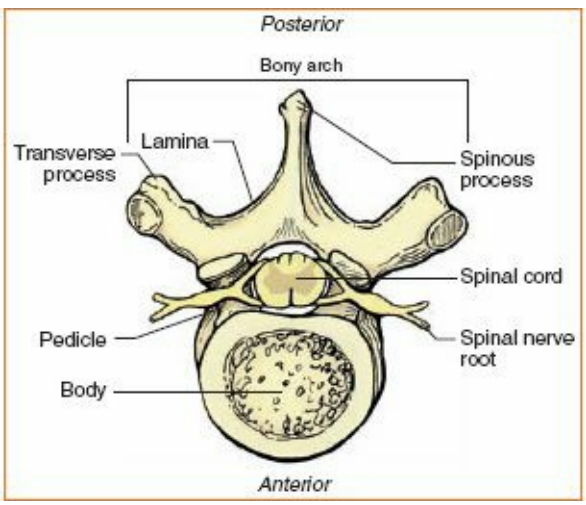


Figure 11-16 The human vertebra.

The cervical spine includes the first seven bones of the vertebral column and its supporting structures. In addition to protecting the vital cervical spinal cord, the cervical spine supports the weight

of the head and permits a high degree of mobility in multiple planes. The atlas (C1) and axis (C2) are uniquely suited to allow for rotational movement of the skull.

The thoracic spine usually consists of 12 vertebrae in addition to the supporting muscles and ligaments found in the vertebral column; the thoracic spine is further stabilized by the rib attachments. The spinous processes are slightly larger, reflecting their role as attachment points for muscles that hold the upper body erect and assist with the movement of the thoracic cavity during respiration. In patients with greater than five lumbar vertebrae, there will be less than 12 thoracic vertebrae. Although this may seem insignificant, for the purposes of radiographic interpretation, thoracic and lumbar bodies are usually counted together rather than separately.

The lumbar spine includes the five largest bones in the vertebral column, and is integral in carrying a large portion of the upper body weight. The lumbar spine is especially susceptible to injury because of this weight-bearing capacity.

The sacrum is composed of five fused vertebrae that form the posterior plate of the pelvis. The coccyx is made up of three to five small fused vertebrae. Coccyx injuries, although often extremely painful, are typically clinically insignificant.

Each vertebra is separated and cushioned by intervertebral disks that limit bone wear and act as shock absorbers. As the body ages, these disks lose water content and become thinner, causing the height loss associated with aging. Stress on the vertebral column may cause a disk to herniate into the spinal canal, resulting in a spinal cord or nerve root injury **Figure 11-18**.

The muscles, tendons, and ligaments that connect the vertebrae allow the spinal column a degree of flexion and extension, limited to an extent by the stabilization they must provide to the spinal column. The vertebral column can sustain normal flexion and extension of 60% to 70% without stressing the spinal cord. Flexion or extension beyond those limits may damage structural ligaments and allow excess vertebral movement that could expose the spinal cord to injury.

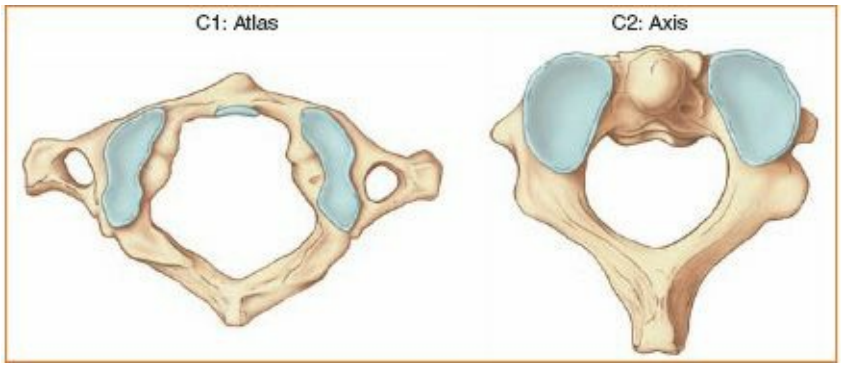


Figure 11-17 Superior view of the structure of the atlas and axis.

Spinal Cord

The spinal cord transmits nerve impulses between the brain and the rest of the body. Located at the base of the brain, it represents the continuation of the CNS. This bundle of nerve fibers leaves the skull through a large opening at its base called the **foramen magnum**. The large size of the foramen magnum relative to other foramina makes it a common location for brain herniation.

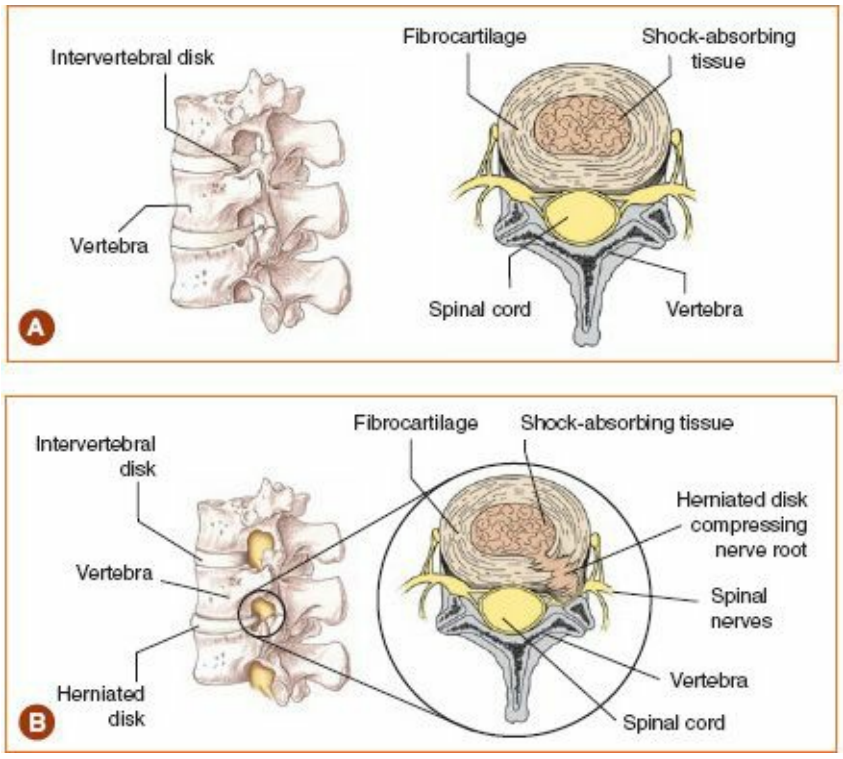


Figure 11-18 **A.** Normal, uninjured vertebral disk. **B.** Herniated disk.

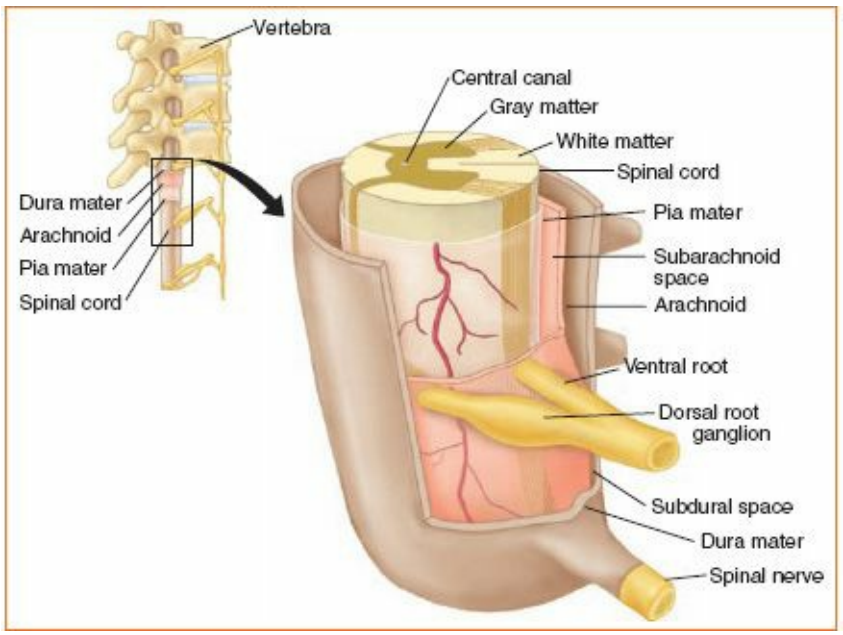


Figure 11-19 The spinal cord and its layers. The meninges enclose the brain and spinal cord.

The spinal cord extends from the base of the skull to L2; here it separates into the **cauda equina**, a collection of individual nerve roots. Thirty-one pairs of spinal nerves arise from the different segments of the spinal cord; each pair is named according to its corresponding segment.

A cross-section of the spinal cord **Figure 11-19** reveals a butterfly-shaped central core of gray matter that is composed of neural cell bodies and synapses. This gray matter is divided into posterior (dorsal) horns, which carry sensory input, and anterior (ventral) horns, which innervate the motor nerve of that segment. Surrounding the gray matter on each side are three columns of peripheral white matter composed of myelinated ascending and descending fiber pathways. Messages are relayed to and from the brain through these spinal tracts.

The brain stem connects the spinal cord to the remainder of the brain. All but two of the 12 cranial

nerves exit from the brain stem. Specific groups of nerves are named based on their source of origin and point of termination. Ascending tracts carry information to the brain, and descending tracts carry information to the rest of the body **Table 11-4**.

Peripheral Nervous System

Spinal Nerves

The 31 pairs of spinal nerves emerge from each side of the spinal cord and are named for the vertebral region and level from which they arise. The eight cervical roots perform different functions in the scalp, neck, shoulders, and arms. The 12 thoracic nerve roots have varying functions; the upper thoracic nerves supply muscles of the chest that help in breathing and coughing, whereas the lower thoracic nerves provide abdominal muscle control and contain nerves of the sympathetic nervous system. The five lumbar nerve roots supply hip flexors and leg muscles, as well as provide sensation to the anterior legs. The five sacral nerves provide for bowel and bladder control, sexual function, and sensation in the posterior legs and rectum. The coccyx has a single nerve root.

TABLE 11-4 Major Spinal Tracts

Spinal Tract	Function
Anterior	
Anterior spinothalamic tracts (ascending)	Carry sensation of crude touch and pressure sensation to the brain
Lateral spinothalamic tracts (ascending)	Carry pain and temperature
Spinocerebellar tracts (ascending)	Coordinate impulses necessary for muscular movements by carrying impulses from muscles in the legs and trunk to the cerebellum
Corticospinal tracts (descending)	Voluntary motor commands
Reticulospinal tracts (descending)	Muscle tone and sweat gland activity
Rubrospinal tracts (descending)	Muscle tone
Posterior	
Fasciculus gracilis and cuneatus	**Proprioception**, vibration, light touch, deep pressure, **two-point discrimination**, and **stereognosis**

Nerve roots occasionally converge in a cluster called a **plexus** that permits peripheral nerve roots to rejoin and function as a group **Figure 11-20**. For example, the cervical plexus includes C1 through C5; the phrenic nerve (C3–C5) arises from this plexus and innervates the diaphragm. The brachial plexus (C5–T1) joins nerves controlling the upper extremities; the main nerves arising from this plexus are the axillary, median, musculocutaneous, radial, and ulnar. The lumbar plexus (L1–L4) supplies the skin and muscles of the abdominal wall, external genitalia, and part of the lower limbs. The sacral plexus (L4–S4) gives rise to the pudendal and sciatic nerves and supplies the buttocks, perineum, and most of the lower

limbs.

Sympathetic Nervous System

The sympathetic nervous system is controlled by the brain's hypothalamus. Information from the brain is transmitted through the brain stem and the cervical spinal cord and then exits at the thoracic and lumbar levels of the spine to reach target structures. The thoracolumbar system provides sympathetic stimulation to the periphery largely through alpha and beta receptors. Alpha receptor stimulation induces smooth muscle contraction in blood vessels and bronchioles. Beta receptors respond with relaxation of smooth muscles in blood vessels and bronchioles, and have chronotropic and inotropic effects on myocardial cells.

An SCI at or above the level of T6 may disrupt the flow of sympathetic communication. Loss of sympathetic stimulation can disrupt homeostasis and leave the body poorly equipped to deal with changes in its environment. Stimulation of sympathetic nerves without parasympathetic input can cause sympathetic overdrive, resulting in autonomic dysreflexia.

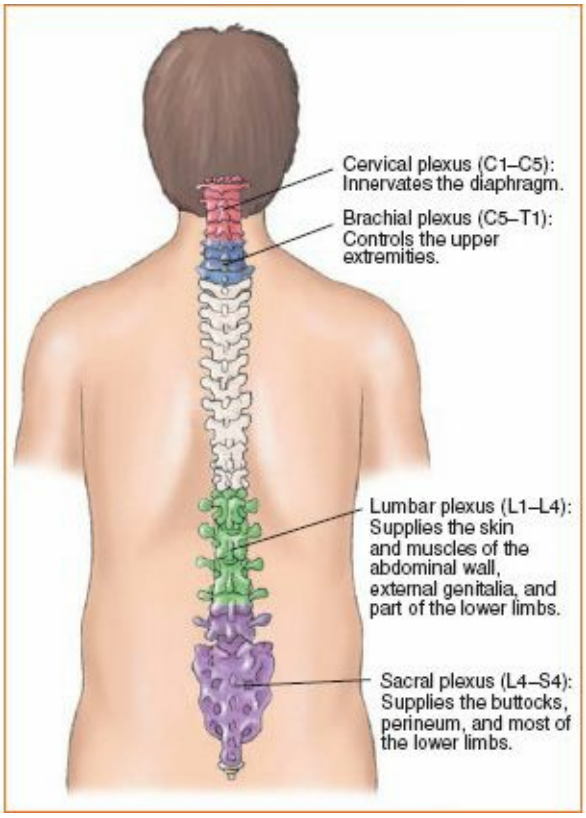


Figure 11-20 Nerve roots converge in plexuses, allowing them to function as a group.

Parasympathetic Nervous System

The parasympathetic nervous system includes fibers arising from the brain stem and upper spinal cord that carry signals to organs of the abdomen, heart, lungs, and the skin above the waist. The vagus nerve travels from its origins outside of the medulla to the heart via the carotid arteries; thus, vagal tone remains intact following a spine injury. When the sympathetic nerves are stimulated and produce autonomic dysreflexia, the parasympathetic nerves attempt to control the rapidly increasing blood pressure by slowing the heart rate. Parasympathetic nerves that supply the reproductive organs, pelvis, and legs begin at the sacral level (S2–S4). The parasympathetic nervous system is also responsible for stimulating salivary gland secretions and peristalsis, micturition (urination), and sexual arousal (erection). Disruption of the lower parasympathetic nerves in the sacrum results in the loss of bowel/bladder tone and sexual function.

Neurologic Examination

The nervous system is a delicate matrix of individual parts that, when functioning properly, afford humans the ability to maintain internal (and external) balance. Although well protected, this system may be affected by both illness and injury. When this occurs, competent medical care often makes the difference in good vs poor outcomes. The foundation of this care is a thorough serial assessment. This integral part of patient care not only allows the tracking of patient trends, but also may allow the CCTP to identify which parts of this delicate system are damaged.

Critical patients with neurologic complications present a variety of examination and management challenges during care and transport. Examination of the patient with neurologic dysfunction is the essential starting point of the entire patient management process. Only when the CCTP has performed a thorough baseline examination can appropriate field diagnoses be made and trends in patient condition followed. The neurologic field diagnosis of the impaired patient is a deductive process that represents a culmination of all of the details from the history, the examination, and laboratory/radiographic studies that may be available. As a CCTP, you will be expected to confidently and competently perform and document a thorough neurologic assessment. The neurologic examination is divided into four main segments: mental and emotional status and assessments of cranial nerves, motor function, and sensory function.

History

The neurologic examination begins at the onset of patient contact. Simple conversation with the patient and family members may prove to be a valuable tool in the examination. If taking a history from the critically ill patient is not possible, the CCTP must also obtain information from secondary sources—for example, other EMS reports, emergency department charts, medical records from previous admissions, and family and friends. The latter individuals can provide information, and also verify patient responses to questions.

The history should focus on the patient's chief complaint and should explore related problems that may influence the chief complaint. The following is a list of significant information that should be obtained from the history:

- The patient's normal baseline status
- Recent trauma that could potentially affect the nervous system
- Recent infections, including sinus, ear, or tooth infections
- Recent headaches or difficulty concentrating
- Feelings of dizziness, loss of balance, blackout episodes, tinnitus, or hearing problems
- Clumsiness or weakness of the extremities
- Numbness, tingling, hypersensitivity, or pain
- Impotence or incontinence (urinary or fecal)
- Visual symptoms such as double vision, blurred vision, or loss of vision
- Tobacco, alcohol, or drug use
- Recent difficulties performing everyday activities, including job performance and social interaction
- SAMPLE history

Physical Examination

Mental and Emotional Status

The mental/emotional component of the neurologic examination assesses the patient's ability to understand and interact with the environment. Specific areas tested in this component include level of consciousness; general behavior; and thought process, including memory, attention and concentration, abstract thought, and judgment. Not every component of the examination will be relevant in all critical care situations and, therefore, may not be tested. Nevertheless, the CCTP should understand how all components are integrated and how they influence decision making for patient care.

Assessment of the level of consciousness (LOC) is an extremely important component of the neurologic examination and should be performed on all patients. The patient's ability to perform should be taken into consideration, as it may be necessary to modify assessment techniques. For example, intubated patients who are otherwise awake and aware may gesture or write answers to questions instead of verbalizing them.

Awareness and arousal are the fundamental constituents of consciousness and should be evaluated and documented repeatedly. The evaluation of arousal is essentially an assessment of the patient's RAS and its connection with the thalamus and the cerebral cortex. Arousal—the lowest LOC—focuses on the patient's ability to respond to a variety of stimuli, and may be described using the AVPU scale **Table 11-5** or terms such as "disoriented," "lethargic," or "obtunded" **Table 11-6**.

In most critical care settings, where time for gathering data may be limited, the Glasgow Coma Scale (GCS), discussed in Chapter 10, is commonly used to document and trend a patient's level of arousal. This scale evaluates three parameters: eye opening, verbalization, and movement. The best response for each category is recorded. A minimum score of 3 indicates a completely unresponsive patient, whereas a score of 15 indicates the patient is awake and alert. Generally, a GCS score of 7 or less indicates a coma.

TABLE 11-5 AVPU Scale
Awake–eyes open spontaneously
Verbal–arouses to stimulation by voice
Painful–purposeful movement to a painful stimulus such as a pectoral pinch
Unresponsive–not responsive to any stimuli

TABLE 11-6 Levels of Arousal
Confusion–loss of the ability to think rapidly and clearly
Disoriented–disorientation to time, followed by place, and then self
Lethargic–limited spontaneous movement or speech; easy arousal with normal speech or touch; may not be oriented to time, place, or person
Obtunded–mild to moderate reduction in arousal with limited responses to the environment; falls asleep unless stimulated verbally or tactilely; answers questions with minimal response
Stuporous–a condition of deep sleep or unresponsiveness from which the person may be aroused or caused to open eyes only by vigorous and repeated stimulation; response is often grabbing at or withdrawal from stimulus
Coma–no verbal response to the external environment or to any stimuli; noxious stimuli such as deep pain yield motor movement ***Light coma***–associated with purposeful movement on stimulation

Coma–associated with nonpurposeful movement only on stimulation

Deep coma–associated with unresponsiveness or no response to any stimulus

Adapted from: McCance K, Huether S. *Pathophysiology–The Biological Basis for Disease in Adults and Children*. St Louis, MO: Mosby; 2002.

TABLE 11-7 Questions Pertaining to Patient Orientation

Category	Sample Questions	Order of Loss
Time	What day is it?	ORDER OF LOSS
	What is the date?	
	What is the month, the year?	
	What is the season?	
	What is the time of day?	
Place	What is the name of the place we are in?	
	What type of vehicle are we in?	
	What is the name of the town or city?	
Person	What is your name?	
	What is your job?	
	Where do you live?	

The evaluation of the patient's awareness is an assessment of the content of consciousness, which is a higher-level function than arousal. In performing this evaluation, the CCTP should assess the patient's orientation to time, place, and person **Table 11-7**.

Assessment requires that the patient provide appropriate answers to a variety of questions. Findings may be documented as alert and oriented (A&O × 3, A&O 2/3, or A&O 1/3). The three aspects of awareness (AAO × 3; **a**wake, **a**lert, and **o**riented) disappear in the order of time, place, and then person (self) as the patient becomes more disoriented. Changes in the patient's answers that indicate increasing degrees of confusion and disorientation may, therefore, be one of the earliest signs of neurologic dysfunction.

Simple observation is the primary tool used to assess the patient's behavior. General behavior is best assessed through observation of gestures, facial expressions, mood, affect, and posture. During this component of the examination, family members or close friends may be able to assist in determining if the responses of the patient are normal or abnormal. A variety of neurologic pathologies—including pain, hypoxia, anxiety, and expanding intracranial lesions—may initially manifest themselves through changes in general behavior.

Most critical patients will not have the ability to undergo lengthy tests to assess cognitive functioning. The **Mini-Mental Examination** **Table 11-8** was developed to provide a simple, easily applied test of higher cognitive functions. It consists of a series of questions that test orientation, registration, attention and calculation, recall, and language. The highest possible score is 30, whereas a score of less than 24 indicates cognitive dysfunction.

Speech Function

Abnormalities of speech may need to be considered early on in the neurologic examination, because they may interfere with the CCTP's ability to accurately assess other, higher-level functions **Table 11-9**. Abnormalities of speech can reflect dysfunction in any component or process of communication.

The term **aphasia** encompasses any loss or impairment of language function as a result of brain damage. Sounds are recognized as language in Wernicke's area, which is connected to the concept area,

where the meaning of the words becomes understood. The concept area is connected to Broca's area, where speech output is generated. Wernicke's area is also directly connected to Broca's area via the arcuate fasciculus. Because the arcuate fasciculus connects the speech comprehension area (Wernicke's area) to the speech production area (Broca's area), damage to this area produces impairment of repetition. These areas are found in the dominant hemisphere of each person's brain (left dominant is most common).

The next sections discuss specific neurologic tests that may or may not be done in the critical care transport environment. With interfacility transports, many of these will already have been done as part of the initial assessment at the hospital. In some cases, the CCTP may perform these if there is not much time for initial assessment or if there is time during transport, but some may not be possible to do in cramped quarters. Which evaluations are performed depend on the patient's condition; if needed, the CCTP will perform certain exams as appropriate. Regardless of whether or not the CCTP actually performs these exams, transport team members must know how to read and interpret these findings and base their care accordingly.

TABLE 11-8 Mini-Mental Examination Components

Parameter	Task	Possible Points*
Orientation	Score 1 point for correct answers to each of the following questions:	
	What is the: time? date? day? month? year?	5
	What is the name of this: hospital? town? country?	5
Registration	Name three objects arbitrarily. Score up to 3 points if, at the first attempt, the patient repeats, in order, the three objects you have randomly named. Score 2 or 1 if this is the number he/she repeats correctly. Encourage the patient, even with prompting, to have all three repeated, so as to test recall later.	3
Attention and calculation	Ask the patient to subtract 7 from 100, and then 7 from the result—repeat this five times, scoring 1 for each time a correct subtraction is performed.	5
Recall	Ask for the three objects repeated in the registration test, scoring one for each correctly recalled.	3
Language	Score 1 point each for two objects (a pencil and a watch) correctly named.	2
	Score 1 point if the following phrase is correctly repeated: "No ifs, ands, or buts."	1
	Have the patient perform a three-stage task. Score 1 for each stage that is successfully performed. For example, the CCTP may state, "with the index finger of your right hand touch the tip of your nose and then your left ear."	3
	On a blank piece of paper, write, "Close your eyes" and ask the patient to obey what is written. Score 1 point if the patient closes his/her eyes.	1
	Ask the patient to write a sentence. Score 1 if the sentence makes sense and contains a subject and a verb.	1
	Ask the patient to construct a pair of intersecting pentagons, each side 1″ long. Score 1 if this is accomplished correctly.	1
TOTAL		30

*Scores of less than 24 indicate cognitive dysfunction. Speech must be intact in order for the test to be valid.
Adapted from: Fuller G. *Neurological Examination Made Easy*. Edinburgh, UK: Churchill Livingstone; 1999.

TABLE 11-9 Abnormalities of Speech

Component or Process	Abnormality
Hearing	Deafness
↓	
Understanding	Aphasia
↓	
Thought and word finding	Aphasia
↓	
Voice production	Dysphonia
↓	
Articulation	Dysarthria

Data from: Fuller G. *Neurological Examination Made Easy*. Edinburgh, UK: Churchill Livingstone; 1999.

Assessment of Cranial Nerves

Assessment of the cranial nerves, which are located between the midbrain and the pons, provides invaluable information on pressure changes within the cranium and its effects on the brain stem in particular. Many of the cranial nerve tests require patient participation, so they may not be feasible in some critically ill or injured patients. Cranial nerve dysfunction may occur as a result of several pathologies, including lesions to the nerve; lesions in the nucleus, the communicating pathways to and from the cortex, the diencephalon, the cerebellum, or other parts of the brain stem; or as generalized problems of nerve or muscle.

Knowledge of all cranial nerves (areas that they serve, expected response to testing, and indications of abnormal response) is necessary to complete a thorough neurologic examination. Nevertheless, certain nerves are more commonly tested in patients who are critically ill or injured: optic (CN II), oculomotor (CN III), trochlear (CN IV), trigeminal (CN V), abducens (CN VI), facial (CN VII), acoustic (CN VIII), glossopharyngeal (CN IX), vagus (CN X), and hypoglossal (CN XII). **Table 11-10** provides an overview of the cranial nerves, their functions, and methods of testing.

Owing to the nature of the environment in which the CCTP works, most patients encountered are critically ill or injured. It may, therefore, be necessary to perform rapid exams that test the function of the most important cranial nerves simultaneously. **Table 11-11** outlines such nerve groups and their exam results.

TABLE 11-10 Cranial Nerves

Cranial Nerve No.	Cranial Nerve Name	Function	Test
I	Olfactory	Sense of smell	Patient smells aromatic substance (coffee or soap but not ammonia or other irritants)
II	Optic	Vision	Visual acuity test–use of Snellen chart Optic disk–fundoscopy Fields–confrontation
III	Oculomotor	Medial and upward/downward movement of the eye Constriction of the pupil Elevation of eyelids Consensual light response Accommodation and convergence	Extraocular movements, pupillary size and equality, and symmetry of response to light shined in eyes
IV	Trochlear	Moves eyes upward and downward	Tested with CN III and VI
V	Trigeminal	Sensation for face, scalp, cornea, and nasal and oral cavities Movement of jaw	Prick the patient's face with a pin and touch the face lightly with a piece of cotton Patient clenches his/her teeth together
VI	Abducens	Moves eyes laterally	Tested with CN III and IV
VII	Facial	Controls facial expression and taste for anterior two thirds of tongue	Direct the patient to smile, whistle, bare the teeth, and pucker lips Also, the patient should close his/her eyes and wrinkle the forehead
VIII	Acoustic	Hearing and equilibrium	Caloric test in unconscious patients Doll's eyes test
IX	Glosso-pharyngeal	Voluntary muscles for swallowing and phonation Secretory and salivary glands Carotid reflex	Test for palatal elevation, swallowing and gag reflexes,* and glottal and palatal sounds Figure 11-21
X	Vagus	Voluntary muscles for phonation and swallowing Involuntary activity of visceral muscles of the heart, lungs, and digestive tract Carotid reflex	Test for palatal elevation, swallowing and gag reflexes,* and glottal and palatal sounds
XI	Spinal accessory	Shrugging of shoulders and turning of head	Ability to shrug shoulders and turn head against resistance
XII	Hypoglossal	Movement of tongue and taste for posterior third of the tongue	Ability to protrude tongue and push tongue against cheek

**Note:* 10% of the normal population lacks a gag reflex.

Olfactory Nerve

Cranial nerve I, which is responsible for olfaction, should be assessed in patients in whom head trauma has occurred, when pathology at the base of the skull is suspected, and in patients who exhibit an altered mental status. Familiar odors should be used to conduct the exam, such as coffee, vanilla, soap, and lemon oil. Irritant substances such as ammonia or vinegar should *never* be used to conduct the examination. The patient should be able to identify the substance presented with the eyes closed and one nostril occluded. Complete or unilateral anosmia (inability to smell) may suggest damage to the olfactory tract.

Another interesting phenomenon is **Foster-Kennedy syndrome**, which involves the olfactory nerve and is caused by a tumor or abscess at the base of the frontal lobe. Signs and symptoms include ipsilateral blindness and anosmia, ipsilateral atrophy of the olfactory and optic nerves, and contralateral papilledema.

Optic Nerve

The optic nerve (CN II) arises from cells in the retina and then passes into the orbit, where it is contained in meningeal sheaths. The nerve then changes its name to the optic tract when the fibers have passed through the optic chiasm. The fibers from the nasal half of the retina decussate within the optic chiasm;

those from the lateral (temporal) half do not. Each eye should be evaluated individually to assess the optic nerve. The tests should include evaluation of visual acuity, visual fields, and the fundi (via funduscopy).

Figure 11-21 The test for cranial nerve IX, the glossopharyngeal nerve, tests for palatal elevation, swallowing and gag reflexes, and glottal and palatal sounds.

TABLE 11-11 Cranial Nerves and Their Exam Results

Cranial Nerve Group	Specific Test	Significance
III, IV, VI	Extraocular movements	Dysconjugate gaze indicates compression or injury of nerve; eyes are unable to turn together in the same direction.
V, VII, IX, X, XII	Swallowing	Potential for aspiration.
III, VI, VIII	Oculocephalic reflex (doll's eyes)	Brain stem dysfunction.
III, VI, VIII	Oculovestibular reflex (caloric test)	Brain stem dysfunction.
IX, X	Gag reflex	Loss of protective reflex as a result of brain stem dysfunction or nerve injury.
V, VII	Corneal reflex	Loss of protective reflex as a result of brain stem compression or nerve injury.

Data from: Kinney MR, Brooks-Brunn JA, Molter N, et al. *AACN Clinical Reference for Critical Care Nursing*. St Louis, MO: Mosby; 1999.

To formally evaluate visual acuity, a Snellen chart **Figure 11-22** should be used. Each eye should be checked individually using the chart. The patient should be asked to read the line on the chart with the smallest letters that he or she is able to read at a distance of 20′. The number beside each line on the chart is the distance at which a person with normal vision can read the letters. This number is the denominator when recording the vision.

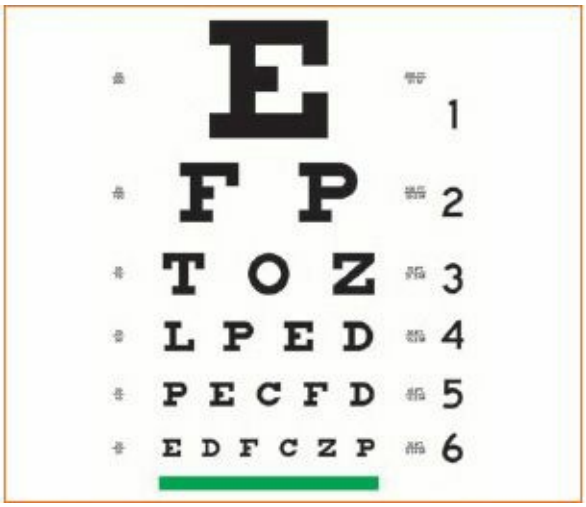


Figure 11-22 The Snellen chart.

Figure 11-23 Rosenbaum Pocket Vision Screener (eye chart).

An alternative to the Snellen chart is the Rosenbaum Pocket Vision Screener **Figure 11-23**, which can be used at a more practical distance of 14″ vs 20′. The ratio is calculated in the same manner as used for the Snellen chart.

Examination of the visual fields provides invaluable information that can often enable the CCTP to determine the location of a lesion on the visual pathway. A normal visual field extends 60° to the nasal side, 100° on the temporal side, and 130° vertically. To evaluate the visual fields, perform the confrontation test. In this test, the CCTP faces the patient at a distance of 2′, and then asks the patient to cover one eye lightly and look at the CCTP's eye directly opposite. The CCTP should then present a stimulus in each of the four quadrants—upper and lower nasal and upper and lower temporal—of the visual field. Acceptable stimuli include finger movement, rapid finger counting, and hand comparison. The CCTP should assess for the presence of a *scotoma*, or spot, which is a deficit of the visual field surrounded by normal vision. The *blind spot* represents the location of the optic disk within the visual field, because the optic disk does not have rods, cones, or ganglion cells necessary for vision.

Figure 11-24 An ophthalmoscope.

The **fundus** of each eye includes the optic disk, macula, and blood vessels on the back wall of the internal eyeball; it should be examined using the ophthalmoscope **Figure 11-24**. During the examination, the patient should look at a distant object, and the CCTP should attempt to observe the optic disk, the blood vessels, and the retinal background. With the ophthalmoscope dial set on zero, the pupillary red reflex (the point at which the retinal reflex is seen glowing pink in the pupil) is located from a distance of about 2′ or 3′. The examiner slowly approaches the patient's eye as if viewing the eye through a keyhole. At the same time, plus or minus lenses, as needed, are dialed on the ophthalmoscope to focus on the patient's retina. The **optic disk** is located by directing the ophthalmoscope toward the nasal side of the patient's retina. The most prominent structure visible, it represents the termination of the optic nerve. **Table 11-12** describes assessment parameters.

Ocular, Trochlear, Trigeminal, and Abducens Nerves

The oculomotor, trochlear, and abducens nerves (CN III, IV, and VI, respectively) are usually examined as a group because they act together in producing eye movement and controlling ocular muscles to ensure that the eyes remain parallel throughout their range of motion. The two parameters that should be closely observed by the CCTP in relation to these nerves are the position of the eyeball and the position of the upper eyelid. The eyeball's position should be observed from a frontal and lateral view as well as from a cephalad view. The upper eyelid should be assessed while having the patient look straight ahead, and the CCTP should look for evidence of drooping or **ptosis**.

To test eye movement, the patient is asked to follow the CCTP's finger in upgaze and downgaze and from side to side; these areas of movement are commonly called the six cardinal fields of gaze. Eye movements can be divided into four types, each controlled by a different part of the brain **Table 11-13**. The brain stem is responsible for integrating inputs from the frontal and occipital lobes and the cerebellum and vestibular nuclei such that both eyes move together in the same direction, called **conjugate movement**. If there is a lack of symmetric movement between the two visual axes, **dysconjugate movement** is said to be present. **Diplopia**, or double vision, may occur if the patient is unable to move the eyeball in one particular direction.

In conducting a neurologic examination on an unconscious patient, the oculocephalic reflex (doll's eye test) and the oculovestibular reflex (caloric test) may be evaluated for reflex movement of the eyeball (see the section on assessment of cranial nerve VIII for this procedure). The presence of these reflexes indicates the brain stem is intact.

During the assessment of cranial nerves III, IV, and VI, assess for the following dysfunctions:

- Gaze abnormalities **Table 11-14**
- Nystagmus—an involuntary rhythmic movement of the eyes that may be horizontal, vertical, or mixed direction with a fast and slow component to the movement **Table 11-15**
- Abnormalities of the pupils in terms of size, shape, equality, and reflexes **Table 11-16**

The oculomotor nerve (CN III) innervates the superior, medial, and inferior rectus muscles, the inferior oblique, and the constrictor of the pupil and the ciliary body, as well as the levator of the eyelid. A complete lesion of the oculomotor nerve results in paralysis of the ipsilateral muscles innervated by the nerve and ptosis, pupillary dilation, and inability to look upward, downward, or inward. Oculomotor nerve palsy is commonly caused by uncal herniation resulting from an expanding intracranial mass.

The trochlear nerve (CN IV) innervates the superior oblique muscle. When the fourth nerve is damaged, the affected ipsilateral eye is higher than the normal opposite eye, and it cannot be turned downward when the eye is rotated inward. The position of the globe is higher relative to the position of the other globe because the superior oblique muscle normally depresses the eyeball. When this muscle is paralyzed, the eyeball will not depress normally relative to the other eye. Thus, it is *higher* than the other eye.

TABLE 11-12 Assessment Parameters of the Eye

Structure	Normal	Abnormal	Interpretation of Abnormal Findings
Optic disk	Round or slightly oval in shape Yellowish-red color Clearly defined margins	Papilledema: swelling of the disk including disappearance of the optic cup (*Note*: you see nothing, and visual acuity is not affected)	Increased intercranial pressure, usually within 24-48 h
		Papillitis: inflammation of the optic nerve (*Note*: you see nothing, and visual acuity is diminished)	Multiple sclerosis, idiopathic
		Optic atrophy: decreased visual acuity and a change in color of the optic disk to light pink, white, or gray	Primary: optic nerve compression, optic nerve ischemia Secondary: following papilledema
Optic cup	Slightly on the nasal side of the center of the optic disk Diameter is normally < 50% of the disk	Cup may appear deep	Chronic (or idiopathic) glaucoma
Blood vessels	Arteries are lightly colored and are two thirds the diameter of veins	Arterial narrowing and vessel irregularity	Hypertensive retinopathy
	Veins are burgundy colored and may pulsate near the disk	Tortuous vessels	Hypertensive retinopathy
Retinal background	Pigmented background: normal in dark-skinned races, if striped it is called *tigroid*	Red lesions	
		Dot hemorrhages: microaneurysms seen adjacent to blood vessels	Diabetic retinopathy
	Pale: clear is normal in fair-skinned people	Blot hemorrhages: bleeds in the deep retinal layer from microaneurysms	Hypertensive retinopathy
		Flame hemorrhages: superficial bleed, shaped by nerve fibers into a fan that point toward the disk	Subarachnoid hemorrhage
		Subhyaloid hemorrhages: irregular superficial hemorrhages usually with a flat top	Diabetes and hypertension
		White/yellow lesions	
		Hard exudates: yellowish, sharply edged lesions that may form a ring around the macula	Diabetes, systemic lupus erythematosus, acquired immunodeficiency syndrome
		Cotton wool spots: white fluffy spots caused by retinal infarcts	Hypertension

TABLE 11-13 Eye Movements

Type of Eye Movement	Definition	Site of Control
Saccadic (command)	Rapid movement from one point of fixation to another	Frontal lobe
Pursuit	Slow eye movement used to maintain fixation on a moving object	Occipital lobe
Vestibular-positional	Eye movements that compensate for movement of the head/neck to maintain fixation	Cerebellar vestibular nuclei
Convergence	Movements that maintain fixation as an object is brought close to the face	Midbrain

TABLE 11-14 Gaze Abnormalities

Type of Gaze	Description	Causes
Normal gaze	Eye movements are smooth and conjugate	N/A
Horizontal gaze	Deviation of both eyes toward the same side	Destructive hemispheric lesion: both eyes deviate *toward* the side of the lesion Irritative hemispheric lesion (eg, hemorrhage): both eyes deviate *away* from the side of irritation, but later the conjugate gaze becomes paralyzed
Vertical gaze	Deviation of both eyes upward	Upper brain stem lesion
Medial-longitudinal fasciculus	Dysconjugate gaze	Lesion between the pons and midbrain
Skewed deviation	One eye looks downward and the other looks upward	Lesion in the pons on the same side as the eye that is directed downward
Roving-eye movements	Spontaneous, slow, random deviation	Seen in comatose patients with intact brain stem oculomotor function
Ocular bobbing	Episodic, intermittent, usually conjugate, downward, brisk eye movement followed by a return to the resting position by a "bobbing action"	Severe destructive lower pontine lesions

TABLE 11-15 Nystagmus Types

Type of Nystagmus	Description	Cause(s)
Retraction nystagmus	Irregular jerks of the eyes backward into the orbit, initiated by an upward gaze	Midbrain tegmental damage
Convergence nystagmus	Slow, spontaneous, drifting, ocular divergence with a final quick, convergent jerk	Midbrain lesion
See-saw nystagmus	A rapid, pendular, dysconjugate see-saw movement accompanied by severe visual field deficits and loss of visual acuity	Lesion around optic chiasma
Downbeat nystagmus	Irregular jerks initiated by a downward gaze	Lower medullary lesion
	A test in which a striped drum is spun in	

Optokinetic nystagmus	front of the eyes, normally evoking nystagmus in the opposite direction of the spin	Present in most patients
Vestibular nystagmus	Mixed nystagmus that can be horizontal, rotational, or both	Vestibular disease
Toxic nystagmus	Nystagmus occurring while the head is in a specific position, caused by certain drugs or alcohol	Induced by treatment with certain drugs, such as phenytoin, barbiturates, and bromides; also caused by alcohol ingestion

The abducens nerve (CN VI) controls the ipsilateral lateral rectus muscle, which makes the eye look outward (laterally). Defects in abduction (from a lesion of the sixth cranial nerve for example) give the patient a *crosseyed* appearance. The normal eye is oriented straight ahead; by comparison, the affected eye is rotated slightly inward (medially) owing to the unopposed action of the medial rectus muscle on that side.

The trigeminal nerve (CN V) has both motor and sensory functions. In sensory testing, its innervation includes the face up to the vertex of the scalp but spares the angle of the mandible. The sensation from the oral and nasal cavities is transmitted through the trigeminal nerve, although these areas are not usually included in the routine neurologic examination.

Pain and temperature should be tested in the three divisions of the fifth cranial nerve: ophthalmic, maxillary, and mandibular. The ophthalmic division innervates the scalp as far back as the vertex of the skull, forehead, cornea, conjunctiva, and skin of the side and tip of the nose. Corneal sensation is tested by gently touching the corneas with a cotton tip or tissue paper while the patient looks in the other direction. This maneuver constitutes the afferent limb of the corneal reflex. The normal response is a rapid, partial, or complete blinking movement of the eyelid elicited by the efferent limb of the corneal reflex via the facial nerve.

The second trigeminal division, the maxillary nerve, conducts stimuli from the skin of the cheek, far lateral aspect of the nose, upper teeth, and jaw. The third division, the mandibular nerve, carries sensory and motor impulses. The sensory distribution is the skin of the lower jaw, pinna of the ear, lower teeth and gums, and the side of the tongue.

The motor fibers supply the muscles of mastication: the temporal, masseter, and pterygoid muscles. The temporal and masseter muscles are examined by having the patient close the jaws together while the CCTP palpates these muscles. The *jaw-jerk reflex* is elicited by a gentle tap on the chin, with resultant closure of the jaw by the masticatory muscles.

TABLE 11-16 Pupillary Exam Results

Size	2–6 mm (average of 3.5 mm)
Shape	Round Patients who have had previous cataract surgery may have the shape of a keyhole
Equality	Diameters are equal Be aware of anisocoria, a nonpathologic unequal pupil found in 15% of the population

Facial Nerve

The facial nerve (CN VII) is a complex nerve with motor, sensory, and parasympathetic fibers. The motor

portion of the nerve innervates the muscles of facial expression and is tested by instructing the patient to wrinkle the forehead, close the eyelids tightly, smile or grimace showing the teeth, and whistle. Two types of facial motor weakness are possible: one with involvement of the upper motor neuron (corticonuclear/corticobulbar pathways), and the other with involvement of the lower motor neurons (*peripheral* seventh nerve palsy). *Central* (upper) motor facial palsy is characterized by the inability to retract the corner of the mouth, whereas forehead function and eyelid closure remain for the most part unaffected. Lesions in the facial nucleus or the nerve proper will cause paralysis of half of the entire face, with inability to wrinkle the forehead or to close the eyelids and lips on the affected side.

The sensory portions of the seventh nerve originate from the taste buds in the anterior two thirds of the tongue and from the posterior wall of the external ear canal. Although this test is rarely performed in the field, taste is examined using sugar, salt, or quinine solutions. The patient is instructed to protrude the tongue; then the test substance is applied with a cotton-tipped applicator on one side of the tongue. The patient must identify the test substance before drawing the tongue back into the mouth. The facial nerve also carries parasympathetic fibers to the maxillary and lacrimal glands.

Vestibulocochlear Nerve

The eighth cranial nerve is made up of two divisions: (1) cochlear, which affects the sense of hearing; and (2) vestibular, which affects the sense of balance.

The vestibular division of the acoustic nerve is assessed using rotational and caloric stimuli to produce changes in the endolymph current in the semicircular canals. Both tests are also used to determine the brain stem function of unconscious patients. Typically, patients with vestibular dysfunction complain of vertigo, nausea and vomiting, and difficulty with balance, especially with movement of the head.

The **caloric test**, or Bárány test, is commonly used to assess vestibular function; it should not be performed on any patient with a ruptured tympanic membrane. This assessment involves raising and lowering of the temperature in the external auditory canal, which induces convection currents in the endolymph of the semicircular canals and stimulates the vestibular nerve endings. With the patient lying down and the head elevated 30° such that the lateral semicircular canal is vertical, cool water (30°C) is instilled into one ear over 40 seconds (usually about 250 mL). If the patient is conscious, he or she should be directed to look forward while the water is being instilled. This step is then repeated in the other ear with warm water (44°C). The direction of nystagmus is then observed. **Table 11-17** identifies the normal and abnormal responses.

TABLE 11-17 Caloric Test Responses

Conscious patient	Cold: nystagmus away from stimulated ear Warm: nystagmus toward stimulated ear Remember the mnemonic COWS (cold-opposite/warm-same)
Unconscious patient	Cold: nystagmus toward the stimulated ear Warm: nystagmus away from stimulated ear

Figure 11-25 Doll's eye test or oculocephalic reflex test.

The **doll's eye test**, or oculocephalic reflex test, is performed on the unconscious patient by rapidly rotating the head from side to side and observing the eye movement **Figure 11-25**. This procedure is contraindicated if neck or cervical spine injury is suspected. If the reflex is intact or doll's eyes are present (indicating brain stem function between CN III, VI, and VIII), the eyes will deviate opposite the movement of the head. The absence of doll's eyes occurs when the eyes move in the same direction of the head as it is turned; this abnormal response indicates severe brain stem dysfunction.

Glossopharyngeal and Vagus Nerves

The glossopharyngeal and vagus nerves (CN IX and X, respectively) are usually tested together owing to their co-innervation of the pharynx. The glossopharyngeal nerve supplies sensory components to the pharynx, tonsils, soft palate, and posterior third of the tongue. It also supplies motor fibers to the muscle that elevates the pharynx. The vagus nerve is responsible for sensation to the pharynx and larynx; motor function of the soft palate, pharynx, and larynx; innervation of the thoracic and abdominal visceral organs; and sensory input from the heart, lungs, and digestive tract.

To test these nerves, direct the patient to open his or her mouth and say, "Ah." The upward movement of the soft palate and any deviation of the uvula should be noted. As part of this assessment, the CCTP may also stimulate the gag reflex (using a tongue depressor or cotton swab), which is also mediated by cranial nerves IX and X. Note that 10% of the population does not have a gag reflex.

Spinal Accessory Nerve

The spinal accessory nerve (CN XI) is tested in two segments. First, the trapezius muscle is palpated, and its strength is evaluated while the patient shrugs his or her shoulders against resistance. Second, the patient turns his or her head to one side and pushes his or her chin against the examiner's hand; the CCTP should then palpate the sternocleidomastoid for tone and strength. This test should not be performed on patients with a suspected injury to the base of the skull or the upper neck.

Hypoglossal Nerve

The hypoglossal nerve (CN XII) innervates the musculature of the tongue. Testing is performed when the CCTP observes the tongue for fasciculations and atrophy, tests for muscle strength by having the patient push the tongue against the inside wall of the cheek, and checks for tongue protrusion. Tongue protrusion is straight when the hypoglossal nerve pair is working together; when only one branch is working, however, the tongue will deviate toward the injured side. Neck trauma, tumor, or brain stem lesions usually cause unilateral weakness. In contrast, bilateral weakness is usually the result of degenerative neurologic diseases.

Assessment of Motor Function

The motor examination includes a consideration of muscle tone and strength. This exam proceeds from the upper limbs, to the neck and truck, and finally to the lower extremities. The symmetry of each side of the body is compared.

The assessment of motor function evaluates how both voluntary and involuntary motor pathways are functioning. The voluntary motor pathway is a descending pathway that originates in the precentral gyrus of the frontal lobe and ends at all levels of the spinal cord (suprasegmental level). The descending neuron synapses with motor neurons of the spinal cord (segmental level), and final motor output is achieved at the muscle level (myoneural junction). Voluntary muscle control involves the orchestrated effort from skeletal structures, muscles, and involuntary motor pathways that control posture, balance, and reflexive activities.

The muscles should be palpated through the normal range of motion. Abnormalities are characterized in the following terms:

- Spasticity or undue resistance of the muscles owing to passive lengthening because of injury to the corticospinal system
- Rigidity or a more constant state of resistance that involves the extrapyramidal motor system
- Flaccidity or a decreased muscle tone (hypotonia)

TABLE 11-18 Muscle Strength Grading Scale

Grade	Description
5	Normal; full strength against resistance
4	Active movement; minimal weakness against resistance
3	Active movement; barely able to overcome gravity and does not overcome active resistance
2	Active movement; does not overcome gravity
1	Slight trace of muscle contraction
0	No muscle contraction

Muscle strength should be assessed as the CCTP puts muscles of the major joints through their normal range of motion. Strength should also be assessed against gravity and finally against resistance. Muscle strength should be rated using a scale of 0 to 5 **Table 11-18**. The CCTP should draw a stick figure and place values at various points along the figure to indicate the assessed motor response.

Assessment of Sensory Function

The purpose of evaluating the sensory system is to determine the patient's ability to perceive various types of sensations with the eyes closed. Sensation arises from specialized receptors in the body that are designed to recognize and transmit stimuli to the CNS (usually the cerebrum or brain stem), where processing takes place and an appropriate response can be initiated. The sensory impulses travel along specialized tracts in the nervous system.

Sensory examination assesses the integrity of both the receptors and the tracts of conduction. It is not useful for all patients, however, as altered levels of consciousness or preexisting conditions may interfere with obtaining a proper examination or may produce abnormalities unrelated to the present illness or injury. The CCTP should be comfortable using a dermatome chart (shown in Chapter 5) to locate sensory

areas and should be especially familiar with certain key dermatomes, such as C4, C7, T2, T10, L5, S1, and S5.

As part of a detailed assessment of sensory function, pain, temperature, and touch should all be examined and documented. These methods of testing will compare symmetric areas on both sides of the body and evaluate distal and proximal areas of the arms and legs for pain, temperature, and touch sensation. The stimuli should be scattered to sample most dermatomes and major peripheral nerves. The patient's eyes should be closed.

Pain is tested by asking the patient to close his or her eyes, applying randomly both sharp (using a disposable pin) and blunt stimuli, and noting the patient's response. Always begin in areas of known altered sensation and move toward areas of normal sensation to find the edges.

TABLE 11-19 Reflex Locations

Reflex	Nerve	Root
Biceps	Musculocutaneous	C5, C6
Brachioradialis	Radial	C5, C6
Triceps	Radial	C7
Finger	Median and ulnar	C8
Knee	Femoral	L3–L4
Ankle	Tibial	S1–S2

Touch is assessed by using a fine wisp of cotton or an alcohol pad. The patient is instructed to close the eyes during the exam, and then his or her skin is stroked with the cotton or alcohol pad symmetrically on alternating sides of the body. This test measures anesthesia or hyperesthesia.

Reflex Testing

Examination of deep tendon reflexes gives information about the integrity of the spinal nerves and may indicate brain stem or spinal cord lesions. A tendon reflex results from the stimulation of a stretch-sensitive afferent nerve from a neuromuscular spindle, which, via a single synapse, stimulates a motor nerve (efferent), leading to a muscle contraction. Tendon reflexes are increased in upper motor neuron lesions and decreased in lower motor neuron lesions **Table 11-19**. The following scale should be used to grade reflexes:

- 0 = absent
- ± = present only with reinforcement
- 1 + = present but depressed
- 2 + = normal
- 3 + = increased
- 4 + = clonus

Pathologic Reflexes

The following list includes the most common pathologic reflexes, all of which indicate pyramidal tract disease. The Babinski reflex (Babinski's sign) is perhaps the most important of the pathologic reflexes.

- **Plantar response**. A designated area is stimulated and the response of the toes is observed. Normally flexion of the toes is observed. An extensor response is an abnormal finding.
- **Babinski's sign**. The lateral aspect of the sole of the foot is rapidly stroked with a key from the heel to the ball of the foot. If a plantar response occurs, it is considered a negative Babinski's sign (normal). An extensor response indicates a positive Babinski's sign (abnormal).
- **Oppenheim's sign**. An extensor response is elicited by stroking the anterior medial tibial muscle.
- **Gordon's sign**. An extensor response is elicited by firmly squeezing the gastrocnemius muscle.
- **Hoffmann's sign**. When the distal phalanx of the index or middle finger is snapped, there is a sudden clawing of the fingers and thumb.

Figure 11-26 Grasp reflex.

Grasp Reflex

The CCTP places his or her fingers on the patient's palm and pulls the hand away, asking the patient to let go of the hand. A normal response is present if the patient is able to let go. An abnormal response consists of the involuntary flexion of the fingers, causing an uncontrollable grasp of the CCTP's hand **Figure 11-26**. Such a positive grasp reflex indicates a frontal lobe lesion.

Superficial Reflexes

Assessment of the cremasteric reflex can be performed in men as the inner aspect of the upper thigh is stroked downward and movement of the testicle in the scrotum is observed. In transport, this test may be done on neonatal or pediatric patients, but would not likely be done on adults during transport. Cremasteric contraction elevates the testicle on the stimulated side. This exam tests the integrity of afferent (femoral nerve L1, L2) and efferent (L1, L2) fibers. Absence of the reflex may indicate a lesion in the reflex arc or a pyramidal lesion above L1, or may be nonpathologic.

Meningeal Irritation and Its Evaluation

Meningeal irritation may result from infections caused by bacteria, viruses, fungi, parasites, and toxins. Infections may be classified as acute, subacute, or chronic. Each type of causative agent results in a different pathophysiology, clinical manifestation, and treatment.

In this condition, the causative agent acts as an irritant, causing an inflammatory reaction around the meninges found in the arachnoid space, the CSF, and the ventricles, and resulting in hyperemia and increased permeability of the meningeal vessels. Eventually, this reaction causes the migration of neutrophils into the subarachnoid space, resulting in the formation of exudates that thicken the CSF and interfere with its normal flow around the brain and spinal cord. These exudates can obstruct the arachnoid villi and produce hydrocephalus. The purulent exudates increase rapidly, causing increased inflammation,

especially around the base of the brain but also extending into the cranial sheaths, spinal nerves, and perivascular spaces of the brain's cortex. The meningeal cells become edematous. Over time, the combination of purulent exudates and cell edema results in increased ICP.

The clinical manifestations of meningeal irritation can be grouped into meningeal, infectious, and neurologic signs. This section focuses on the meningeal signs, which include generalized throbbing headache progressing in severity, progressive photophobia, nuchal rigidity, Kernig's sign, and Brudzinski's sign.

The test for **nuchal rigidity** should not be performed if cervical spine injury is suspected. While the patient is lying flat, the CCTP should place his or her hand behind the patient's head and gently flex the head forward until the chin touches the chest if possible. Marked resistance to head movement in any direction is suggestive of meningeal irritation.

Miscellaneous Tests and Signs

Lhermitte Phenomenon

In **Lhermitte phenomenon**, forward flexion of the neck produces an electric shock feeling, usually running down the back. The presence of this sensation indicates cervical pathology.

Vital Signs

As part of the neurologic examination, the CCTP should obtain vital signs. The centers for control of vital functions are found within the brain stem. Thus, any changes in vital signs, which may be subtle, can provide invaluable information to the CCTP concerning the critical care patient's overall neurologic status. During the examination, both before and during transport, the CCTP should pay particular attention to vital signs pertaining to the respiratory system, the cardiovascular system, and body temperature **Figure 11-27**. **Table 11-20** lists the functions of each control center. The following physiologic variables should be checked repeatedly, as indicated by the patient's condition:

- Blood pressure
- Pulse rate and rhythm
- Respiratory rate
- Pulse oximetry
- End-tidal carbon dioxide (quantitative)
- Central venous pressure
- Temperature
- ICP

Respiratory System

Because much of the control over the respiratory system comes from structures within the brain stem, respiratory patterns often provide the CCTP with valuable information. Abnormal respiratory patterns may signal that neurologic injury/dysfunction is present and allow the CCTP to determine at which anatomic level injury has taken place.

When CCTPs assess respirations, they should pay particular attention to the rate, rhythm, and characteristics of the inspiratory and expiratory phases. A variety of conditions other than neurologic damage may produce alterations in respirations, such as metabolic disturbances and drug overdoses. The breathing patterns that may be observed can be classified as relating to certain types of hemispheric or brain stem activity **Table 11-21**. Chapter 6 provides explanations and illustrations of abnormal respiratory patterns.

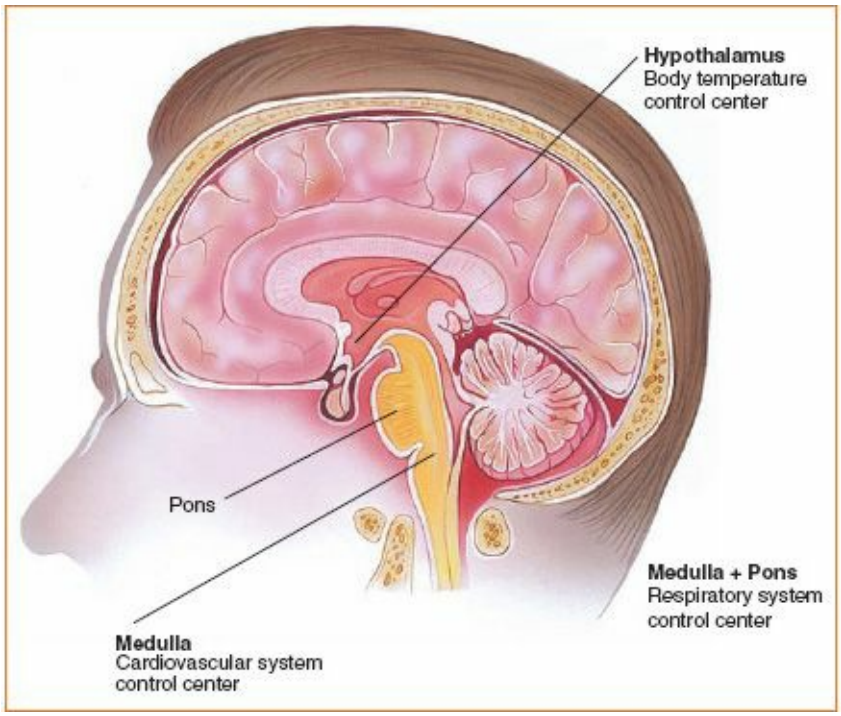


Figure 11-27 The locations of the respiratory system, the cardiovascular system, and body temperature control centers in the brain.

Cardiovascular System

The medulla is the control center for several elements of the cardiovascular system. Inhibitory impulses arising from the medulla travel to the heart via the vagus nerve, where acceleratory impulses travel through the spinal nerves arising from T1 through T5. Given this relationship, the patient's heart rate may indicate not only probable neurologic injury, but also the presence of other pathologic processes. The CCTP should be especially aware of the presence of tachycardia, bradycardia, or arrhythmias. **Table 11-22** gives a nonexhaustive list of probable causes for each variance in pulse rate.

TABLE 11-20 Control Center Location and Function

System	Location	Function
Cardiovascular System		
Cardiac center	Medulla	Regulation of rate and force of contraction
Vasomotor center	Medulla	Regulation of blood pressure
Respiratory System		
Dorsal respiratory group	Medulla	Initiates inspiration; sets the basic rhythm of respiration
Ventral respiratory group	Medulla	Almost inactive except when increased ventilatory effort is needed
Apneustic center	Pons	Exact function is unclear; sends signals to the dorsal respiratory group to prevent apnea (especially in injury)
Pneumotaxic center	Pons	Modifies inspiration rate
Temperature Regulation		

Anterior hypothalamus (response to heat)	Hypothalamus	Cutaneous vasodilation, sweating, increased respiration, anorexia, apathy, and inertia
Posterior hypothalamus (response to cold)	Hypothalamus	Shivering, hunger, increased catecholamine release, cutaneous vasoconstriction, horripilation (bristling of hairs on the skin)

When assessing the heart rate, remember that the presence of multiple illnesses or injuries may mask the characteristic effect on heart rate that would occur with an illness/injury if it were present alone. For example, bradycardia caused by increasing ICP may be counterbalanced by tachycardia in response to hypovolemic shock, thereby hiding the presence of both problems.

Hypertension—an increase in blood pressure—is the effect on blood pressure most often seen as a result of neurologic injury, especially in the case of rising ICP. As cerebral perfusion decreases, ICP rises; in response, the body attempts to maintain adequate perfusion by increasing blood pressure. Hypertension is commonly seen in conjunction with bradycardia and an abnormal respiratory pattern, collectively known as **Cushing's triad**.

TABLE 11-21 Breathing Patterns

Breathing Pattern	Location of Injury
Normal	Response to external stressor—not associated with central nervous system dysfunction
Posthyperventilation apnea	Associated with diffuse bilateral metabolic or structural disease of the cerebrum
Cheyne-Stokes respirations	Bilateral dysfunction of deep cerebrum, diencephalonic structures (thalamus and/or hypothalamus), or basal ganglia
Central neurogenic hyperventilation	Dysfunction of midbrain or upper pons
Apneustic breathing	Respiratory control mechanisms located at the pontine level
Cluster breathing	Dysfunction of lower pontine and upper medullary areas
Ataxic (Biot's) breathing	Dysfunction of medullary control centers
Agonal gasps	Failing medullary control centers

Adapted from: McCance K, Huether S. *Pathophysiology—The Biological Basis for Disease in Adults and Children*. St Louis, MO: Mosby; 2002.

TABLE 11-22 Conduction Variances

Conduction Variance	Probable Causes
Bradycardia	Increasing ICP SCI involving the sympathetic pathways
Tachycardia	Shock

	The end-stage of a neurologic injury
Arrhythmias	Increasing ICP Other intracranial pathologic conditions

Cerebral injury rarely causes hypotension except in the last stages of injury. The CCTP should, however, take all means necessary to ensure that the patient does not become hypotensive (systolic blood pressure, < 90 mm Hg), because even one episode is associated with high mortality owing to its detrimental effects on cerebral perfusion.

Pulse pressure is also a valuable tool in the evaluation of neurologic injury. The CCTP may observe widening (> 40 mm Hg) of the pulse pressure in the presence of increasing ICP.

Body Temperature

Monitor the patient's body temperature closely for changes. Alterations in body temperature—hypothermia or hyperthermia—are possible in the presence of neurologic injury, especially if the hypothalamus is involved. Take special note of hyperthermia because it can cause increased production of harmful metabolic by-products, such as carbon dioxide and lactic acid, that may in turn contribute to secondary injury.

Neurologic Diagnostics

This section discusses neurologic diagnostics of which the CCTP should have background knowledge.

Computed tomography (CT) scanning provides a mathematically reconstructed cross-sectional view of the body, including the brain, and is performed on almost every patient with abnormal neurologic findings. The image that is seen is obtained by passing intersecting x-ray beams through the area of interest and measuring the density of substances through which the x-ray beams pass. As the density of a substance increases, its image appears more white. As the density of a substance decreases, its image appears more black. In a normal CT scan of the head, bone appears white, blood appears off-white, brain tissue appears shaded gray, CSF appears offblack, and air appears black. A CT scan can be performed with or without the use of an IV contrast medium. The benefit of using contrast is for the enhancement of vascular areas and better detection of vascular lesions. Patients receiving contrast should be monitored during infusion of the dye and for 10 to 20 minutes after for evidence of anaphylactic reaction. Patients should also be monitored for evidence of acute tubular necrosis. Noncontrast CT is typically used to view the intracranial area for evidence of intracranial hemorrhage, cerebral edema, or displacement of structures.

Magnetic resonance imaging (MRI) produces images with greater detail than does CT, and has become the standard diagnostic study for many conditions. A non–iodine-containing contrast medium is usually administered IV prior to performing the procedure. The image is created by placing the patient in a large magnetic field, and introducing radiofrequency waves that cause resonance of nuclei. A computer then uses the given resonance to create an image.

Cerebral angiography allows visualization of the lumen of vessels to provide information about patency, size, irregularities, or occlusion. The procedure is typically used in the diagnosis of cerebral aneurysms, arteriovenous malformations, and carotid artery diseases. The procedure requires that a catheter be inserted into the femoral artery, threaded through the aorta and into the origin of cerebral circulation. Once the catheter is in place, a radiopaque contrast medium is injected, allowing visualization of circulation with the use of serial radiologic imaging. Complications associated with this procedure include contrast medium hypersensitivity and renal dysfunction.

Transcranial Doppler (TCD) ultrasound allows monitoring of cerebral blood flow velocity through thinner areas of the skull—temporal bone (transtemporal), the eye (transorbital), and the foramen magnum (transoccipital). Blood flow velocities can be measured in the anterior, middle, or posterior cerebral arteries and the vertebral and basilar arteries depending on the angle of the Doppler probe. This procedure is commonly used in patients following rupture of an intracranial aneurysm to assess for vasospasm, a common problem following rupture of a cerebral vessel. The procedure can also be used to identify intracranial lesions following a stroke and to detect cerebral blood flow changes associated with increased ICP.

Electroencephalography (EEG) involves the recording of electrical impulses generated by the brain to localize abnormal electrical activity. The procedure is commonly used in patients with suspected seizure activity, cerebral infarct, metabolic encephalopathies, altered consciousness, infectious disease, some head injuries, and confirmation of brain death. Waveforms are generated by placing electrodes on the head, which allow electrical impulses to be transferred to a recording unit. Five types of waves are seen most commonly: delta, theta, alpha, beta, and gamma.

Delta bands are normally associated with adults during sleep, and are also normal in infants. Pathologic causes include various types of lesions (including subcortical, diffuse, and deep midline lesions), as well as metabolic encephalopathy and hydrocephalus. Theta bands are normally associated with young children, drowsy older children, and drowsy adults, and are also sometimes seen with meditation. Pathologic causes are similar to those for delta bands (focal subcortical lesions, deep midline disorders, metabolic encephalopathy, and some kinds of hydrocephalus). Alpha bands are normal when relaxing or closing the eyes, but are also associated with coma. Beta bands are normal when a person is active, working, or anxious. They are also an effect of benzodiazepines. Finally, gamma bands are normal and are associated with performing cognitive motor functions.

Intermittent slowing with triphasic wave morphology is associated with metabolic encephalopathy. Continuous generalized slowing in the delta or theta range is associated with anoxic damage. Other abnormal EEG findings that are related to a poor prognosis are the combination of alpha waves that do not change with stimulation and a coma state (alpha coma), occasional generalized bursts of activity with intermittent inactivity (burst suppression), generalized spikes at fixed intervals of 1 to 2 Hz, and no activity (electrocerebral silence).

Lumbar puncture is typically performed by entering the subarachnoid space in the lumbar region of the vertebral column to obtain diagnostic information or to provide a therapeutic intervention. CSF samples can be taken and evaluated for the presence of subarachnoid blood (subarachnoid hemorrhage) or infection, or for laboratory analysis (refer back to Table 11-1 on CSF composition). The most common complication is headache.

Traumatic Brain Injury

Although it is protected by the rigid cranial vault, the brain may be damaged when the skull is impacted by high forces. Current understanding of the pathophysiology of TBI emphasizes the importance, or role, of two types of injury. Primary (direct) injury is caused by traumatic forces that cause physical or functional disruption of brain tissue, whereas secondary (indirect) processes occur after injury and cause brain dysfunction or cellular damage (eg, hypoxia and ischemia). Secondary injuries (ie, physiologic derangements) occur rather frequently in the period following primary injury, but often go unrecorded with standard monitoring equipment. Missing the presence of these injuries correlates with poor outcomes.

Primary Brain Injury

The head is only one part of a collision in an accident; the brain is the other component involved. Primary brain injury can occur as a result of two mechanisms: contact phenomena injuries and acceleration-deceleration injuries.

Contact phenomena injuries occur as the direct result of trauma to the head; they include all local effects such as scalp laceration, skull fracture, hematoma, and intracerebral hemorrhage. For years, clinicians believed that contact phenomena injuries were the major injuring factors in TBI. Later research proved that the brain itself is often damaged by the abrupt changes in velocity that cause strains, compression, tension, and shearing injuries to cerebral tissue. Sudden changes in velocity (acceleration-deceleration) cause the brain to move across its bony interior, resulting in damage. When the head is subjected to **translational force**, the head's center of gravity moves along a linear path; when it is subjected to **rotational force**, the head moves around its center of gravity. Rotational injuries result when forces cause the brain to twist within the cranial cavity, resulting in stretching or tearing of neurons in the white matter as well as tearing of blood vessels that secure the brain to the skull.

Areas of the brain associated with rough portions of the skull (frontal and temporal lobes) have higher incidences of injury compared with areas with smoother surfaces, such as the occipital lobe. The term **contrecoup injury** is used to describe a situation in which an impact occurs on one side of the head, causing the brain to move within the cranial vault and forcibly contact the opposite side of the skull, resulting in damage on that side of the brain. This phenomenon is also referred to as transitional injury, because forces can be transferred to the opposite side of the brain **Figure 11-28**.

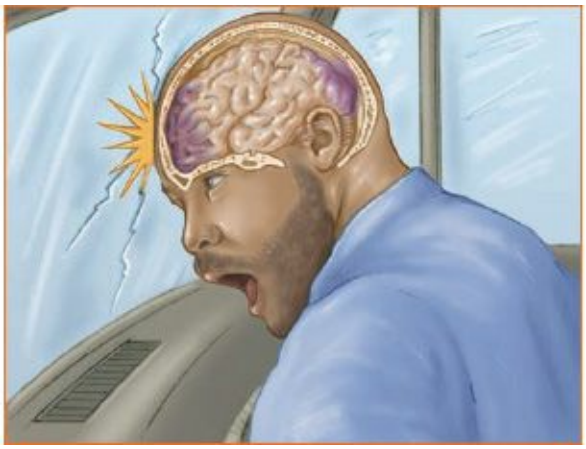


Figure 11-28 Contrecoup injury.

Most current literature cites work by Ommaya and his centripetal theory as the best model to explain the lesions observed after head injury. Ommaya's research simulated acceleration-deceleration injuries and found that certain types of brain injuries can be reproduced without any contact force acting on the head. Additionally, his research showed that the higher the magnitude of the injuring forces, the deeper the functional and structural damage of brain structures. As forces increase, the brain stem eventually becomes involved. Even without direct involvement of the brain stem, interruption of afferent nerve impulses may be sufficient to produce prolonged loss of consciousness.

Secondary Brain Injury

Secondary injury involves the more delayed mechanisms of brain damage. These physiologic derangements, if left uncorrected, will adversely affect brain function integrity. Secondary injuries may be classified as insults of either systemic or intracranial origin **Table 11-23**.

The pathophysiology of secondary brain injury is well understood. After brain injury, there is a failure of the normal protective mechanisms by which blood flow is maintained in the face of falling

perfusion pressure, or by which blood flow is increased during hypoxemia. As a consequence, ischemic or hypoxic damage is more likely to occur **Figure 11-29**. Mechanically injured neurons are more susceptible to the effects of hypoxemia and cerebral hypoperfusion and are vulnerable to damage by the high concentrations of neurotransmitters (eg, glutamate and aspartate) and toxic metabolites that accumulate in the extracellular environment in the wake of TBI.

TABLE 11-23 Causes of Secondary Insult to the Brain

Systemic Origin	Intracranial Origin
Hypoxia	Hematoma
Hypercarbia	Swelling, vasospasm
Arterial hypotension	Raised ICP
Severe hypocarbia	Infection
Fever	Epilepsy
Hyponatremia	Hydrocephalus
Anemia	

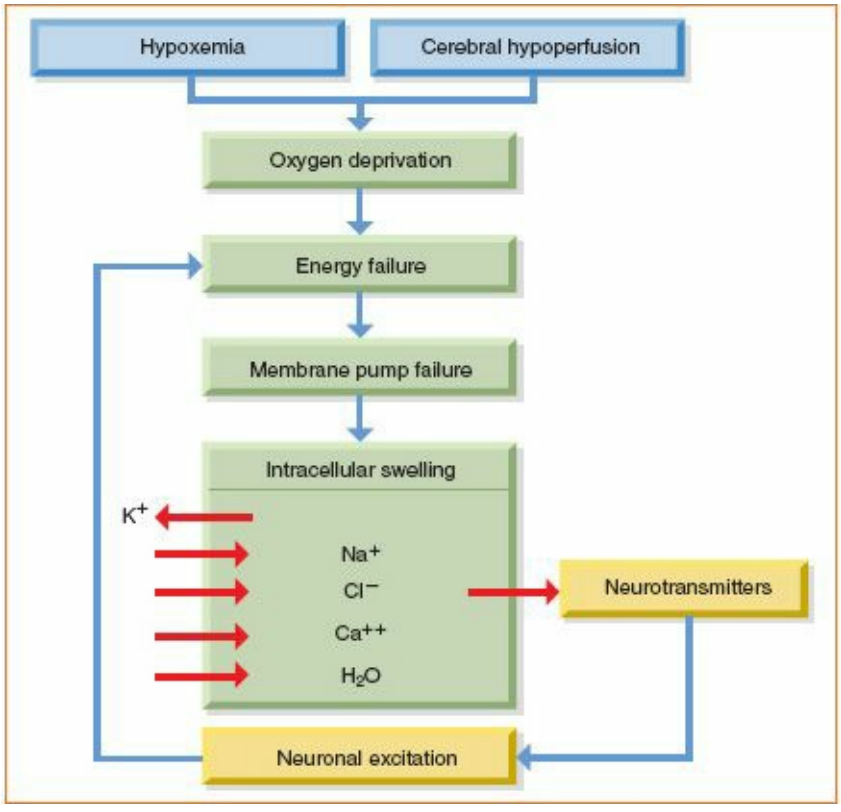


Figure 11-29 Pathophysiology of secondary brain injury. *Adapted from* McCance K, Huether S. *Pathophysiology—The Biological Basis for Disease in Adults and Children*. St Louis, MO: Mosby; 2002.

Neuronal swelling, edema, and cerebral hyperemia from carbon dioxide retention combine to raise the ICP and lower the **cerebral perfusion pressure (CPP)**. This relationship can be expressed mathematically as **mean arterial pressure (MAP)** minus ICP equals CPP:

$$MAP - ICP = CPP$$

where:

$$MAP = DBP + \frac{1}{3}(SBP - DBP)$$

Let's say a patient has a blood pressure of 190/100 mm Hg. The calculation of MAP would be:

$$\begin{aligned} MAP &= 100 + \tfrac{1}{3}(190 - 100) \\ &= 100 + \tfrac{1}{3}(90) \\ &= 100 + 30 \\ &= 130 \end{aligned}$$

CPP is the primary determinant of blood flow to injured brain tissue when the injury has impaired the ability of the cerebral circulation to autoregulate in response to fluctuations in systemic blood pressure **Figure 11-30**. The situation is exacerbated if the ICP is further raised by the presence of a space-occupying lesion or by swelling, or if the CPP is further reduced by extracranial blood loss. The resulting positive feedback loop rapidly causes irreversible clinical deterioration if cerebral perfusion and oxygenation are not quickly improved. Current guidelines identify the ideal CPP to be somewhere between 50 and 70 mm Hg.

Specific Injuries

Scalp Injuries

Injury to the scalp is oftentimes the first indication that a patient has a brain injury. The forces involved in the primary injury determine the type and extent of scalp injury that may be visible. Common injuries to the scalp include abrasions, contusions, lacerations, and avulsions. Like facial injuries, scalp injuries tend to bleed profusely owing to their highly vascular nature.

Abrasions involve the top layer of the scalp, which may be rubbed away, as evidenced by minor bleeding. Treatment usually focuses on controlling bleeding and cleaning and dressing the area. It is important to remember that some of these scalp injuries can occur simultaneously secondary to the mechanism of injury.

When examining an abrasion, the CCTP may also note an underlying contusion. Contusions are scalp bruises with possible accumulation of blood in the subcutaneous layer. Definitive control of bleeding is often best achieved by stapling or suturing significant scalp lacerations. It is also important to rule out more significant (deeper) injuries.

Lacerations can be more serious. In these injuries, the scalp is violently torn, leaving a jagged opening that may extend through multiple layers of tissue and even down to the skull itself. Bleeding may be profuse, and deeper injury to the brain tissue or skull is very likely.

Avulsions occur as a result of forceful, partial, or complete tearing away or separation of the scalp tissue, which appears as a flap. The primary concerns with this type of injury are the underlying integrity of the skull and infection as a result of contaminants. Treatment should focus on evaluation of the skull for fractures and a thorough cleaning of the tissue under the avulsion.

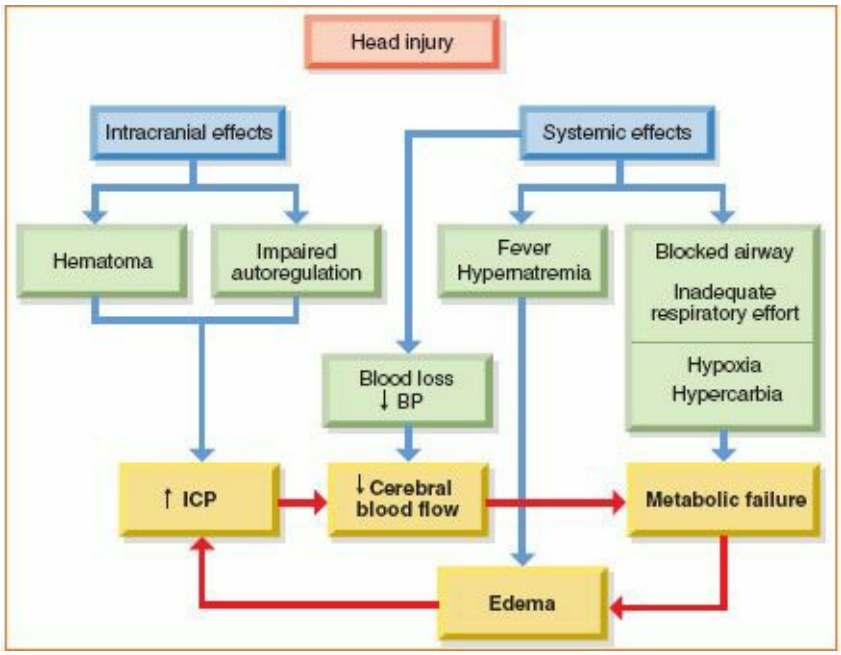


Figure 11-30 Cerebral perfusion pressure as the primary determinant to blood flow to injured brain tissue. *Adapted from* McCance K, Huether S. *Pathophysiology—The Biological Basis for Disease in Adults and Children*. St Louis, MO: Mosby; 2002.

Skull Fractures

Skull fractures occur as a result of a direct, and often highforce, blow to the cranium; they are present in more than 50% of patients with a severe TBI. Any of the bones that make up the skull—the frontal, parietal, temporal, occipital, sphenoid, and ethmoid bones—may be fractured. Between the skull and dura, there are large blood vessels that can be damaged and, if the impact is severe enough, can result in hemorrhaging within the brain.

Although not a part of the cranium, the bones of the face—the maxillary, zygomatic, nasal, lacrimal, and palatine bones; vomer; mandible; and part of the ethmoid and sphenoid bones—may also be fractured. Such fractures may indicate possible underlying brain injury.

Skull fractures are classified into linear, depressed, comminuted, and basilar types.

Linear skull fractures are characterized by a single fracture line and are often an incidental finding on CT. This fracture occurs when two or more bones of the skull separate at the suture line. Hematomas, soft-tissue swelling, and point tenderness are usually present over the site of impact **Figure 11-31A**. A simple closed linear fracture usually does not require any surgical intervention. It is also important to understand stellate fractures. A **linear stellate fracture** is a fracture with multiple linear fractures radiating from the site of impact. These fractures have the potential to do damage depending on the number and depth of the fracture(s). **Diastatic stellate fractures** are described as separation of an epiphysis relating to a center site of impact. Diastatic stellate fractures are prevalent in abused children. The presence of a diastatic fracture to a long bone in a child may arouse suspicion of possible head trauma or shaken baby syndrome.

When a portion of the skull is depressed, and the scalp and/or dura may or may not be torn, the injury is referred to as a **depressed skull fracture** **Figure 11-31B**. This type of fracture may have soft-tissue swelling over the site of the trauma and a bony step-off may be palpated. Depressed skull fractures may be classified as open or closed, with these terms indicating whether there has or has not been a breach in the dura mater in conjunction with the fracture, respectively. An open depressed skull fracture **Figure 11-31C** is the more serious of the two.

Three types of depressed skull fractures are distinguished: true, flat, and ping-pong ball fracture. The

true fracture is the most common, having contact with the cranial vault. The **flat fracture** features a depressed segment without any connection with the cranial vault and is the least common type. The **ping-pong ball fracture** is a pediatric greenstick fracture of the skull. Although these distinctions may be of importance in the hospital setting, during the prehospital phase of the transport, it is most important to identify the existence of a depressed skull fracture, to classify it as open or closed, and to treat it appropriately.

A fracture that occurs when the skull is splintered or shattered into many pieces is referred to as a **comminuted fracture**. These pieces may then act as projectiles and cause direct injury to the meningeal layers or the brain tissue itself. A great deal of blunt force is required to cause this type of skull fracture.

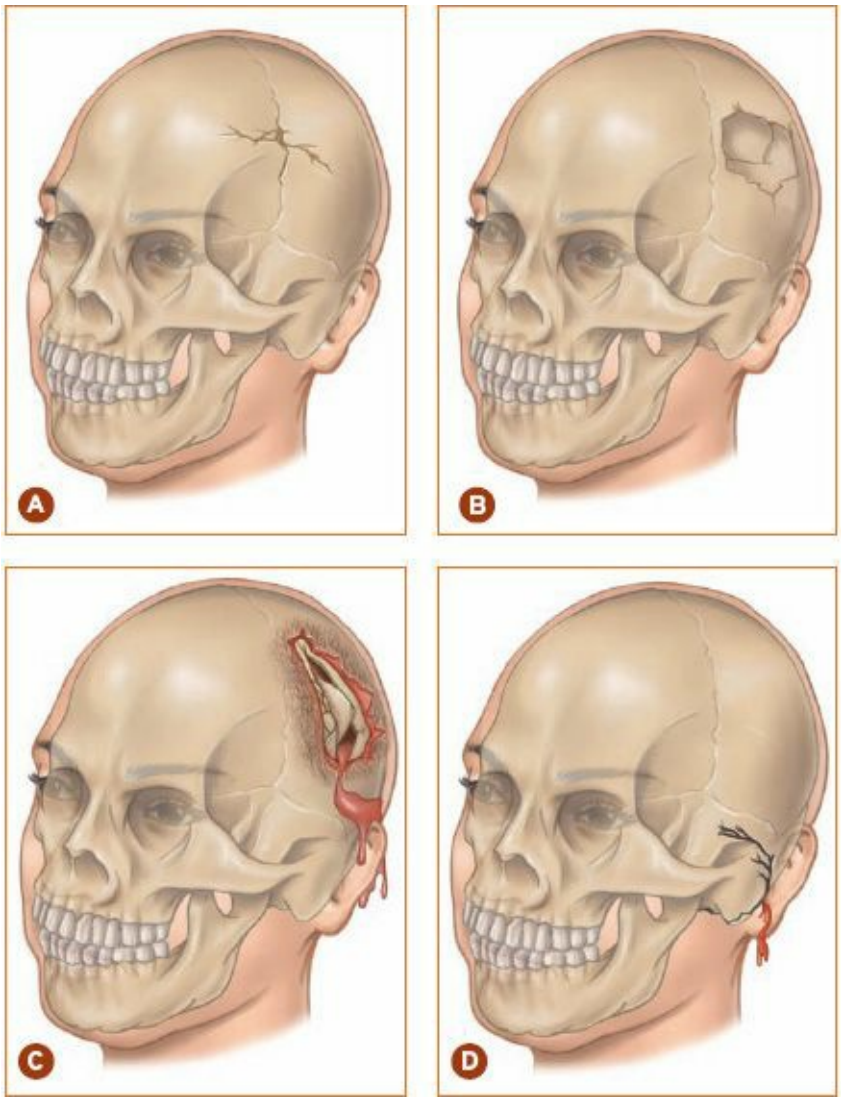


Figure 11-31 Types of skull fracture. **A.** Linear. **B.** Depressed. **C.** Open. **D.** Basilar.

Basilar skull fractures involve fractures along the base of the skull **Figure 11-31D**. This area is permeated with multiple foramina that accommodate the spinal cord, nerves, and blood vessels. Because of the many hollow or open spaces within the skull (eg, the sinuses, eye orbits, nasal cavities, external auditory canals, and middle and inner ears), the skull is weakened here and prone to basal fractures. Basilar fractures usually result from a blow to the parietal, temporal, or occipital regions of the skull, of which the temporal area is the most commonly affected.

Tearing of the dura mater during a basilar skull fracture may also occur, resulting in a wound between the brain and the external environment. This condition may present with leakage of CSF, which may be combined with blood through the dural tear either into the nasal cavity or via the external auditory

canal **Figure 11-32**. Openings in the dura mater also provide a conduit for infection to enter the meninges or brain tissue. The **halo test** for leaking CSF is accomplished by collecting escaping fluid from the nose, mouth, or ears, onto a gauze pad. The appearance of a "target" or "halo" symbol consisting of a dark red circle surrounded by a lighter yellowish one is considered a positive halo sign **Figure 11-33**. The CCTP must exercise caution when using the halo test as the sole diagnostic evaluation of a CSF leak, because other fluids such as lacrimal fluid, nasal fluid, or saliva may cause a similar response.

Other clinical signs may be evident based on the location of the injury. Fractures involving the auditory canal and lower lateral areas of the skull may result in the migration of blood to the mastoid region, posterior and slightly inferior to the ear, resulting in discoloration referred to as retroauricular ecchymosis or **Battle's sign** **Figure 11-34**. Likewise, orbital fractures and hemorrhaging into the surrounding tissue is known as periorbital ecchymosis or **raccoon eyes** **Figure 11-35**. The development of raccoon eyes and Battle's sign may take several hours to develop post-injury and is rarely seen by the CCTP during transport and/or initial treatment of the patient.

Facial Fractures

As many as 60% of patients with significant facial injuries sustain trauma to other organ systems, including the brain. Unfortunately, many facial injuries have very grotesque presentations accompanied by copious bleeding and may potentially distract the CCTP from other, more serious injuries. Fractures of the facial bones are usually very painful and require massive amounts of analgesia to maintain patient comfort. This is especially true with mandible fractures, which can cause life-threatening airway compromise if the patient is in the supine position. Because of the instability of the mandible, the tongue no longer has support and can become lodged in the back of the airway, causing an obstruction.

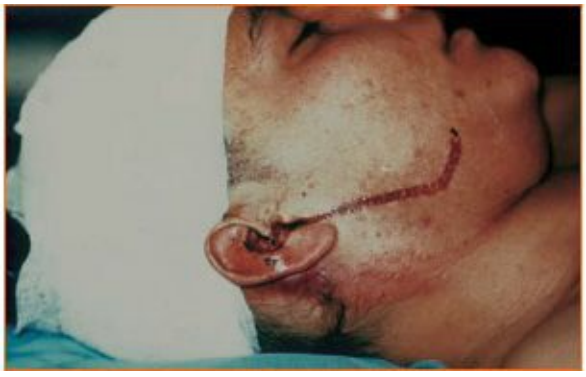


Figure 11-32 Blood leaking from the external auditory canal after a head injury may contain cerebrospinal fluid and suggests a basilar skull fracture.

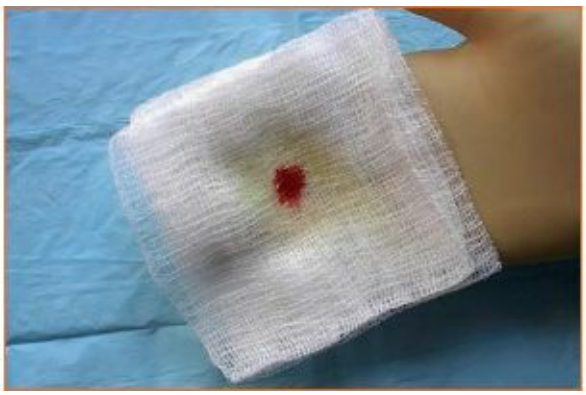


Figure 11-33 Halo sign.

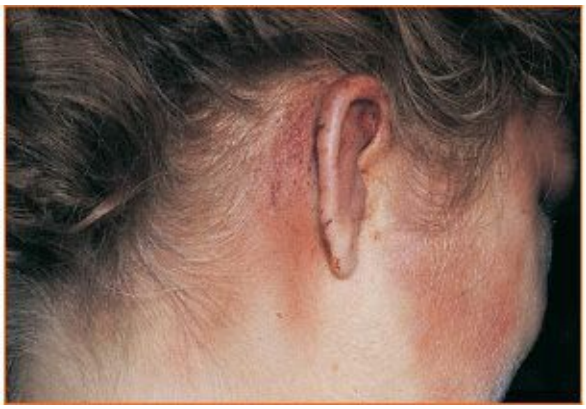


Figure 11-34 Battle's sign or ecchymosis behind the ear or over the mastoid process.

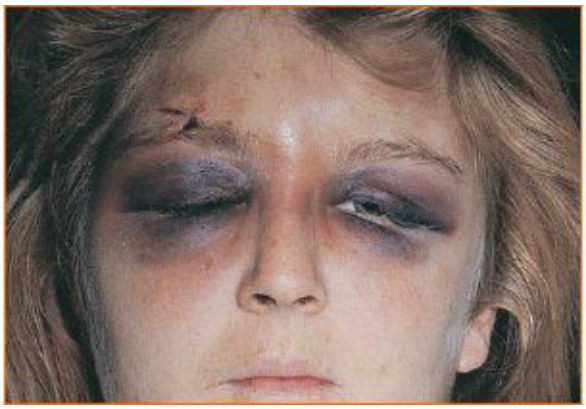


Figure 11-35 Raccoon eyes or ecchymosis under or around the eyes.

Airway control is the first assessment priority with facial injuries. After the airway has been secured, the CCTP can assess for the presence of facial fractures by grasping the hard palate and rocking it back and forth. The CCTP should also check for malocclusion and dental trauma, test extraocular muscles to rule out orbital fractures, and look for facial elongation (monkey face) and periorbital ecchymosis.

Some of the most serious facial injuries are maxillary fractures, which are classified using the **Le Fort criteria**, and result from high-energy forces to the face **Figure 11-36**.

- **Le Fort I** is a transverse fracture separating the hard palate from its bony frame. This fracture exhibits slight instability to the maxilla with no associated displacement.
- **Le Fort II** is a fracture involving the central maxilla and palate, and is a pyramidal fracture (the fractured portion is shaped like a pyramid). This fracture extends through both maxilla and the nasal bones.
- **Le Fort III** is effectively a craniofacial disruption, with fractures extending from the frontozygomatic sutures, orbits, and nasoethmoidal regions. This fracture is unstable and is associated with oral and nasal patency problems. It can leave the patient with a monkey face appearance as a result of the protrusion of the face secondary to its instability.

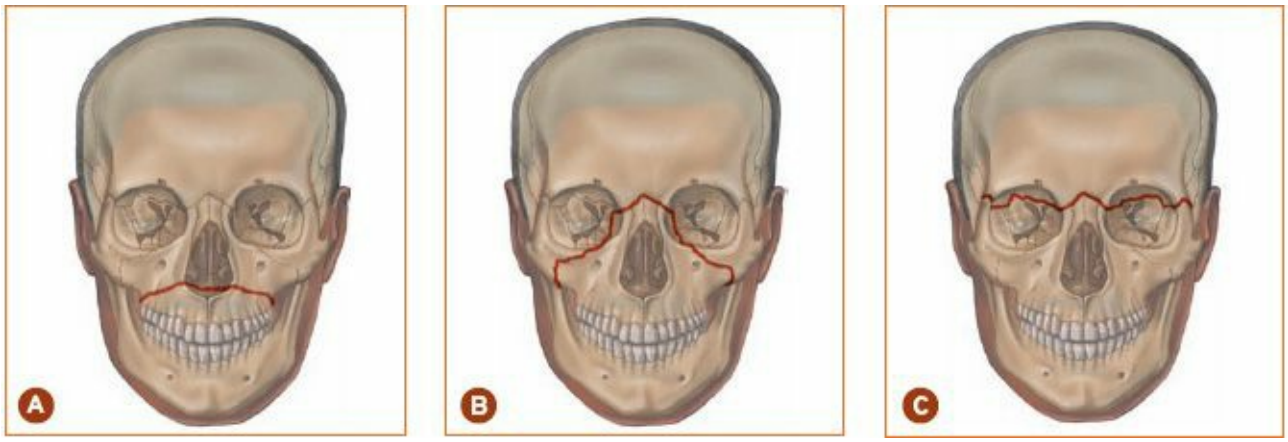


Figure 11-36 Le Fort fractures. **A.** Le Fort I. **B.** Le Fort II. **C.** Le Fort III.

- **Le Fort IV** is essentially a Le Fort III fracture with a concurrent frontal bone fracture.

Le Fort II and III fractures usually result in leakage of the CSF.

Brain Injuries

Injuries to the brain can be either focal or diffuse. Focal lesions are those large enough to be observed directly. Diffuse injuries are not associated with gross localized and visible lesions and include concussions and diffuse axonal injuries **Table 11-24**.

Focal Injuries

A cerebral contusion is a bruising of the brain tissue. The patient with isolated cerebral contusions may exhibit altered mental status, but rarely coma. Contusions commonly occur with rapid deceleration injuries and typically appear on the tips of the frontal and temporal lobes, where the skull base has an irregular shape. Contusions appearing on the same side of the impact are known as coup injuries, whereas injuries on the side opposite the impact are known as contrecoup injuries. Treatment for patients with cerebral contusions is largely supportive because no surgical intervention is usually needed. The patient should be monitored for evidence of rising ICP as a result of hematoma or edema formation; if either of these conditions occurs, it would be treated aggressively.

Lacerations that result in penetration of the cranium may cause tearing of the cortical surface of the brain. A major difference between a contusion and a laceration is that the pia mater–arachnoid mater is torn over a laceration and intact over a contusion. Treatment for cerebral lacerations is identical to that for cerebral contusions, with the CCTP monitoring for signs of rising ICP.

TABLE 11-24 Brain Injury Types

Focal	Diffuse
Contusion Hematoma Epidural Subdural	Axonal injury
Intracerebral	Hypoxia/ischemia
Swelling	Diffuse vascular
Infarct	Fat embolism

Pressure necrosis	Subarachnoid
Abscess	Meningitis

An injury resulting in the accumulation of blood between the inner periosteum and the dura mater is known as an **epidural hematoma (EDH**; also called extradural hematoma). Most EDHs are arterial in origin, occurring as a result of a blow to the temporal region and concomitant disruption of the middle meningeal artery. Blood may accumulate quickly in the potential space as a result of the high arterial pressure gradually stripping away the dura from the inner table of the skull, forming an oval-shaped mass that exerts pressure on the underlying brain tissue **Figure 11-37**. An EDH can be caused by venous bleeding but is limited by the space between the dura and the skull. As the hematoma enlarges, however, the temporal lobe may be displaced and may squeeze over the tentorium.

Special Populations

Indirect and hemorrhagic brain injuries are much more common in the elderly population. This is the result of atrophy of the brain experienced with aging, allowing these patients to tolerate more swelling before herniation occurs and making it easier to tear the bridging veins.

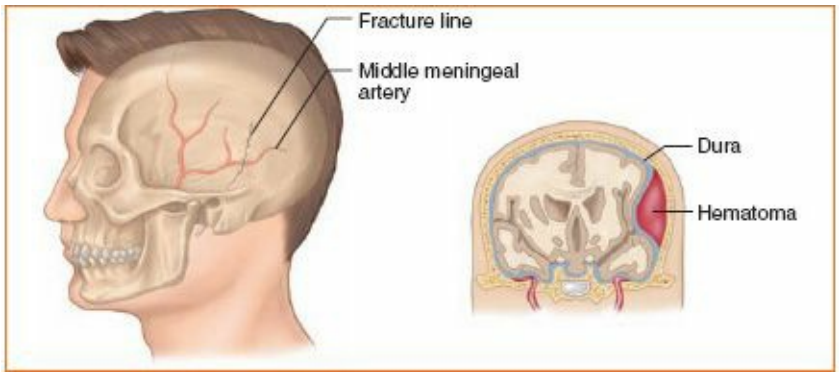


Figure 11-37 An epidural hematoma, resulting from a blow to the temporal region of the head and concomitant disruption of the middle meningeal artery, with the accumulation of blood between the inner periosteum and the dura mater.

The patient with an EDH may present with a history of a brief period of unconsciousness, followed by a lucid period lasting for minutes to several hours. The lucid period ends with a rapid deterioration of consciousness from drowsiness, to lethargy, and to coma as a mass effect and herniation develop. If tentorial herniation (herniation of the tentorium of the cerebellum) occurs, the third cranial nerve may become compressed, causing an ipsilateral fixed and dilated pupil **Figure 11-38**. Tentorial herniation may also occur on the opposite side, causing either contralateral or bilateral blown pupils (Kernochan's notch syndrome). As ICP continues to rise, the patient may develop bradycardia, Cheyne-Stokes respiratory patterns that progress to brain stem patterns, and brain distortion. Other clinical manifestations may include headache, seizures, hemiparesis or hemiplegia, decorticate or decerebrate posturing **Figure 11-39**, respiratory distress, and death. The most common presentation includes vomiting, seizures, unilateral hyperreflexia with a positive Babinski's sign, and elevated CSF pressure.

Signs and Symptoms

Epidural Hematoma

- Brief period of unconsciousness, followed by a lucid period lasting for minutes to several hours
- Following the lucid period, rapid deterioration of consciousness (drowsiness to lethargy to coma)
- Ipsilateral fixed and dilated pupil
- Contralateral or bilateral blown pupils (Kernochan's notch syndrome)
- Headache
- Bradycardia
- Cheyne-Stokes respiratory pattern, progressing to brain stem breathing patterns
- Possible seizures
- Hemiparesis
- Hemiplegia
- Decorticate or decerebrate posturing
- Respiratory distress
- Death
- Vomiting
- Seizures
- Unilateral hyperreflexia with a positive Babinski's sign
- Elevated CSF pressure
- Elevated ICP with brain distortion

Transport Management

Epidural Hematoma

- Provide rapid transport.
- Consider administering an osmotic diuretic (mannitol).

Figure 11-38 Ipsilateral fixed and dilated pupil.

Large hematomas produce a mass effect (ie, act as a space-occupying lesion) and increased ICP. If herniation occurs or will likely occur, the hematoma will be evacuated with a craniotomy. The torn vessel will also be repaired during the craniotomy.

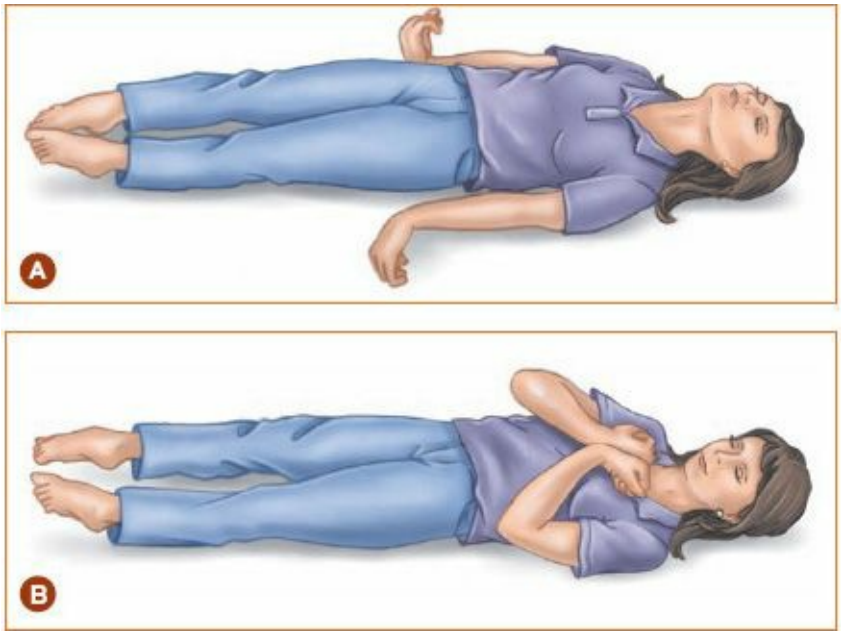


Figure 11-39 Posturing indicates significant intracranial pressure. **A.** Decerebrate (extensor) posturing. **B.** Decorticate (flexor) posturing. (Think of the arms being pulled to the "core" of the body.)

The bleeding can sometimes be so severe that, in some cases, the brain is actually displaced (midline shift) within the skull. Some medications, such as osmotic diuretics (mannitol), can be given in the early stages of midline shift, which will slow the progression until surgical intervention can be obtained.

Mannitol is an osmotic diuretic that is used to reduce increased ICP resulting from an increase in CSF. It is also used to reduce intraocular pressure. In the case of brain hemorrhage, increases in CSF result from inflammation to brain tissue from blood or damage to the subarachnoid villa, which impedes reabsorption of CSF. Mannitol is also useful for patients with cerebral edema not related to a brain bleed.

Mannitol has little effect in reducing the volume of blood accumulated by the hemorrhage. The dosage for adults and children older than 12 years is 1.5 to 2 g/kg in a 15%, 20%, or 25% IV solution given over 30 to 60 minutes via an infusion pump.

Brain stem herniation is a primary concern and occurs when the expanding blood pushes the brain down the foramen magnum, causing a shearing-type force on the brain. Once this takes place, death is imminent, because this condition is irreversible.

High-energy impacts may also result in bleeding that accumulates between the dura and arachnoid mater, known as a **subdural hematoma (SDH)**. Subdural hematomas are six times more common than EDHs and have a higher mortality rate. Most SDHs result from a disruption of the bridging veins located over the crest of the brain and their subsequent bleeding. SDHs are categorized based on the time that elapses between injury and the appearance of signs and symptoms as well as the appearance of blood and fluid composition **Figure 11-40**.

Acute SDH—that is, SDH in which signs and symptoms appear within 24 hours of the injury—is commonly seen with acceleration-deceleration injuries. With this type of injury, clotted blood may be seen on CT as crescent-shaped collections of blood with high attenuation usually along the cranium. Patients usually present with a gradual or sometimes rapid deterioration in level of consciousness, pupillary changes, and hemiparesis or hemiplegia.

In subacute SDH, signs and symptoms appear between 2 days and 2 weeks after injury because the blood accumulates more slowly. This type of hematoma consists of a mixture of clotted and fluid blood; on CT, it may appear as areas that are isodense, hypodense, or of mixed density depending on the age of the lesion **Figure 11-41**. Patients with subacute SDH present with gradual mental status and/or

personality changes as well as many of the symptoms for the acute hematoma that become observable as the lesion expands.

Signs and Symptoms

Subdural Hematoma

- Gradual or rapid deterioration in level of consciousness
- Pupillary changes
- Hemiparesis
- Hemiplegia
- Gradual personality changes
- Headache (progressing in severity)
- Slowed cerebral functioning
- Difficulty speaking
- Confusion
- Drowsiness
- Possible seizures

Transport Management

Subdural Hematoma

- Provide supportive care.

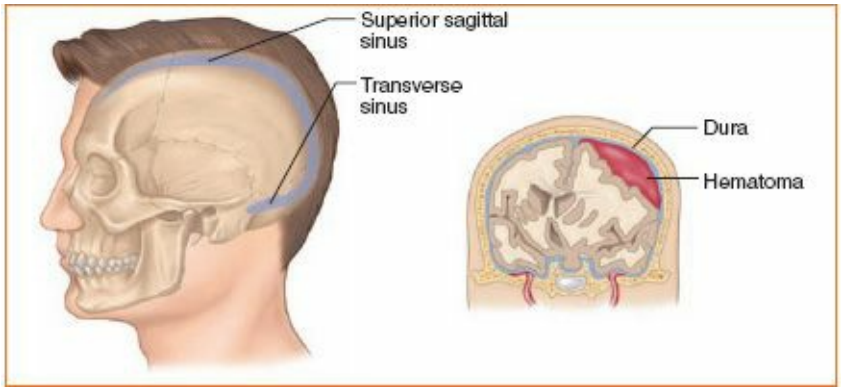


Figure 11-40 A subdural hematoma, in which venous bleeding occurs beneath the dura mater but outside the brain.

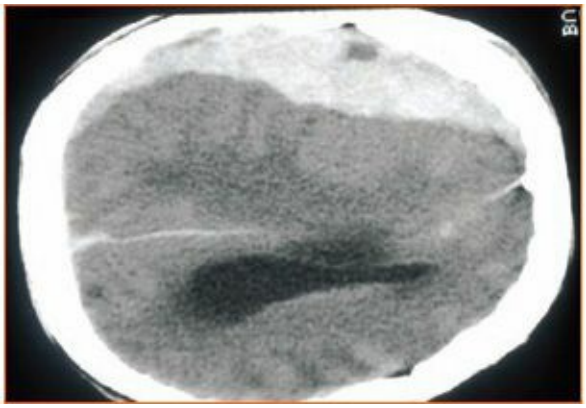


Figure 11-41 Computed tomographic scan of an isodense subdural hematoma.

Chronic SDH manifests as clinical signs and symptoms greater than 2 weeks after the injury. This type of hematoma is composed of fluid only. Because the lesion expands very slowly, the patient may experience headache (progressing in severity), slowed cerebral functioning, confusion, drowsiness, and possibly seizures.

Treatment of SDHs depends on the size of the hematoma and the patient's symptoms, and ranges from watchful waiting to surgical intervention. Small SDHs may be observed and allowed to reabsorb without surgery. All SDHs accompanied by signs and symptoms of increasing ICP require surgical intervention to evacuate the expanding mass and stop the bleeding.

Intracerebral hemorrhages result from direct bleeding into the brain parenchyma and can be observed in as many as 15% of patients with brain injuries **Figure 11-42**. Many of these hemorrhages occur in the same areas as cerebral contusions, making it appear that the rough internal areas of the cranium are oftentimes to blame for these injuries. Intracerebral hemorrhages act as space-occupying lesions, producing headaches, deteriorating levels of consciousness, hemiplegia on the contralateral side, and a dilated ipsilateral pupil.

Patients may also develop a **delayed traumatic intracranial hemorrhage (DTICH)** within the first 3 to 10 days following an injury to the occipital-parietal region via a coup-contrecoup mechanism. Serial CT scans should be performed within the first 72 hours after injury if DTICH is suspected, as part of the effort to decrease the mortality and morbidity that result from such bleeding.

The presentation of a patient with a **subarachnoid hemorrhage (SAH)** depends on the size of the hemorrhage and the coexisting injuries. With this type of injury, bleeding occurs between the arachnoid mater and the dura mater of the brain, resulting in the patient reporting the "worst headache in my life." Patients with SAH are also more likely to have hypoxia, hypotension, and higher ICP, all of which contribute to secondary brain injury. Signs and symptoms include altered mental status, coma, paralysis, slurred speech, mood changes, and seizures, and appear to be due to increased ICP and meningeal irritation.

Signs and Symptoms

Subarachnoid Hemorrhage

- Worst headache of patient's life
- Hypoxia
- Hypotension
- Increased ICP

- Altered mental status
- Coma
- Paralysis
- Slurred speech
- Mood changes
- Seizures

Transport Management

Subarachnoid Hemorrhage

- Provide supportive care, including intubation and mechanical ventilation for patients with an altered level of consciousness.
- Carefully monitor blood pressure, respiratory rate, and level of consciousness.
- Provide appropriate analgesia.
- Consider administering benzodiazepines to reduce stress.
- Insert an indwelling catheter to monitor fluid balance.
- Give an antiemetic to all awake patients.
- Consider administering calcium channel blockers for vasospasm.

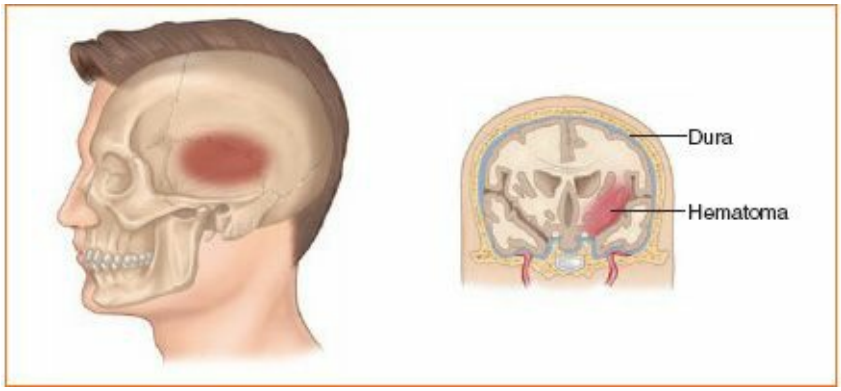


Figure 11-42 An intracerebral hemorrhage is a result of direct bleeding into the brain parenchyma.

Diffuse Injuries

Concussion, also known as **mild TBI**, is a traumatically induced physiologic disruption of brain function that occurs without structural damage. This injury is manifested by at least one of the following signs and symptoms:

- Any loss of consciousness
- Any loss of memory for events immediately before (retrograde) or events immediately after (anterograde) the event that precipitated the injury
- Any alteration in mental status at the time of the injury (eg, feeling dazed, disoriented, irritable, drowsy, giddy, or confused)
- Focal neurologic deficits in which the patient is not unconscious for longer than 30 minutes; after 30 minutes, the patient has a GCS score of 13 to 15

Deep areas of the brain may also be involved in a TBI; such injuries are referred to as **diffuse axonal injury (DAI)**. The mechanism for DAI is usually very rapid acceleration-deceleration forces. In these injuries, shearing forces damage the integrity of the axon at the node of Ranvier, which consequently alters the axoplasmic flow (the flow in the cytoplasm of the axon). Areas especially susceptible to this type of injury include the corpus callosum, the brain stem, and sometimes the cerebellum, because they contain a significant amount of white matter.

DAI accounts for about 50% of all primary brain injuries and 35% of TBI-related deaths. It is characterized by microscopic findings of wide and asymmetric axonal swelling that appear within hours of the injury and may be persistent for much longer, as well as focal hemorrhagic lesions. The resulting injury is graded based on pathologic findings:

- **DAI Grade 1:** histological evidence of axonal damage in the white matter of the cerebral hemispheres, corpus callosum, brain stem, and, less commonly, cerebellum.
- **DAI Grade 2:** in addition to evidence of axonal injury, lesions in the corpus callosum.
- **DAI Grade 3:** in addition to the findings seen in grades 1 and 2, lesions in the dorsolateral quadrant of the rostral brain stem.

The clinical presentation of DAI includes a sequence of events beginning with immediate unconsciousness, leading to a longer period of confusion and associated posttraumatic amnesia, and followed by a protracted recovery time. DAI can be clinically classified as mild, moderate, or severe based on the patient's signs and symptoms.

- **Mild DAI:** coma lasts from 6 to 24 hours, with the patient beginning to obey commands by 24 hours. Death is rare with mild DAI, but cognitive and neurologic deficits are common.
- **Moderate DAI:** coma lasts longer than 24 hours, but prominent brain stem signs are absent. Patients who survive usually have an incomplete recovery.
- **Severe DAI:** coma is protracted and associated with prominent brain stem signs (eg, decortication, decerebration, and an abnormal respiratory pattern). Patients usually die or experience severe disability.

Treatment for all types of DAI is supportive.

Signs and Symptoms

Diffuse Axonal Injury

- Sequence of events
  - Immediate unconsciousness
  - Longer period of confusion and post-traumatic amnesia
  - Protracted recovery time
- Mild DAI
  - Coma lasts from 6 to 24 hours with the patient beginning to obey commands by 24 hours
- Moderate DAI
  - Coma lasts longer than 24 hours, but prominent brain stem signs are absent
- Severe DAI
  - Coma is protracted and associated with prominent brain stem signs (decortication, decerebration, and abnormal respiratory pattern)

Transport Management

Diffuse Axonal Injury

- Provide supportive care.

Intracranial Pressure

Physiology

In adults, the normal ICP is 0 to 15 mm Hg. ICP is not a static state, but rather may be influenced by multiple factors. The recording of ICP shows two forms of pressure fluctuations: a rise with cardiac systole owing to distention of the intracranial arteriolar tree that follows and a slower change in pressure with respiration, with ICP falling with each inspiration and rising with each expiration. Straining and compression of the neck veins can also cause a sudden, considerable rise in pressure.

The **Monro-Kellie doctrine** describes the cranium as a rigid box that is filled with a nearly incompressible brain and whose total volume tends to remain constant. This doctrine assumes that the cranium volume includes three compartments: (1) brain and medulla spinalis with meninges, (2) CSF blood vessels with blood, and (3) CSF space. Expansion of any of these three volumes does so at the expense of the other two elements; such an event may be precipitated by intracranial bleeding, cerebral edema, mass formation, or inability to regulate the production and removal of CSF.

CSF is constantly secreted. After circulating, it is absorbed at a rate equal to the rate at which it is produced. CSF circulation is a slow process, with only 500 to 700 mL being circulated each day. At any given time, the cranium contains 75 mL of CSF. Excesses of CSF can be displaced through the foramen magnum into the spinal theca, a sac between C1 and C2. This may help in cases of cribriform plate damage if the channel for drainage becomes blocked. The spinal dural sheath can accept a quantity of CSF because it does not adhere to the canal closely and it is surrounded by a layer of loose areola tissue and a plexus of epidural veins. In addition, during periods of increased ICP, blood through venous emissaries increases.

Intracranial circulation of blood is approximately 1,000 L/d, delivered at a pressure of 100 mm Hg. At any given time, the cranium contains 750 mL of blood. Obstruction to venous outflow leads to an increase in the volume of intracranial blood, resulting in increased ICP. As the ICP increases, the cerebral venous pressure increases in parallel so that it remains 2 to 5 mm higher than the ICP, a differential that prevents the collapse of the venous system.

Increases in ICP can result from changes in CSF, edema, expansible masses, and changes in vessel size. The ability of the skull contents to compensate for the increased pressure depends on the location and rate of expansion of the lesion and the **brain tissue compliance** or volume-buffering capacity of the system. (Brain tissue compliance is defined as the change in brain volume resulting from a change in pressure.)

Cerebral Blood Flow

The brain accounts for only 2% of total body weight, yet its blood flow represents 15% of resting cardiac output and it uses 20% of the total amount of oxygen consumed by the body. Every 24 hours, the brain requires 1,000 L of blood in addition to its normal diet of 71 L of oxygen and 100 g of glucose. The **cerebral blood flow (CBF)** remains constant over a wide range of arterial pressures between 60 mm Hg and 150 mm Hg; with a rise above 150 mm Hg, however, blood flow increases. CBF ceases when MAP drops to 20 mm Hg, except in the chronically hypertensive patient, in whom this autoregulation limit

appears to be reset. The exact nature of this autoregulation is not known; we know only that many factors, including direct reaction of the cerebral arterial smooth muscles to stretching, various by-products of metabolism, and direct control by nerves surrounding vessels, seem to have a role.

With a normal cerebrovascular system and blood pressure, even moderate alterations of P_{CO_2} are capable of markedly altering CBF. Within the range of 30 to 60 mm Hg, there is a 2.5% change in CBF as the P_{CO_2} changes by 1 mm Hg. With a pressure of less than 20 mm Hg or more than 80 mm Hg, there is no further change. As the body ages and with the development of arteriosclerosis, there is a marked decrease in P_{CO_2} influence.

The effect of P_{O_2} changes is not as obvious as with changes involving carbon dioxide (CO_2). Moderate variation of oxygen above or below the normal level does not affect CBF. The presence of P_{O_2} causes vasoconstriction of a nonischemic brain along with a reduction in CBF. Alternatively, in the ischemic area of the brain, increasing the P_{O_2} has no effect. Cerebral vasodilation begins with a P_{O_2} of 50 mm Hg, resulting in increasing CBF. By the time the P_{O_2} falls to 30 mm Hg, CBF may have already tripled.

ICP influences the CBF through the CPP, which is the difference between MAP and ICP. An increase in ICP leads to a fall in the CPP. Every effort should be made to maintain the CPP at 50 mm Hg or more during treatment of increased ICP **Figure 11-43**.

For the CCTP, the following key points related to CBF are crucial:

- A number of physiologic factors may affect or change CBF. Increases in CBF as a result of hypoxia or hypercapnia (increased levels of CO_2 in blood), for example, will cause an increase in ICP once the normal compensating mechanisms have been exhausted.
- Poor airway and ventilatory control contributes to hypoxia, hypercapnia, and hypotension, and will further damage the already critically ill brain.

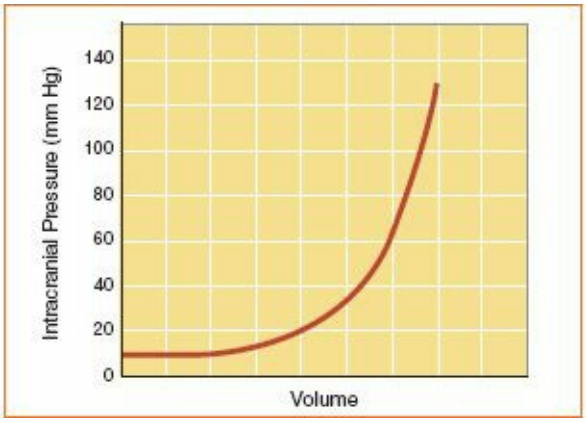


Figure 11-43 An intracranial pressure curve.

Cerebral Perfusion Pressure

CPP is responsible for the movement of blood through the brain and is the result of the difference between MAP and ICP (CPP = MAP – ICP). This relationship represents the pressure gradient driving CBF, and hence oxygen and metabolite delivery. The uninjured brain is capable of autoregulating its blood flow to provide a consistent flow of blood, regardless of blood pressure, by altering the resistance of cerebral blood vessels.

These homeostatic mechanisms are often lost after head trauma. As a consequence, a patient with TBI may have increased cerebral vascular resistance, making the brain more susceptible to changes in blood pressure. Those areas of the brain that are ischemic or at risk for ischemia are critically dependent

on an adequate CBF and, therefore, CPP.

For the CCTP, the following key points related to CPP are crucial:

- Maintenance of CPP reduces mortality in severe head injury.
- CPP should be maintained between 50 and 70 mm Hg to reduce the risk of poor outcomes for patients with increased ICP.
- Systemic hypotension (systolic blood pressure, <90 mm Hg) is associated with a poor prognosis.
- Maintenance of an adequate CPP is the cornerstone of modern brain injury therapy.

After brain injury, and especially in the patient who has suffered multisystem trauma, CBF may be dangerously low, bordering on ischemia. To prevent further neuronal death (secondary brain injury), the flow of well-oxygenated blood must be restored as soon as possible. The optimal level of CPP has not yet been determined, but the CPP target lies within the range of 50 to 70 mm Hg. When CPP is maintained above 70 mm Hg, with fluids and pressors, there is a significantly increased risk of adult respiratory distress syndrome (ARDS).

CPP may be maintained by raising MAP or lowering ICP. In practice, ICP is usually controlled to within normal limits (< 20 mm Hg), and MAP is raised therapeutically to a level of greater than 90 mm Hg. Although it is unknown whether ICP control is necessary, given that outcomes from severe TBI related to ICP seem to lie more in its role in determining CPP, it is clear that ICP plays a role in herniation. Current data therefore support initiating treatment when ICP exceeds 20 mm Hg.

Substantial evidence indicates that early hypotension (systolic blood pressure of less than 90 mm Hg) is associated with increased morbidity and mortality following severe brain injury. Even patients with one episode of hypotension during their intensive care unit (ICU) stay have a significantly poorer prognosis. To ensure that patients remain normovolemic, the CCTP must be vigilant in controlling other sites of hemorrhage while providing adequate oxygenation.

Many patients with TBI, because of the extent of their injuries, will require intensive neurologic monitoring. As previously discussed, CCTPs should perform serial neurologic examinations, especially for patients with TBI. CCTPs may also be required to maintain invasive neurologic monitoring devices. Each device must be maintained in accordance with the manufacturer's requirements, and it is the responsibility of each CCTP to become familiar with all aspects of each device prior to using it on a patient. Because of the often subtle, yet devastating, effects created by rising ICP, invasive monitoring can be a valuable tool that gives a more detailed picture of the patient's overall neurologic condition.

Pathophysiology of Increased Intracranial Pressure

The brain is encapsulated within the skull and surrounded by CSF that is generated in the choroid plexus and reassimilated by the subarachnoid villa to balance ICP between 5 and 15 cm H_2O. Because the skull is essentially an immovable structure, any change causing an additional change in volume will create an increase in ICP. The most common causes of increased ICP are cerebral hemorrhage, tumors, hydrocephalis, and trauma.

CSF volume variations result from many causes, including overproduction of CSF, decreased absorption of CSF, and obstructions in cerebral circulation. All of these conditions lead to an increased volume of CSF within the cranial vault and thus increased pressure on brain tissue.

Brain tissue is the most difficult component within the cranial vault to manipulate without surgical intervention. The parenchyma can respond to increases in ICP, changes in CSF and/or blood volumes, and changes in vessel size (which affect the volumes carried). Minimal volume changes incite the tissue to react by partial collapse of the cisterns, ventricles, and vascular system, which leads to decreased production and increased absorption of CSF.

Increased ICP is defined as a sustained elevation in pressure above 20 cm H_2O. During compensation, the ICP rises and plateaus, at which time the increased level of CSF absorption keeps pace with the increase in volume. Intermittent expansion causes only a transient rise in ICP at first. When a sufficient amount of CSF has been absorbed to accommodate the volume, ICP returns to normal. Once compensatory mechanisms are no longer effective, however, pressure increases rapidly, followed by shifting of the brain tissue toward open spaces in the skull. This shift, which results in decreased blood flow to the medulla, is accompanied by dilation of small arteries in the pia mater and some slowing of venous flow, followed by pulsatile venous flow.

The following compensatory mechanisms help maintain ICP:

- Shunting of CSF into the spinal subarachnoid space
- Increased CSF absorption
- Decreased CSF production
- Shunting of venous blood out of the skull

When ICP rises to the level of the arterial pressure, a cascade of events known as Cushing's triad begins. Cushing's triad is the combination of progressively increasing systolic blood pressure, with a progressively decreasing diastolic blood pressure (widening pulse pressure) and bradycardia. It is a sign of increased ICP, which occurs as the result of Cushing's reflex. Vasoconstriction occurs as the body attempts to shift fluid volumes in the cranium, thereby reducing ICP. If ICP continues to rise, brain displacement accelerates, putting pressure on the medulla oblongata by pushing it into the foramen magnum, resulting in disturbances in breathing, heart rate, and blood pressure. The three effects that result from this process—hypertension, bradycardia, and respiratory irregularities—are collectively Cushing's triad. Cushing's triad is a late sign and often indicates that severe and even irreversible injury has taken place within the cranial cavity. Identification is important because it suggests severe ischemia in the brain. Possible causes are cerebral hemorrhage, a brain tumor, or brain herniation, which can be fatal.

Special Populations

Children are much more sensitive to fluid shifts than adults. Be very careful when administering fluid to children.

The rise in ICP to the level of systemic arterial pressure extinguishes cerebral circulation. This situation will be reversed only if arterial pressure rises sufficiently higher than ICP, which restores CBF. If this effort fails, brain death occurs.

The cause of a rise in ICP and the rate at which it occurs are also important. Many patients with benign **intracranial temperature (ICT)** or obstructive hydrocephalus show few or no ill effects despite their increased ICP, because the brain itself is normal and autoregulation is intact. Conversely, cerebral blood flow may be compromised even when ICP is relatively low if parenchymal lesions—such as tumors, hematomas, and contusions—cause the brain to shift and disrupt autoregulation. In acute hydrocephalus, there is rapid deterioration because there is no time for compensation.

Signs and Symptoms

Increased Intracranial Pressure

- Cushing's triad (widening pulse pressure, bradycardia, and abnormal respiratory patterns)
- Hypertension (increased blood pressure)
- Increased respiratory rate
- Pulmonary edema

Transport Management

Increased Intracranial Pressure

- Intubate and mechanically ventilate the patient. Do not hyperventilate the patient unless he or she acutely deteriorates.
- Consider administering an osmotic diuretic (mannitol).
- Consider administering dexamethasone or methylprednisolone sodium succinate to control inflammation and swelling.
- Control blood pressure to maintain systolic pressures at less than 150 mm Hg.
- If a surgical drain (bolt) has been installed, drain the CSF if the ICP is greater than 20 cm H_2O.
- For patients who have sustained a hemorrhage and who have been taking warfarin (Coumadin) (patients with atrial fibrillation), administer either platelets or whole blood en route. These patients may also require a paralytic agent.

Disruption of brain function secondary to ICP may include the following conditions:

- Reduction in CBF.
- Transtentorial or foramen magnum herniation resulting in selective compression and ischemia of the brain stem.
- Transtentorial herniation with brain stem compression that leads to clinical deterioration even with adequate CBF. A temporal mass may cause uncal herniation without raised ICP. Similarly, a frontal mass can cause axial distortion and impair brain stem perfusion.

Clinical Manifestations of Increased ICP

The clinical features of increased ICP are a result of Cushing's triad and are indicated by arterial hypertension, bradycardia, and respiratory changes. It has traditionally been thought that the hypertension and bradycardia result from ischemia or pressure on the brain stem. Some have suggested, however, that cerebral ischemia might lead to removal of supratentorial inhibition of brain stem vasopressor centers and that bradycardia occurs independently of the rise in blood pressure.

Respiratory changes depend on the level of brain stem involved. Midbrain involvement results in Cheyne-Stokes respiration. When the midbrain and pons are involved, there is sustained hyperventilation. With upper medulla involvement, rapid and shallow respirations are followed by ataxic breathing in the final stages.

Increased ICP also has pulmonary effects. Pulmonary edema seems to be due to increased sympathetic activity as a result of the effects of raised ICP on the hypothalamus, medulla, or cervical spinal cord. This is a direct result of the disruption of the pulmonary capillary integrity and increased left arterial pressures.

Brain Herniation

Brain herniation is a condition in which a portion of the brain is displaced because of increased ICP, resulting in progressive damage to brain tissue that may include life-threatening damage to the brain stem. This condition is commonly the result of brain swelling (cerebral edema) from a head injury, but it also may be the result of hemorrhagic stroke or space-occupying lesions, such as primary brain tumor, metastatic brain tumor, or other lesions within the brain. Brain herniation may also occur with bacterial meningitis.

The most common type of brain herniation occurs when a portion of the temporal lobe is displaced (**uncal herniation**), resulting in compression of cranial nerve III, the midbrain, and the posterior cerebral artery. Uncal herniation typically leads to coma and respiratory arrest.

Another critical type of brain herniation occurs when part of the cerebellum is displaced through the foramen magnum (tonsillar herniation). This compresses the brain stem, resulting in destruction of the respiratory center, apnea, decreased perfusion to the rest of the brain, and death. Because this herniation involves the brain stem and spinal cord, the patient will likely be in a coma, present with decerebrate or decorticate posturing, have ipsilateral pupil dilation, and have difficulty maintaining respirations, heart rate and rhythm, and blood pressure. These patients need immediate surgery to install a drain to remove blood and fluids to alleviate pressure. From a transport point of view, management includes the following:

Signs and Symptoms

Brain Herniation

- Tachycardia (inability to control heart rate and rhythm)
- Decreased blood pressure (inability to control blood pressure)
- Decreased respiratory rate (inability to control respirations—irregular breathing)
- Abnormal posturing (decorticate/decerebrate)
- A worsening (progressive) LOC (GCS score of 5 or less)
- Pupil changes (dilation of one or both pupils or failure of pupils to constrict with light)

Transport Management

Brain Herniation

- Intubate the patient and place him or her on a mechanical ventilator, hyperventilating to maintain CO_2 levels of 25 to 35 mm Hg.
- Administer mannitol or an equivalent osmotic diuretic.
- Consider administering corticosteroids such as dexamethasone, especially in the case of a brain tumor.
- Provide rapid transport to a neurosurgical facility.

1. Intubate the patient and place him or her on a mechanical ventilator. The patient should be hyperventilated to maintain CO_2 levels of 25 to 35 mm Hg.
2. Mannitol or an equivalent osmotic diuretic should be administered.
3. Corticosteroids such as dexamethasone should be considered, especially in the case of a brain

tumor.

4. Provide rapid transport to a neurosurgical facility.

Other areas of the brain may suffer herniation, although such injuries occur with less frequency than either uncal herniation or foramen magnum herniation.

ICP Monitoring

The primary goals of ICP monitoring are identification of ICP trends and evaluation of therapeutic interventions to minimize ischemia in the brain-injured patient. Intracranial hypertension (sustained ICP ≥ 15 mm Hg) results when the brain's protective mechanisms to shunt CSF to the subarachnoid space or to vasoconstrict cerebral arterioles fail to maintain the ICP below 15 mm Hg. Intracranial hypertension compromises the relationship between systemic blood pressure and the resistance that must be overcome to accomplish cerebral perfusion. When CPP falls below 50 mm Hg, secondary brain ischemia, herniation, and ultimately brain death occur. ICP monitoring allows for early detection of intracranial hypertension and subsequent aggressive management.

Indications for ICP Monitoring

The primary reason for ICP monitoring is a critical injury or illness involving components of the skull, specifically the brain. The following indications warrant ICP monitoring:

- Severe TBI (see previous criteria)
- Intracranial hemorrhage
- Cerebral edema
- Post-craniotomy
- Space-occupying lesions, such as epidural and subdural hematomas, tumors, abscesses, or aneurysms that occlude the CSF pathway
- Patients with Reye syndrome who develop coma, posturing, and abnormal responses to noxious stimuli
- Encephalopathy from lead ingestion, hypertensive crisis, or hepatic failure
- Meningitis/encephalitis resulting in malabsorption of CSF
- GCS score of less than 8 or a positive CT (there is evidence of a brain hemorrhage or evidence of a condition that will lead to a brain hemorrhage)

Contraindications for ICP Monitoring

The relative contraindications for ICP monitoring are as follows:

- CNS infection
- Coagulation defects
- Coagulopathy
- Scalp infection
- Severe midline shift resulting in ventricular displacement
- Cerebral edema resulting in ventricular collapse

ICP Monitoring Methods

ICP can be monitored using either noninvasive or invasive techniques. Noninvasive ICP monitoring simply involves assessment for clinical deterioration in neurologic status. Signs such as bradycardia, increased blood pressure, and pupillary dilation are reliable signs of rising ICP. Invasive methods,

however, provide a quantitative measure of pressure, and some allow for CSF drainage. Several invasive devices are available **Figure 11-44**, but the most commonly used are the intraventricular catheter and subarachnoid screw. ICP devices are placed in the hospital setting and never during transport. It is more common to transport a patient to a facility to have an ICP device inserted than to transport a patient with the device already in place.

Intraventricular monitoring remains one of the most popular techniques for monitoring ICP, especially in patients with ventriculomegaly (an enlarged ventricle of the brain). An additional advantage of this method is that it is both diagnostic and therapeutic, with the potential for draining CSF. Insertion of a ventricular catheter is not always a simple matter, however, because placement of such a device can cause hemorrhage and infection.

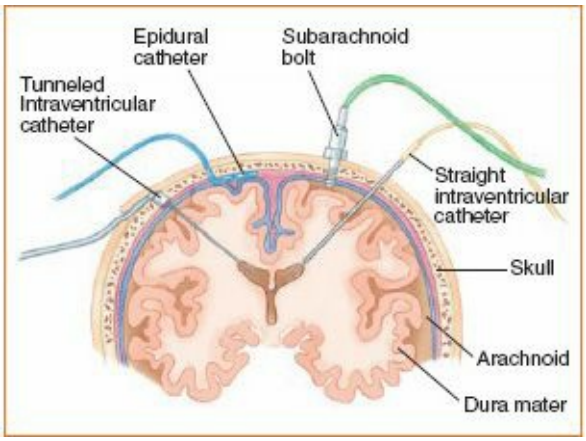


Figure 11-44 Invasive methods for intracranial pressure detection and cerebrospinal fluid drainage.

Advantages of an intraventricular cannula (IVC) are as follows:

- Allows for draining of CSF so as to lower ICP
- Allows CSF cultures to be obtained
- Provides increased accuracy in ICP monitoring
- Is accurate and reliable

Risks associated with use of an IVC include the following problems:

- Infection
- Injury to brain
- Clot formation
- Hemorrhage risk
- Collapsed ventricle
- Difficult or impossible placement

The epidural catheter lies beneath the skull, above the dura mater. It offers two advantages: a decreased rate of infection compared to the IVC and a lower risk that its placement will injure the brain. However, this device is also less accurate than the IVC, and it cannot be used to drain CSF.

The technique of inserting a ventricular catheter requires the insertion of a soft catheter through a burr hole into the lateral ventricle. The catheter allows for both monitoring of ICP and for therapeutic drainage of CSF to reduce ICP. Intraventricular catheters connect to a transducer set, which is never pressurized. Complications associated with the intraventricular catheter include infection and hemorrhage. The device must also be carefully leveled at the foramen of Monro (halfway between the

outer canthus of the eye and the tragus of the ear). Any fluid drained must be monitored for amount, color, and clarity at hourly intervals. Manipulation of the catheter by the CCTP should be avoided, and recalibration should be performed only if the CCTP has been given approval by medical direction and has been properly trained to operate the particular device in use.

The subarachnoid screw (or "bolt") is a device that also may be used to monitor ICP, although its accuracy is lower than the more direct ventriculostomy drain just described. Oftentimes a fiberoptic monitor is inserted through the bolt. Fiberoptic monitoring devices have a pressure sensor at the tip and do not require leveling or zeroing. The fiberoptic cable, however, is fragile and can break if twisted, stretched, or bent. An alternative to fiberoptic monitoring is to use a fluid-filled catheter connected to an arterial pressure monitoring system. Manipulation of the catheter by the CCTP should be avoided, and recalibration should be performed only if the CCTP has been given approval by medical direction and has been properly trained to operate the particular device in use.

The subdural bolt is inserted beneath the dura mater and above the pia mater. The subarachnoid bolt is placed beneath the arachnoid mater and above the pia mater.

Advantages of these devices include the following:

- Not as invasive as IVC or epidural catheter
- Lower rate of infection
- Less injury to the brain
- Easier to place

Disadvantages of these devices include the following:

- Less accurate monitoring
- Cannot drain CSF
- Risk of bleeding and brain injury
- Higher rate of infection than epidural catheter
- Requires closed, intact skull

A fiberoptic transducer-tipped probe is a catheter with a pressure-sensing device that is placed into the subdural space, brain parenchyma, or ventricle. This non–fluid-filled continuous intact system offers the advantages of a good waveform, reliable and accurate pressure readings, and lack of air bubble formation within the catheter. Disadvantages include the inability to access ICP unless an IVC setup is used and the inability to zero the unit once it is in place.

Additional commonly used devices are the hollow screw-and-bolt devices and the subdural catheter. The Richmond screw and Becker bolt are used extradurally. The Ladd device uses a fiberoptic system to detect distortions in ICP; it can be used in the subdura, on an extradural basis, and even extracutaneously.

Mechanically coupled surface monitoring devices include cardio search pneumatic sensors that are used subdurally or extradurally. Electronic devices (Camino and Galtesh design) are fully implantable systems and are valuable in the small group of patients who require long-term ICP monitoring for brain tumors, hydrocephalus, or other chronic brain diseases. A Cosmon ICP telesensor can be implanted as part of a shunt system. The Ommaya reservoir is an alternative that can be implanted for the purpose of administering medications, removing CSF, and obtaining CSF pressure readings. The Ommaya reservoir is placed directly in the spinal fluid; therefore, any medication given (such as chemotherapy agents) can be injected directly into the CSF. The same medications injected into the bloodstream are much less effective because they are blocked by the blood-brain barrier. This device is most commonly used to treat cancers of the brain and spinal column.

Alternatively, a fluid-filled catheter that is placed in the subdural space and connected to an arterial

pressure monitoring system is cost-effective and serves the purpose adequately. The ICP waveform obtained through this system resembles a dampened arterial blood pressure waveform and is considered normal when the pressure is between 0 and 15 mm Hg **Figure 11-45**. In addition, a stopcock within the system allows for therapeutic drainage of CSF and for sampling for infection surveillance.

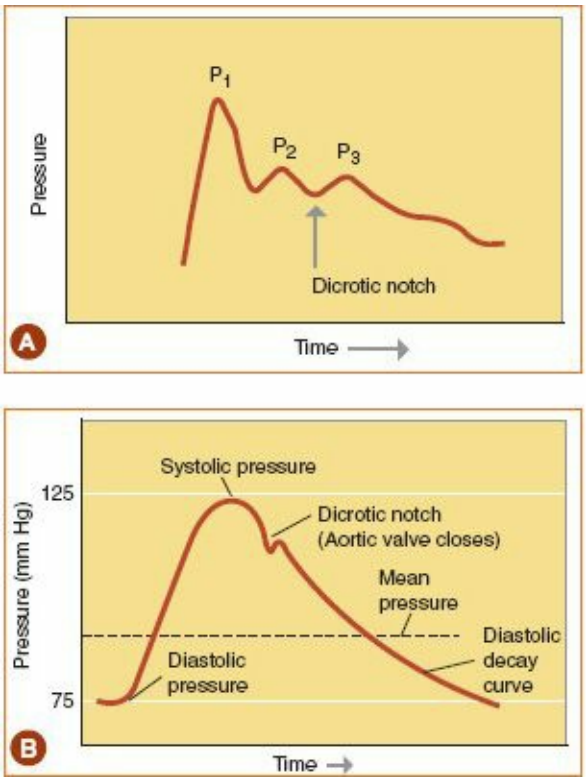


Figure 11-45 **A.** An intracranial pressure waveform. **B.** An arterial blood pressure waveform.

ICP monitoring undoubtedly helps in the management of conditions in which prolonged intracranial hypertension is expected. Indeed, monitoring is the only means by which providers can collect data that allow for selection of the most appropriate therapy and then accurately study the effectiveness of that therapy. ICP monitoring should be employed in all salvageable patients with severe TBI, a GCS score of less than 8 but greater than 3 after resuscitation, and an abnormal CT scan (hematomas, contusions, swelling, herniation, or compressed basal cisterns).

ICP monitoring is also indicated for patients with severe TBI with a normal CT scan if two or more of the following are noted at admission:

- Age over 40 years
- Unilateral or bilateral motor posturing
- Systolic blood pressure of less 90 mm Hg

Patients with severe TBI are intubated to protect the airway and allow maximal oxygenation. Standard monitoring for these patients is required, including oxygen saturation (SpO_2), electrocardiography (ECG), MAP, and urine output. Unless end-tidal CO_2 ($ETCO_2$) can be continuously monitored, these patients also require frequent determination of arterial blood gases, which can be achieved by placement of an intra-arterial catheter. Patients are maintained euvolemic (normal fluid volume status), and central venous pressure measurements are used to guide therapy. Central venous pressure measurement is covered in Chapter 16: Hemodynamic Monitoring.

Normocapnia (normal CO_2 tension) is vital for maintenance of ICP, and patients should have continuous measurement of $ETCO_2$ levels using a capnometer. These measurements represent the baseline

requirements for monitoring of patients with TBI. Patients receiving pressors to maintain MAP and/or CPP would benefit from an arterial line for continuous monitoring of drug effects.

Key points in monitoring ICP are as follows:

- The baseline requirements are SPO_2, ECG, MAP, $ETCO_2$, central venous pressure, and urine output.
- ICP and blood pressure monitoring are the only way to reliably determine CPP and assess cerebral hypoperfusion in patients with severe brain injury.
- Multimodality monitoring, including jugular venous oxygen saturation (SjO_2), brain tissue oxygen tension ($P_{br}O_2$), and TCD ultrasound, should be used when ICP and CPP cannot be maintained by standard methods.

The role of multimodal monitoring devices remains under investigation but approaches a long desired need in neurocritical care to obtain information about cerebral blood flow and metabolism in the brain. Treatment thresholds have been published for SjO_2 (< 50%) and for $P_{br}O_2$ (< 15 mm Hg), which, to date, are the only monitoring systems that have yielded sufficient outcome data in patients with TBI. A full discussion of these technologies is beyond the scope of this text.

As mentioned earlier in this section, CPP is maintained by supporting MAP and/or reducing ICP. ICP transducers should measure the pressure range of 0 to 100 mm Hg, with an accuracy of 2 mm Hg in the range of 0 to 20 mm Hg and at least 10% accuracy for the rest of the measurable range.

ICP monitoring is a complex task that requires knowledge and understanding of the technical components of fluid-filled monitoring systems, the pathophysiology of the CNS, and the interaction between these systems. Personnel performing the setup, data collection, or maintenance procedures should be appropriately trained and credentialed and should be competent in:

- The technical setup and operation of the pressure monitoring system
- CNS physiology and pathophysiology
- ICP waveform analysis
- Appropriate response to adverse reactions
- Application of universal precautions

Complications of ICP Monitoring

In general, complications related to ICP monitoring are rare. The greatest concern is ventriculitis (meningitis), although it has never been demonstrated in prospective studies of clinically significant intracranial infections following ICP measurement. Bacterial colonization does occur (5% ventricular/subarachnoid, and 15% parenchymal), however, and its incidence increases markedly after five consecutive days of monitoring. Irrigation of ICP devices to restore patency of the ventriculostomy catheter using either an isotonic sodium chloride solution or antibiotic solution significantly increases the risk of colonization. Definitive treatment is removal of the ICP bolt.

The risk of hematoma formation associated with ICP monitors varies. Parenchymal catheters have a higher incidence of hematoma than do other monitoring methods. Malfunction of the devices does occur, and readings of more than 50 mm Hg may be inaccurate with higher rates of obstruction and loss of signal.

Complications of ICP monitoring can be summarized as follows:

- Intracranial infection
- Intracerebral hemorrhage
- Air leakage into the ventricle or subarachnoid space
- CSF leakage

- Overdrainage of CSF, leading to ventricular collapse and herniation
- Occlusion of the catheter with brain tissue or blood
- Inappropriate therapy because of erroneous ICP readings owing to dampened waveforms, electromechanical failure, or operator error (ie, inappropriate leveling)

Benefits and Problems of ICP Monitoring

When clinical monitoring is not possible, such as during hyperventilation therapy and high-dose barbiturate therapy, ICP monitoring may prove helpful. Preoperative monitoring, for example, assists in assessment of **normal pressure hydrocephalus (NPH)** before a shunting procedure takes place. ICP monitoring can provide an additional assessment in the event of suspected brain death because brain perfusion effectively ceases once ICP exceeds diastolic blood pressure.

Problems associated with ICP monitoring include its cost, the risk of infection, and the risk of hemorrhage. Effective maintenance of ICP monitoring requires a team effort.

Multimodality Monitoring

Although maintenance of CPP is important, it measures only one parameter affecting the delivery of oxygen to the neurons. Ultimately, the CBF and oxygen content of the blood are the prime parameters of concern. Although CPP provides a pressure gradient governing CBF, flow is subsequently affected by the resistance of the cerebral vessels. Neuronal demand for oxygen is governed by the metabolic rate of the neurons—that is, neurons with high activity levels require greater amounts of oxygen than do quiescent neurons. Globally, this is described as the **cerebral metabolic rate for oxygen ($CMRO_2$)**:

$$CMRO_2 = CBF \times OEF \times SpO_2$$

The **oxygen extraction fraction (OEF)**, or how much oxygen is extracted as it passes by and is, therefore, available to support brain functions, can be measured by the **Fick principle** based on measurements of the arterial and venous oxygen content. The Fick principle is used to determine cardiac output, such that the amount of oxygen uptake of blood as it passes through the lungs is equal to the oxygen concentration difference between mixed venous and arterial blood.

Thus, monitoring only the ICP and CPP provides little information about the overall state of the injured brain and no information about oxygen delivery and usage. Multimodality monitoring involves using a combination of jugular venous bulb oximetry, brain tissue oxygen tension, and transcranial Doppler ultrasound, which allows a greater understanding of cerebral circulation and oxygen consumption.

Jugular venous bulb oximetry involves placing a sampling catheter in the internal jugular vein, directed upward, so that its tip rests in the jugular venous bulb at the base of the brain. Blood samples drawn from here measure the **mixed venous oxygen saturation (SvO_2)** of blood leaving the brain, which is normally in the range of 50% to 75%.

The SvO_2 may fall when there is an imbalance between oxygen consumption and delivery, when there is a fall in CBF, when there is a rise in oxygen use, or when there is increased cerebrovascular resistance (CVR). Vascular spasm and a rise in CVR are very common after brain injury and are significantly worsened by hyperventilation. Jugular venous bulb oximetry should be used whenever the patient has experienced prolonged hyperventilation.

Brain tissue oxygen tension ($P_{br}O_2$) monitoring is accomplished by placement of a commercial probe (capable of measuring temperature and oxygenation) into the brain tissue through a bolt. Placement technique is similar to ICP monitoring placement and can be done simultaneously with ICP monitoring

placement.

Brain tissue oxygenation monitoring is more sensitive for monitoring focal disruption of blood flow to particular areas of the brain (depending on catheter placement). When compared to jugular venous bulb oximetry, which only detects global ischemic insults, brain tissue oxygen tension appears to be more accurate and may be more practical for continuous monitoring.

Transcranial Doppler ultrasound is a noninvasive method of assessing the state of the intracranial circulation. The velocity of flow can be measured in the middle, anterior, and posterior cerebral arteries; the ophthalmic artery; or the internal carotid artery. Doppler waveform analysis can give further information about the state of blood flow, such as flow acceleration and pulsatility index ([systolic velocity–diastolic velocity]/mean velocity).

Vasospasm is common after head injury and can be an important cause of neurologic deterioration. Vasospasm usually occurs in the presence of traumatic subarachnoid hemorrhage. Vasospasm leads to an increase in flow velocity; therefore, TCD is useful for monitoring at-risk patients for signs of vasospasm.

TCD is indicated for patients whose intracranial hypertension and CPP cannot be maintained by standard therapy.

All of the previously mentioned monitoring technologies assess oxygen delivery to and extraction by the brain; none measures cerebral activity directly. A full EEG is too complex for continuous use in the ICU, but the **cerebral function monitor (CFM)** or **cerebral function analysis monitor (CFAM)** can provide summed, averaged, and (in CFAM's case) analyzed outputs of the general state of brain activity. A CFM records a single channel EEG from two electrodes placed on either side of the head and a third electrode that acts as a ground. The CFM is used to assess brain wave activity, indicate the effects of medication administration, and identify seizure activity. It is particularly useful in evaluating the effects of sedation and paralytics.

Performing ICP Monitoring

Prior to performing any procedure, the appropriate equipment should be available and the patient should be prepared for the procedure. It is necessary to ensure the procedure is done in a controlled environment with adequate support staff. Every effort must be made to anticipate difficulties, and providers must have a thorough understanding of troubleshooting techniques. This section outlines the equipment required, precautions, the step-by-step procedure, adverse reactions and interventions to mitigate them, and postprocedure checks and documentation.

Equipment

The equipment necessary for ICP monitoring includes the following items:

- External drainage system with mounting card
- Pressure transducer with 48″ pressure tubing
- Sterile 0.9% sodium chloride (NaCl) with sterile 20-mL syringe
- Pressure monitoring cable
- IV tubing
- Manual resuscitator, mask, and 100% oxygen source
- Cardiopulmonary monitor
- BSI attire

Procedure

Skill Drill 11-1 shows the procedure for monitoring ICP with the Cordis EDS device, one of the most

commonly used ICP monitoring devices. The procedure is also described here:

1. Assemble the Cordis EDS as instructed per the package insert and per the mounting card diagrammatic instructions, using 0.9% NaCl to flush the system. Place the 48″ tubing, with connected pressure transducer, on the plastic mounting card **Step 1**.
2. Avoid tension or kinking of the pressure monitoring cable.
3. Place the patient in a semi-Fowler's position and position the zero reference point at the outer canthus of the eye **Step 2**.
4. Obtain the ICP waveform on the cardiopulmonary monitor. Turn the stopcock nearest the patient so that the ICP waveform is visualized **Step 3**.
5. Zero the transducer at the distal stopcock **Step 4**.
6. Record the ICP on the printer and mark and post the strip. Calculate and record the CPP on the strip **Step 5**.
7. For continuous drainage, turn the stopcock nearest the patient so that drainage resumes and pressure monitoring is discontinued. *Note:* It is recommended that pressure monitoring and CSF drainage not be done simultaneously **Step 6**.
8. Maintain a clean and intact dressing. If the dressing is soiled or contaminated, change it using a sterile technique.

Precautions

To minimize the risk of an infectious agent entering the CNS during ICP monitoring, an aseptic technique must be used at all times when assembling, manipulating, or accessing the fluid-filled monitoring system. Tight connections must be maintained, and the system must remain free of air to ensure maximal accuracy. Never use a flush device for ICP monitoring. Use only sterile 0.9% NaCl to fill the pressure tubing. Never use a heparinized solution.

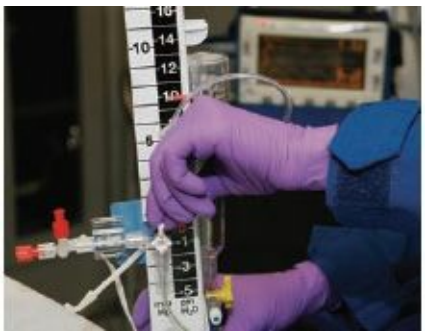
To ensure optimal accuracy, proper leveling and zeroing of the system must be maintained. The proper level for the transducer is at the foramen of Monro measured at the level of the outer canthus of the eye or, alternatively, at the spine for lumbar drainage. Utmost care must be taken when positioning and turning the patient to avoid accidental decannulation or disconnection of the tubing. Patients are maintained at a 30° to 45° head-up and neutral position when necessary to minimize the ICP. Use extreme caution when positioning patients and performing therapy so as to minimize increases in ICP and associated degradations in CPP.

Skill Drill 11-1

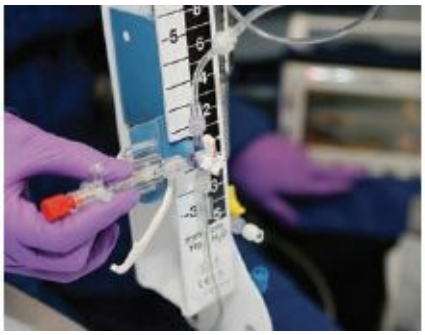
Performing ICP Monitoring

1 Assemble the device as instructed per the package insert and per the mounting card using 0.9% NaCl to flush the system. Place the 48″ tubing, with connected pressure transducer, on the plastic mounting card. Avoid tension or kinking of the cable.

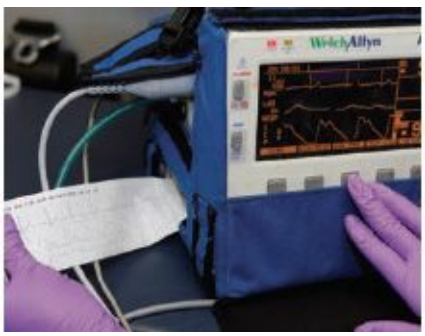
2 Place the patient in a semi-Fowler's position and position the zero reference point at the outer canthus of the eye.



3 Obtain the ICP waveform on the cardiopulmonary monitor. Turn the stopcock nearest the patient so that the ICP waveform is visualized.



4 Zero the transducer at the distal stopcock.



5 Record the ICP on the printer and mark and post the strip. Calculate and record the CPP on the strip.

6 For continuous drainage, turn the stopcock nearest the patient so that drainage resumes and pressure monitoring is discontinued. Maintain a clean and intact dressing.

Avoid flexion and hyperextension of the neck and positioning the patient in a Trendelenburg position, all of which may increase ICP. An alarm for high ICP must be maintained in the "on" position at all times.

Use care when manipulating the drainage system to avoid getting the filter wet. The drainage cylinder should remain in an upright position at all times. If the filter should get wet and drainage is slowed or stopped, some time may be required for the filter to dry out sufficiently before adequate drainage is reestablished.

The Cordis EDS and pressure transducer systems **Figure 11-46** are intended for single-patient use only. Do not sterilize any part of the disposable system for reuse.

Simultaneous drainage and pressure monitoring are not recommended. To ensure precise pressure measurements, perform only pressure monitoring while keeping the stopcock closed to the drainage system.

Only a small amount of CSF (not to exceed 3 mL) should be drained at one time. Rapid cerebral decompression from CSF overdrainage may result in herniation; such overdrainage may occur if the system is inadvertently left open or if the patient is maintained at a higher level than the reference point on the system (as per physician order). The height of the drainage cylinder determines the rapidity at which CSF will drain. The physician's order for drainage must include a pressure reading in either mm Hg or cm H_2O and the height (as marked on the mounting card) at which the drainage cylinder must be maintained. The zero reference point for ICP monitoring always remains the outer canthus of the eye (or for lumbar drainage, the spine).

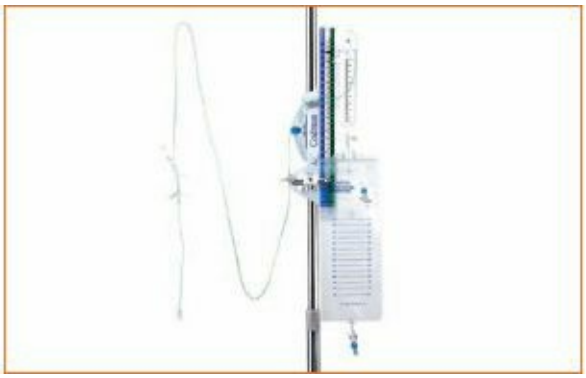


Figure 11-46 The Cordis EDS.

Adverse Reactions and Interventions

Some patients may experience adverse reactions as a result of ICP monitoring. These reactions and the appropriate interventions to mitigate them are discussed here.

First, if blood is visualized in the pressure tubing (from intracranial hemorrhage), notify the physician.

If a good waveform and/or accurate ICP cannot be obtained (ie, a dampened waveform appears), a flush of the fluid-filled monitoring system (with sterile 0.9% NaCl) may be attempted. All connections should first be checked for tightness. Never flush the system while it is open to the patient. Close the stopcock to the patient and then attempt to flush the monitoring system. Resume pressure monitoring by reopening the system to the patient. If a poor waveform persists, troubleshoot the system as you would any other, including debubbling, releveling and zeroing, and changing the electrical cable. If all of these maneuvers fail to correct the poor waveform, notify the physician. The catheter may be occluded with blood or tissue, requiring physician intervention.

An acutely low ICP may indicate acute decompression due to leakage or overdrainage of CSF. Never attempt to tamponade or block leakage from the site around an ICP monitoring device. In this situation, the physician should be notified immediately.

In the event of acute decompensation (ie, sustained ICP $\geq$ 15 mm Hg), be prepared to hyperventilate the patient with a manual resuscitator (bag-mask device) connected to 100% oxygen. Notify the physician.

Notify the physician immediately if the patient shows signs of decompensation—altered level of consciousness, restlessness, agitation, lethargy, confusion, motor weakness, seizures, alteration in breathing pattern, increases in blood pressure, bradycardia, vomiting, decortication/decerebration, or coma.

Post-Procedure Care and Documentation

Once ICP monitoring is established, do the following.

1. Ensure that all connections are tight and that the drainage system is positioned at the precise height for drainage according to the physician's order before leaving the bedside.
2. Relevel and zero the system every 4 hours and as needed.
3. Change the transducer pressure tubing every 72 hours as per the local policy.
4. Post a strip of the ICP in the patient's chart at least once per shift. It is probable that ICP will be measured much more frequently. Each posted strip should also have documentation of the corresponding CPP.

Treatment Options for Increased ICP

The best treatment for increased ICP is obviously the removal of the causative lesion, such as a tumor, hydrocephalus, or hematoma. Postoperative increased ICP has become less common with more widespread use of the microscope and special techniques to avoid brain retraction. A patient with a basal meningioma (a tumor near the base of the meninges) that has been completely removed usually has a smooth postoperative period. By contrast, a convexity or even falx meningioma (a tumor near the falx cerebri of the meninges) may be easily removed, but the postoperative period may be difficult. In such a case, problems may arise because of impairment of venous drainage, from either intraoperative injury to veins or postoperative diuretic therapy (as practiced in some centers).

Debate persists about whether increased ICP is the cause or the result of brain damage. Not all midline shifts seen in CT scans indicate increased ICP; they simply mean that ICP was high during the shift. A midline shift takes longer to reverse even after ICP returns to normal. In any event, increased ICP tends to be a temporary phenomenon that lasts for a short time unless there is a fresh secondary injury due to a clot, hypoxia, or an electrolyte disturbance.

First-Line vs Second-Line Management

Treatment is aimed at preventing the secondary events. Clinical and ICP monitoring definitely help in this regard. First-line management consists of general measures aimed at making the patient comfortable, along with effective handling of the ABCs of trauma management. Careful attention to nutrition and electrolytes, bladder and bowel functions, and appropriate treatment of infections should be instituted promptly. Adequate analgesia is often forgotten; it is a must even in unconscious patients.

Second-line management of increased ICP involves induced cerebral vasoconstriction, which may take the form of hyperventilation, hyperbaric oxygen, or hypothermia. Osmotherapy consisting of mannitol, glycerol, urea, or furosemide may also be implemented. Anesthetic agents, including barbiturates, gamma-hydroxybutyrate, and etomidate, and paralytic agents may also be used. Paralytics essentially paralyze the patient for the duration of transport and must be used with a ventilated patient. Preventing muscle contraction and movement can prevent exacerbation of a brain hemorrhage in certain cases. One example would be a patient with a brain hemorrhage who is taking warfarin (Coumadin). Warfarin cannot be reversed quickly even with administration of plasma, so administering a paralytic will be helpful to eliminate movement, reduce stress, and improve oxygenation. Usually a long-term nondepolarizing paralytic such as verconium bromide (Norcuron) is used. Paralytics should never be used without adjunctive analgesics or sedatives, because an awake paralyzed patient may develop increased ICP.

Surgical decompression remains controversial. It is performed to combat increased ICP, which is assumed when there is neurologic deterioration or when monitoring reveals that ICP is higher than 25 cm H_2O.

Specific Therapeutic Options

Transport treatment is aimed at reducing ICP and providing rapid transport to a neurologic facility for definitive treatment. Hyperventilation aims at keeping the P_{CO_2} between 25 and 35 mm Hg, so that CBF falls and capillary blood volume is reduced, thereby reducing the ICP. Although widely used, the reduction of CBF carries a significant risk of inducing cerebral ischemia, especially during the first day following injury when CBF has been demonstrated to approach half normal values. Prophylactic hyperventilation is not recommended. Hyperventilation should also be avoided in the first 24 hours following injury when CBF is often critically reduced. As a temporizing measure, hyperventilation is recommended to control elevations in ICP. Some have claimed that a P_{CO_2} of less than 20 mm Hg results in ischemia, although no experimental proof has definitively shown such a relationship. The present trend is to maintain ventilation with P_{CO_2} in the range of 30 to 35 mm Hg and with P_{O_2} in the range of 120 to 140 mm Hg to prevent hypoxia (standard of care for head injuries). When clinical deterioration such as pupillary dilation or widened pulse pressure is observed, hyperventilation may be instituted (preferably by increasing minute volume) until the ICP decreases. Whenever hyperventilation is used, cerebral oxygen delivery should be continuously assessed using jugular venous oxygen saturation or brain tissue oxygen tension measurement.

Controversies

A small group of surgeons initiate second-line management measures in conditions in which ICP is expected to rise without waiting for the rise to actually occur. Many other physicians believe that institution of measures to reduce ICP invariably compromises CBF; they prefer to wait for the rise in ICP before initiating second-line measures.

There is also debate about what the second line of management should be. Some prefer

hyperosmolar therapy; others prefer measures to induce cerebral vasoconstriction, thereby reducing CBF and reducing ICP. Finally, some providers believe it prudent to initiate both.

Ways to manage increased ICP during transport include:

1. Intubate and mechanically ventilate the patient. For intracranial hemorrhage, hyperventilate the patient, maintaining a CO_2 level of 25 to 35 mm Hg.
2. Consider administering an osmotic diuretic. Osmotic diuretics are low-molecular-weight substances that produce a rapid loss of sodium and water by inhibiting their reabsorption in the kidney tubules and the loop of Henle. They also increase the osmolality of plasma, increasing the diffusion of CSF. Osmotic diuretics are used to reduce intracerebral pressure created by excess CSF. Mannitol is the most commonly used osmotic diuretic, but glycerol is also used.
3. Consider administering dexamethasone or methylprednisolone sodium succinate to control inflammation and swelling. Dexamethesone and methylprednisolone sodium succinate are corticosteroids and are used to control inflammation and swelling of brain tissue.
4. Blood pressure should be controlled to maintain systolic pressures of less than 150 mm Hg.
5. If a surgical drain (bolt) has been installed, CSF should be drained if the ICP is greater than 20 cm H_2O.
6. Patients sustaining an intracranial hemorrhage and who have been taking warfarin (Coumadin) (patients with atrial fibrillation) should have either plasma or platelets administered en route and may require a paralytic.

Hyperosmolar therapy using mannitol, similar to hyperventilation, also bears risks of further reducing perfusion to the brain. Mannitol is still effective at reducing ICP, but only if it is used properly. Mannitol is not inert and harmless. Several theories have been advanced concerning the mechanism by which this drug reduces ICP:

- Mannitol increases cardiac output, which leads to better CPP and cerebral oxygenation
- Mannitol causes a decrease in CSF production
- Mannitol's diuretic effect may occur mainly around the lesion, where the blood-brain barrier integrity is impaired, so that the drug has no significant effect on the normal brain. For this reason, intra-axial lesions respond better to mannitol than do extra-axial lesions.
- Mannitol may withdraw water across the ependyma of the ventricles in a manner analogous to that produced by ventricular drainage.
- Mannitol may cause cerebral vasoconstriction, with the resultant changes in CBV being responsible for reduction in ICP.

Mannitol is usually given in a 20% solution in bolus doses as opposed to a continuous infusion. Boluses of 0.25 to 0.5 g/kg given over 10 to 20 minutes may be used and repeated depending on the patient response.

Mannitol's effect on ICP reaches its maximum 30 minutes after infusion and lasts for a variable period ranging from 90 minutes to 6 hours or more. The correct dose is the smallest dose that will have sufficient effect on ICP. When repeated doses are required, the baseline serum osmolality gradually increases. When this level exceeds 330 mOsm/L, mannitol therapy should cease; there is no role for dehydration. Further use is ineffective and is likely to induce renal failure.

Diuretics such as furosemide, given either alone or in conjunction with mannitol, help to hasten

mannitol's excretion and reduce the baseline serum osmolality prior to the next dose. It is speculated that furosemide complements mannitol and increases the output. Furosemide may be given before mannitol, so that it reduces circulatory overload.

Barbiturates can lower ICP when other measures fail but have no prophylactic value. Barbiturates and hypnotic drugs such as propofol and etomidate act by reducing $CMRO_2$ and producing a coupled reduction in CBF. (They also reduce brain metabolism and energy demand, which facilitates better healing.) The consequent reduction in CBV leads to a parallel fall in ICP. Among the barbiturates, phenobarbital is most widely used for this purpose. A loading dose of 10 mg/kg over 30 minutes and 1 to 3 mg/kg every hour is widely used. Close monitoring of ICP and hemodynamic instability should accompany any barbiturate therapy.

High-dose corticosteroid therapy (such as methylprednisone or dexamethasone) was popular some years ago as a means to lower ICP and is still used for this purpose today. Corticosteroids restore cell-wall integrity and help in recovery to reduce edema.

Decompressive craniotomies, such as subtemporal decompression, are not recommended. Herniation of the brain through a defect may cause further injury, further edema, and further increased ICP in some patients who undergo this procedure. In occasional cases, when every other measure has failed, a decompression craniotomy may be justified. This action would need to be substantiated by clear evidence of neurologic deterioration and a CT scan that shows an intracranial lesion.

Hyperbaric oxygen and hypothermia are experimental techniques (used especially frequently in Japan) to induce cerebral vasoconstriction and reduce the CBV and the ICP. Considerable evidence supports the use of mild to moderate hypothermia (32°C to 34°C) for increased ICP, especially when target temperatures are maintained for longer than 48 hours. Cooling reduces the cerebral metabolic rate, leading to a reduction in CBF, ICP, and glutamate release. Fever increases the cerebral metabolic rate (CMR) and $CMRO_2$ and occurs commonly in brain-injured patients, especially after intracranial hemorrhage. Therefore, temperature monitoring and control are vital to patient outcomes.

Spinal Cord Injuries

CCTPs may be called on to provide appropriate transportation of patients with spinal cord injuries (SCIs). These injuries may be highly suspicious as a result of the mechanism or identified at the transferring facility by radiographic imaging. These injuries may also be isolated or associated with other injuries and can complicate management priorities. Rapid and safe transportation of the patient with an SCI to a facility that can provide definitive care is optimum.

Mechanism of Injury

Acute injuries of the spine are classified according to the mechanism, location, and stability of the injury. Vertebral fractures often occur with or without associated SCI. Simply put, the spine is composed of three anatomic columns. First described by Denis, the spine is divided longitudinally into the anterior, middle, and posterior columns. The anterior column includes the anterior longitudinal ligament, the anterior half of the vertebral body, and the anterior half of the intravertebral disk. The middle column is composed of the posterior half of the vertebral body, the posterior half of the intravertebral disk, and the posterior longitudinal ligament. Finally, the posterior column consists of all other structures posterior to the posterior longitudinal ligament and includes pedicles, lamina, spinous process, interspinous ligaments, and the ligamentum flavum. Disruption of two of three of these columns is, by definition, an unstable injury.

Generally the types of forces or mechanisms that affect the spinal column include flexion/extension, vertical compression/longitudinal distraction, rotational, and combined mechanisms.

Flexion-Extension Injuries

Flexion-extension injuries are typically the result of rapid deceleration or from a direct blow to the occiput. They typically involve the cervical region; the spinal cord may be injured as a result by compression in the canal. Hyperextension of the head and neck can result in fractures, ligamentous injury, and SCI of variable stability. The C5-6 level is most susceptible to this injury from a mechanical standpoint.

Vertical Compression

Vertical compression forces are transmitted through vertebral bodies and directed either inferiorly through the skull or superiorly through the pelvis or feet. They typically result from a direct blow to the crown (parietal region) of the skull or rapid deceleration from a fall through the feet, legs, and pelvis. Compression forces can cause the herniation of discs, subsequent compression on the spinal cord and nerve roots, and fragmentation into the canal. Forces transmitted through the vertebral body cause fractures, ultimately shattering and producing a “burst” or compression fracture, sometimes without, but also with varying associated SCI. In falls, most injuries involve the thoracolumbar junction; the lower cervical spine is often involved with axial loads to the spinal column **Figure 11-47**.

Special Populations

Osteoporosis in older adult patients contributes to a high rate of spinal injury, often as a result of minimal trauma (eg, ground level falls or falls from a seated position). The CCTP should maintain a high index of suspicion of spinal injury in this population with special attention to possible C1–C2 injuries that include dens fractures and transverse ligament disruption.

Special Populations

Patients with a history of ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis are a special population that warrants extra care and attention. Because of these conditions, the patients are at increased risk of spinal fractures. Even nondisplaced fractures in this population can be unstable and lead to potential SCI.

Figure 11-47 A compression fracture.

Rotation With Flexion

Rotation-flexion injuries often occur in the thoracolumbar interface. The injury patterns can involve stable or unstable fractures and dislocations.

Primary Spinal Cord Injury

Primary spinal cord injury is a result of the initial trauma. Penetrating trauma may either result in transection of neural elements or cause concussive injury to the spinal cord (as in gunshot wounds), and may be either complete or incomplete injuries. Blunt trauma may displace ligaments and bone fragments, resulting in compression of points of the spinal cord or an incomplete dislocation of the vertebral body. Hypoperfusion and ischemia may also result from this type of injury to the spinal vasculature. Necrosis from prolonged ischemia leads to permanent loss of function.

Spinal cord contusions are caused by fracture, dislocation, or direct trauma. They are associated with edema, tissue damage, and vascular leakage. Hemorrhagic disruption may cause temporary to permanent loss of function despite normal radiographs.

Cord laceration usually occurs when a projectile or bone enters the spinal canal. Such an injury is likely to result in hemorrhage into the cord tissue, swelling, and disruption of some portion of the cord and its associated communication pathways.

Secondary Spinal Cord Injury

Secondary spinal cord injury occurs as a result of progression of the primary SCI. The injury can cause an ensuing cascade of inflammatory responses that may result in further deterioration. These effects can be exacerbated by exposing neural elements to further hypoperfusion, hypoxemia, hypoglycemia, and hypothermia. Although some SCIs may be unavoidable, the prehospital provider should minimize further injury through stabilization—that is, through spinal motion restriction and neutral alignment. In addition,

minimizing heat loss and maintaining oxygenation and perfusion are key elements in the care of a patient with a possible SCI.

SCIs are classified as complete or incomplete. **Complete spinal cord injury** involves complete disruption of all tracts of the spinal cord, with no sensory and motor function more than three segments below the level of injury. When the injury affects the patient in the cervical spine, quadriplegia can result. Injury in the thoracic spine can result in paraplegia. In an **incomplete spinal cord injury**, the patient retains some degree of cord-mediated function more than three segments below the level of injury. The degree of SCI is best determined 24 hours after the initial injury; the initial dysfunction may be temporary, and there is some potential for recovery.

Types of Incomplete Spinal Cord Syndromes

Anterior cord syndrome results from the displacement of bony fragments into the anterior portion of the spinal cord, often the result of flexion injuries or fractures. The anterior spinal artery provides blood to the anterior two thirds of the spinal cord; disruption of this flow will present as an anterior cord syndrome. Typical physical findings include motor paralysis below the level of the insult with loss of pain and temperature sensation with preservation of proprioception.

In **central cord syndrome**, hyperextension injuries to the cervical area present with hemorrhage or edema to the central cervical segments. This type of damage is rarely associated with fractures or bone disruption but more often occurs in conjunction with tears to the anterior longitudinal ligament. Central cord syndrome is frequently seen in older patients, who may already have a significant degree of cervical spondylosis and stenosis as the result of arthritic changes. A brief episode of hyperextension can exert pressure on the spinal cord within the relatively diminished spinal canal. Within the central cord, motor (efferent) fibers are distributed in a unique fashion, with more cervical and thoracic motor and sensory tracts than in the periphery of the cord. The patient with central cord syndrome will present with greater loss of function in the upper extremities than in the lower extremities, with variable loss of sensation to pain and temperature. The patient may also have some bowel and bladder dysfunction. The prognosis for central cord syndrome is typically good; many patients regain all motor function or have only some residual weakness in the hands.

Special Populations

Children who are younger than 10 years have heads that are proportionally larger than their body and, as a result, will frequently have spinal injuries from the occiput to C3. Children older than 10 years have injury patterns similar to adults. Because the head is proportionally larger in younger children, it is important to appropriately immobilize children in a neutral position. This may require padding under the shoulders and back to maintain a neutral alignment in relation to the head.

Brown-Sequard syndrome occurs when penetrating trauma is accompanied by hemisection of the cord and complete damage to all spinal tracts on the involved side. Injury to the corticospinal motor tracts causes motor loss on the same side as the injury, but below the lesion. Damage to the dorsal column causes loss of sensation to light touch, proprioception, and vibration on the same side as the injury (below it). Disruption of the spinothalamic tracts causes loss of sensation to pain and temperature on the opposite side of the injury, below the lesion.

Posterior cord syndrome is associated with extension injuries. This relatively rare syndrome produces dysfunction of the dorsal columns, presenting as decreased sensation to light touch,

proprioception, and vibration, while most other motor and sensory functions remain intact. Recovery of function is less likely than with central cord syndrome, but the overall prognosis remains good with therapy and rehabilitation.

Signs and Symptoms

Spinal Cord Injury

- May have flaccid paralysis below the level of injury
- May have loss of sensation to pain, temperature, and proprioception below the level of injury; loss may be unilateral
- May have disproportionate upper extremity motor impairment compared with lower extremities
- Bradycardia
- Hypotension
- Hypothermia
- Priaprism in males

Transport Management

Spinal Cord Injury

- Identify and correct life-threatening problems (ABCs).
- Maintain spinal stabilization and neutral alignment of the spinal column.
- In patients with multiple injuries, provide appropriate volume resuscitation with intravenous fluids and blood component therapy as appropriate.
- Rapidly identify and treat hypotension and hypoxia.
- Maintain normothermia.
- In patients an with an isolated SCI, support blood pressure after adequate resuscitation with vasoactive medications such as dopamine or phenylephrine.
- Patients may require atropine, transcutaneous pacing, or transvenous pacing for bradycardia.
- Provide rapid, safe transportation to a definitive care facility.

Controversies

Patients should be evaluated for decubitus ulcers, and any findings must be appropriately documented in the medical chart. Consideration during packaging should include appropriate padding of pressure points. Areas to pay particular attention to include the coccyx, shoulders, occiput, and heels.

Assessment of SCIs

A patient with an SCI should have a thorough assessment by the CCTP before transport. During this initial assessment, the CCTP should perform a comprehensive neurologic exam. This will provide a baseline to compare with future assessments when looking for acute changes. SCIs may be severe enough to threaten life and often leave the patient with significant disability. It is important to take the necessary precautions

and properly immobilize trauma patients when indicated. In most cases, the patient will be transported to the accepting facility with full spinal immobilization on the backboard until the receiving unit can properly identify the presence of a spinal injury.

The initial assessment of the patient with a suspected SCI follows the same assessment that is completed in a patient with a head injury. If not in place already, immediate attention to manual stabilization of the cervical spine is imperative, until a cervical collar can be placed. Because cervical collars only provide axial traction, they do not fully stabilize the head. To provide proper full immobilization for transport, the head must be immobilized using an appropriate immobilization device (eg, padded blocks) in addition to a cervical collar when using a backboard.

As part of the initial assessment prior to transport, the CCTP should take the time to perform a physical exam of the patient prior to packaging. For patients with SCI, it may be particularly important to focus on the following aspects of patient assessment. When appropriate, visually inspect the neck, looking for obvious deformities and feeling for crepitus or the presence of pain while maintaining inline stabilization. Evaluate the patient's airway prior to transportation. If the patient is not already intubated, perform a thorough and complete assessment of the airway. Consider the likelihood of respiratory failure and the need for definitive airway management during transportation. Patients with high SCI may have altered ability to breathe and ventilate during transport. They may also tire from their work of breathing as the result of muscular weakness from the SCI or from the inability to clear secretions. Managing an airway in a patient with cervical fractures or SCI can be difficult in the best of circumstances. If judged to be appropriate, intubate the patient at the referral facility under controlled circumstances and with appropriate support, if available, depending on the referral facility's resources (eg, anesthesia, surgeon, emergency medicine).

Assess the back for tenderness and deformity, if not previously done, and when appropriate. Palpate the spine for tenderness and deformity (such as step offs). Assess for strength in upper and lower extremities and the presence of sensation with comparison of both sides. Ongoing patient assessment, particularly of the patient's pulse, motor, and sensory function, is extremely important, because increased swelling can contribute to progressive deterioration of the patient's neurologic status. On arrival at the receiving facility, it will be important to provide an accurate and up-to-date patient report to the accepting medical staff.

Controversies

When spine clearance cannot be done clinically, most emergency medicine textbooks advocate clearing the spine with conventional x-rays. However, this practice is rapidly being replaced with CT scans of the head, neck, and chest. CT can be done more rapidly and with less patient movement and offers much clearer images than conventional films. However, there are reasons why a rapid CT scan cannot be performed by a transferring hospital while awaiting arrival of the CCTP team, such as when a CT scan would delay the transport. It is also prudent to limit CT exposure when possible to decrease a younger person's risk of cancer later in life; with younger patients radiographs may be a better choice, especially when suspicion for injury is low. It is also prudent to avoid exposing women of childbearing age to excess radiation, which can predispose their future children to birth anomalies. Additionally, removing the patient from a backboard helps alleviate discomfort and prevent pressure injuries from prolonged immobility on a hard surface.

The CCTP should always confirm whether or not clearance has been done, and obtain specific details from the transferring personnel. Appropriate documentation of this clearance should be evident in the medical record prior to transportation.

In general, patients with identified or highly suspected cervical or thoracolumbar injury should not be cleared and should be transported in the appropriate manner (immobilized). However, patients with stable lower spine fractures do not necessarily need to be transferred on a backboard. In most cases, patients with the presence of neurologic deficits despite the absence of identified radiologic abnormalities should be assumed to have injury and should be appropriately immobilized. Isolated cervical spine injury may be initially managed with an appropriately fitting collar in place. Thoracolumbar fractures should be managed with spine immobilization during transport. If in doubt, perform full spinal immobilization.

Patients should be transported with either the head of the bed at 30° or in **reverse Trendelenburg** to minimize the risk of aspiration in both ventilated and nonventilated patients. Communication with medical control and preestablished protocols on appropriate transportation methods will assist in providing the most appropriate transportation method.

Spinal shock is the temporary local neurologic condition that occurs immediately after spinal trauma and involves a spinal cord concussion. The patient may present with variable degrees of acute spinal injury, potentially with flaccid paralysis, flaccid sphincters, and absent reflexes. Sensory function below the level of injury will be impaired. Spinal shock usually subsides within 24 to 72 hours when the spinal cord mediated reflex arc below the level of injury resumes function. Patients who regain this arc, yet still have absence of distal motor or sensory function below the level of injury, have a complete cord injury and are unlikely to regain neurologic function. Those with some motor and sensory function below the level of injury are considered to have incomplete injury.

Neurogenic shock results from the temporary loss of autonomic function, which controls cardiovascular function, at the level of injury. Marked hemodynamic and systemic effects are seen and typically result in a triad of hypotension, bradycardia, and hypothermia. Hypotension occurs as the result of absent or impaired peripheral vascular tone with the loss of alpha-receptor stimulation; blood pools in the enlarged vascular space, causing relative hypovolemia and making the patient extremely sensitive to sudden position changes; and cardiac preload decreases, resulting in decreased stroke volume and cardiac output. Bradycardia results from the unopposed vagal response; the adrenal gland loses its sympathetic stimulation and does not produce epinephrine or norepinephrine to compensate for the loss of vascular tone. Hypothermia and absence of sweating are also seen because of the loss of sympathetic stimulation and thermoregulation. The classic presentation of neurogenic shock is a hypotensive, bradycardic patient whose skin is warm, flushed, and dry below the level of the spinal lesion.

Management of SCI

A patient with an SCI should be treated as a true neurologic emergency, and the CCTP should make every effort to rapidly assess, treat, and transport the patient to the closest appropriate facility while maintaining appropriate inline stabilization of the spinal column.

Patients with an SCI should be transported in a way that minimizes jostling and movement to prevent further SCI. Immediate identification and aggressive management of hypotension and hypoxia will also help to prevent further insult of the spinal cord.

Priorities of management include rapid identification and correction of life-threatening problems. The airway should be appropriately secured and managed, especially with higher-level cervical fractures. Breathing should be supported via mechanical ventilator or bag-mask assist for transportation of patients who are intubated or have tracheotomies. End-tidal capnography and pulse oximetry are helpful in assessing and monitoring respiratory status. Careful assessment, including pulse oximetry, of breathing in nonintubated patients is important when monitoring for progressive respiratory failure.

Frequent monitoring of blood pressure by noninvasive means or by an arterial line during transport will alert providers to hypotension. Appropriate large-bore IV access (minimum of two IV sites) or appropriate central venous access should be in place prior to transport, as should bladder catheterization.

Adequate volume resuscitation with appropriate IV fluids and blood component therapy for patients with multiple injuries should occur. Patients with multiple injuries should be suspected to be experiencing hemorrhage and hemorrhagic shock first and foremost unless these are ruled out at the transferring facility. Patients with identified isolated neurogenic shock should be treated with volume resuscitation and vasoactive medications. Patients with SCI may have a loss of thermoregulation from vasodilatation and require monitoring of temperature and treatment to maintain normothermia, including warm IV fluids, blankets, and temperature control in the transporting vehicle. Because bradycardia sometimes results from SCI, the use of atropine, transcutaneous pacers, and transvenous pacemakers may be warranted in high-level cervical lesions (C1-C5).

Pharmacotherapy in SCI

Patients with SCI may require various pharmacologic interventions during transport. Pain associated with fractures or other injuries will require judicious use of narcotics. IV opiates can be useful in the management of pain associated with transportation, movement, and the injury itself. Fentanyl, a short-acting synthetic opiate, has less of an effect on blood pressure and may be used as first-line therapy for these patients to control pain and to reduce the risk of hypotension. Midazolam (Versed) or lorazepam (Ativan) may be used for sedation after appropriate pain control has been established and other correctible causes of agitation such as hypoxia have been excluded. The risk of secondary injury as the result of movements from acute agitation must be balanced with potential airway and ventilatory compromise as well as a reliable neurologic exam.

The use of corticosteroids in the acute phase of SCI remains controversial. In the United States, the practice remains relatively widespread within the initial eight hours after injury. Dosing of IV corticosteroids, developed as a result of the National Acute Spinal Cord Injury Study II and III, calls for use of methylprednisolone in acute nonpenetrating SCI less than 8 hours after injury. The regimen includes a 30-mg/kg bolus of methylprednisolone over 15 minutes. After a 45-minute interval following infusion, a drip of 5.4 mg/kg/h continues over the next 23 hours. One of the principle side effects of corticosteroid administration is hyperglycemia, and patients who have either been given or are receiving a methylprednisolone drip should have point-of-care glucose monitoring and appropriate treatment of hyperglycemia with insulin.

Patients with SCIs may also have neurogenic shock requiring vasoactive medications to support blood pressure once adequate resuscitation has occurred. Agents such as dopamine and phenylephrine may be used to support blood pressure. Patients in neurogenic shock may also have bradycardia, requiring atropine on an urgent basis to support the heart rate and blood pressure.

Controversies

Recent outcomes data on use of corticosteroids immediately following initial injury and during interfacility transfer have failed to show significant improvements. As a consequence, many spine surgeons are beginning to abandon this practice.

Complications of SCI

The complications of SCI are a consistent cause of the high morbidity and mortality and the high financial

cost associated with this type of injury. Acute-phase complications of SCI include the potential for aspiration or respiratory arrest, especially with high cervical injuries. Lower cervical lesions may preserve the diaphragm, but the loss of intercostal muscles ultimately impairs coughing and deep breathing, predisposing the patient to atelectasis and pneumonia. Patients should be transported either in a reverse Trendelenburg position or with the head of the bed elevated to 30° as appropriate. Deep vein thrombosis and pulmonary embolism are late complications that may result from immobility and can become potentially life threatening. Consideration of sequential compression devices to the lower legs should be given for prolonged transports to reduce the risk of deep vein thrombosis subsequent to pulmonary embolism. Pressure ulcers from immobility are also problematic in SCI. Proper padding and judicious use of spinal immobilization boards are important during the transfer phase. Gastric stress ulceration can be reduced with the use of appropriate prophylaxis agents (eg, proton pump inhibitors and histamine 2 blockers). Urinary tract infections can be reduced by appropriately placing sterile Foley catheters, securing the device to the leg, and keeping the Foley bag in a dependent location to prevent backflow of urine into the bladder.

Autonomic dysreflexia is typically a late complication of SCI but may occur acutely. It commonly occurs with injuries above T6 and results from the loss of coordinated autonomic responses. This happens when there is an irritation, pain, or stimulus to the nervous system below the SCI. The affected area sends signals to the brain, but they are unable to reach the brain. The body responds with a reflex action constricting the blood vessels and causing an increase in blood pressure. If left uncontrolled, this can cause a stroke, seizures, and even death. Patients present clinically with evidence of a massive, uninhibited, uncompensated cardiovascular response as the result of noxious stimulation of the sympathetic nervous system below the level of injury. Unabated sympathetic nervous system stimulation results in vasoconstriction and hypertension (sometimes over 200 mm Hg systolic). This leads to compensatory parasympathetic stimulation and causes bradycardia and vasodilation above the level of the lesion, whereas vessels below the SCI remain constricted. This selective vasodilation results in flushed, diaphoretic skin and nasopharyngeal vessel congestion. Other symptoms include headache, nausea, and anxiety. Common precipitators include bladder distention, bowel impaction, bladder infection, pressure ulceration, fractures, and constrictive clothing. Indwelling catheters should be evaluated for obstruction or kinking and flushed to ensure patency. Irritating foci should be identified and treated appropriately, such as removing light clothing, resolving bowel/bladder problems, or treating a decubitus ulcer.

Signs and Symptoms

Autonomic Dysreflexia

- Hypertension
- Headache
- Nasal congestion
- Anxiety
- Nausea
- Blurred vision
- Bradycardia common, but tachycardia may also occur
- Diaphoresis and flushing above SCI
- Piloerection above SCI

Transport Management

Autonomic Dysreflexia

- Monitor blood pressure frequently.
- Search for, identify, and correct inciting stimuli (ie, reposition the patient and check the bladder and bowels for irritation or obstruction).
- If the source cannot be found or minimized, reduce the blood pressure with a beta-blocker or vasodilators.

If the source of autonomic dysreflexia cannot be found or minimized to an effective extent, it may be necessary to reduce blood pressure with vasodilators. Agents to consider are ones with a rapid action and a short duration (eg, nitrates, nitropaste, hydralizine, and labetolol). A trial of beta-blockers may also help to reduce cardiovascular response. Left untreated, the patient may experience seizures, intercranial hemorrhage, and even death.

Stroke

Mechanism of Injury

Stroke, also referred to as cerebrovascular accident, occurs when a disruption of blood flow to the brain results in neurologic deficit persisting for more than 24 hours. When deficits completely resolve within 24 hours, the event is referred to as a **transient ischemic attack (TIA)**. Perfusion to oxygen-sensitive brain tissue may be interrupted when a blood vessel is occluded or when the structural integrity of a blood vessel is compromised, allowing blood to escape into the brain tissue or into the subarachnoid space. Care must be initiated quickly to restore proper blood flow to reduce loss of normal neurologic function.

Types of Stroke

Ischemic strokes occur when cerebral blood flow is decreased, usually because of an occlusion of a blood vessel. The occlusion can be due to either a thrombus or an embolus. Hypoperfusion secondary to hypotension can also cause an ischemic stroke. Between 75% and 85% of all strokes are ischemic strokes.

Thrombotic strokes are largely the result of an accumulation of atherosclerotic plaques inside cerebral blood vessels, especially at bifurcations, which narrow the lumen of the vessel and make blood flow difficult. Rupture of a local plaque can activate localized clotting, which can significantly diminish or completely stop blood flow distal to the occlusion.

Embolic strokes occur when an embolus from the heart or lower circulation travels and lodges in a smaller cerebral vessel, resulting in loss of blood supply. Only 30% of ischemic strokes are thought to be caused by an embolus. Myocardial complications, such as atrial fibrillation, valvular heart disease, myocardial infarction, ventricular aneurysm, and cardiomyopathy, are known risk factors for embolus formation.

Focal ischemic strokes take place when an area of marginally perfused tissue, the ischemic penumbra, surrounds a core of ischemic cells. As a reduction of blood flow continues, ischemic cells become injured and finally die, resulting in infarction and irreversible loss of brain tissue that eventually softens and becomes necrotic. The size of the stroke depends on the size and location of the occluded

vessel and the availability of collateral blood flow.

Global ischemic strokes result when severe hypotension or cardiac arrest produces a transient drop in blood flow to all areas of the brain.

Stroke Side Effects

Cerebral edema sufficient to produce clinical deterioration occurs in 10% to 20% of patients with ischemic stroke and can result in intracranial hypertension. The edema is a result of a loss of normal metabolic function of cells and peaks at 3 to 5 days. Secondary hemorrhage at the site of an ischemic stroke lesion can also occur and is known as **hemorrhagic conversion**. Patients who have had a stroke may also exhibit seizure activity as a result of damaged neurons.

Assessment

The characteristic sign of stroke is the sudden onset of focal neurologic signs that oftentimes occur in combination. The CCTP can also use abbreviated prehospital tools to identify patients suffering from stroke, such as the Cincinnati Prehospital Stroke Scale **Table 11-25** or the Los Angeles Prehospital Stroke Screen (LAPSS) **Table 11-26**. The National Institutes of Health stroke scale is also available, but is very involved; although useful in the hospital setting, it is not practical for evaluation during transport. In addition to focal neurologic signs and stroke tools, noncontrast CT scanning is important to help determine whether the patient's symptoms are being caused by an occlusion or bleeding.

Management

Once stroke is suspected, the CCTP should determine the time of symptom onset. In general, the CCTP should support the patient's cardiopulmonary function, provide continuous monitoring of neurologic function, and maintain normal blood glucose levels. Stroke patients are at risk for respiratory compromise from aspiration, upper airway obstruction, and hypoventilation. Frequent reassessment of airway and respiratory function is necessary because hypoxemia will worsen ischemic brain injury, and contribute to a worse outcome. Stroke patients should be given supplemental oxygen.

TABLE 11-25 Cincinnati Prehospital Stroke Scale*

Assessment	Normal	Abnormal
Facial Droop. Ask the patient to smile and show the teeth.	Both sides of the face move equally well.	One side of the face does not move as well as the other.
Arm Drift. Ask the patient to close the eyes and hold the arms out with palms up for 10 seconds.	Both arms move the same, or both arms do not move.	One arm does not move, or one arm drifts down compared with the other.
Abnormal Speech. Ask the patient to say, "The sky is blue in Cincinnati" or "You can't teach an old dog new tricks."	The patient uses the correct words with no slurring.	The patient slurs words, uses inappropriate words, or is unable to speak.

*Interpretation: If any assessment criterion is abnormal, the probability of a stroke is 72%.

TABLE 11-26 Los Angeles Prehospital Stroke Screen*

1. Age > 45 y	❑	❑	❑
2. History of seizures or epilepsy absent	❑	❑	❑
3. Symptoms < 24 h	❑	❑	❑
4. At baseline, patient is not wheelchair bound or bedridden	❑	❑	❑
5. Blood glucose between 60 and 400 mg/dL	❑	❑	❑
6. Obvious asymmetry (right vs left) in any of the following three exam categories (must be unilateral):	❑	❑	❑
	Equal	**Right Weak**	**Left Weak**
Facial smile/grimace	❑	❑ Droop	❑ Droop
Grip	❑	❑ Weak grip ❑ No grip	❑ Weak grip ❑ No grip
Arm strength	❑	❑ Drifts down ❑ Falls rapidly	❑ Drifts down ❑ Falls rapidly
*Interpretation: If criteria 1 through 6 are marked yes, the probability of a stroke is 97%.			

Fibrinolytic Therapy

In some cases, the CCTP may provide transport of stroke patients who are receiving fibrinolytic therapy. If a patient's symptoms do not resolve, a CT scan shows no hemorrhage, and the symptom onset is within 3 hours, fibrinolytic therapy may be used to restore circulation to ischemic brain tissue. The only fibrinolytic agent currently used for the treatment of ischemic stroke is tPA. Prior to initiation of therapy, inclusion and exclusion criteria for IV fibrinolytic therapy should be reviewed (refer to the American Heart Association inclusion/exclusion criteria) in order to decrease the risk of complications associated with fibrinolytic therapy, including intracranial hemorrhage, orolingual angioedema, acute hypotension, and systemic bleeding. Exclusion criteria generally include these types of concerns:

- History of intracranial neoplasm, arteriovenous malformation, or aneurysm
- Bleeding concerns, either as the result of current bleeding or a bleeding disorder
- History of stroke
- Recent noncompressible vascular puncture
- Major surgery or trauma in the past 3 months
- Systolic blood pressure of greater than 180 mm Hg
- Pregnancy and up to 1 month postpartum

Intracerebral Hemorrhage

Intracerebral hemorrhage is bleeding directly into the cerebral tissue that causes cerebral tissue destruction, cerebral edema, and increased ICP. The most common cause of spontaneous intracerebral hemorrhage is hypertension-induced vessel rupture. Other causes include anticoagulation or fibrinolytic therapy, coagulation disorders, drug abuse, and hemorrhage into cerebral infarcts or brain tumors.

Symptoms of intracerebral hemorrhage may begin as local neurologic dysfunction related to the area of the brain in which a vessel has ruptured. The patient may also report the presence of a severe headache, nausea, and vomiting. As ICP continues to rise, however, the patient will oftentimes become unconscious (a key finding that helps differentiate from ischemic stroke) and may require ventilatory support. The time it takes for deterioration to progress is related to the amount and speed of bleeding.

The CCTP should provide adequate attention to the management of cardiopulmonary function. Intubation may be necessary, and should be provided as soon as the patient's level of consciousness becomes inadequate for airway control or vomiting begins to endanger airway patency. If intracerebral hemorrhage has been confirmed prior to transport, orders may be given to reduce blood pressure to decrease ongoing bleeding. An over-aggressive reduction in blood pressure, however, may compromise

cerebral perfusion pressure, especially in the patient with an elevated ICP.

Signs and Symptoms

Left Hemisphere Ischemic Stroke

- Right hemiparesis
- Right-sided sensory loss
- Right visual field deficit
- Poor right conjugate gaze
- Dysarthria
- Aphasia
- Difficulty in reading, writing, or calculating

Right Hemisphere Ischemic Stroke

- Left hemiparesis
- Left-sided sensory loss
- Left visual field deficit
- Poor left conjugate gaze
- Dysarthria
- **Extinction** of left-sided stimuli
- Neglect of the left visual space
- Spatial disorientation

Brain Stem/Cerebellum/Posterior Hemisphere Ischemic Stroke

- Motor or sensory loss in all four limbs
- Crossed signs
- Limb or gait ataxia
- Dysarthria
- Dysconjugate gaze
- Nystagmus
- Amnesia
- Bilateral visual field deficits

Small Subcortical Hemisphere or Brain Stem (Pure Motor) Ischemic Stroke

- Weakness of face and limbs on one side of the body without abnormalities of higher brain function, sensation, or vision

Small Subcortical Hemisphere or Brain Stem (Pure Sensory) Ischemic Stroke

- Decreased sensation of face and limbs on one side of the body without abnormalities of higher brain function, motor function, or vision

Adapted from: Adams HP, Brott TA, Crowell RM, et al. Guidelines for the management of patients with acute ischemic stroke: a statement for healthcare professionals from a special writing group of the

Stroke Council, American Heart Association. *Circulation*. 1994;90(3):1588. Available at: http://www.strokecenter.org/education/ais_evaluation/brain_stem.htm. Accessed March 9, 2009.

Transport Management

Stroke

- Determine the time of symptom onset.
- Support the patient's cardiopulmonary function.
- Provide continuous monitoring of neurologic function.
- Maintain normal blood glucose levels.
- Frequently reassess airway and respiratory function.
- Give supplemental oxygen.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage is bleeding into the subarachnoid space, and is usually arterial in nature. Most subarachnoid hemorrhages are caused by rupture of a cerebral aneurysm or arteriovenous malformation (AVM). Other causes include hypertensive intracerebral hemorrhages that progress to subarachnoid hemorrhage and bleeding from a cerebral tumor. This chapter focuses on the two most frequent causes of subarachnoid hemorrhage.

Aneurysms are an outpouching of the wall of a blood vessel that results from weakening of the wall of the vessel. Most aneurysms are congenital, whereas others can be attributed to trauma or infection, or are of an idiopathic nature. As an individual with a congenital cerebral aneurysm matures, blood pressure rises and places stress on the wall of the weakened vessel. Eventually, ballooning of the vessel occurs, which may lead to compression of adjacent brain tissue and focal neurologic dysfunction. If the aneurysm ruptures, arterial blood is sent into the subarachnoid space at high pressure. As blood fills the space, ICP rises and cerebral perfusion pressure falls.

Arteriovenous malformations (AVMs) are tangled masses of arterial and venous blood vessels that shunt blood directly from thickly walled arteries to thinly walled veins without the benefit of pressure being reduced by an intervening capillary bed. One or more cerebral arteries, known as feeders, provide blood to an AVM. These feeders enlarge over time, increase the volume of blood shunted through the malformation, and increase the overall mass effect on adjacent brain tissue. Eventually, veins may no longer be able to accommodate the high mean pressure, and therefore rupture, causing blood to fill the subarachnoid space. As this happens, ICP rises and cerebral perfusion pressure falls.

Signs and Symptoms

Subarachnoid Hemorrhage

- Onset of pain
- Worst headache of patient's life
- Loss of consciousness
- Nausea
- Vomiting

- Focal neurologic deficits
- Photophobia
- Nuchal rigidity

Transport Management

Subarachnoid Hemorrhage

- Take measures to maintain the patient's cardiopulmonary function.
- Manage the patient's airway.
- Provide ventilatory support if necessary.
- Limit movement of the patient's head; elevate the head during transport.

Assessment

The patient with a subarachnoid hemorrhage characteristically has an abrupt onset of pain, oftentimes described as "the worst headache I have ever had." A loss of consciousness, nausea, vomiting, focal neurologic deficits, photophobia, and nuchal rigidity may accompany the headache. As ICP increases, the patient may become comatose or die.

Management

Management of the patient with a subarachnoid hemorrhage requires that attention be given to maintaining cardiopulmonary function. Airway management and ventilatory support are frequently needed as the patient's level of consciousness and vomiting endanger the airway. The CCTP may also take steps to prevent an unnecessary rise in ICP, such as limiting head movement and elevating the head during transport. Common complications of subarachnoid hemorrhage include rebleeding, which is an occurrence of a second subarachnoid hemorrhage in an unsecured aneurysm, cerebral vasospasm, hyponatremia, and hydrocephalus.

Laboratory Assessment

Laboratory studies, other than routine blood studies, biopsy, and CSF analysis, have not traditionally been used in the diagnosis and management of neurologic emergencies. For most patients, a complete blood count (CBC), basic chemistry panel, coagulation study, and cardiac biomarkers should be obtained.

The CBC serves as a baseline study and may reveal a cause of stroke such as polycythemia or thrombocytosis or provide evidence of a concurrent illness such as anemia. The chemistry panel also serves as a baseline study and may reveal the origin of coma such as hypoglycemia or provide evidence of concurrent illness such as renal insufficiency. Coagulation studies may reveal coagulopathy and are useful when fibrinolytics or anticoagulants are considered for use. Cardiac biomarkers are important because of the association of cerebral vascular disease and coronary artery disease. Finally, a toxicology screening may be useful in selected patients in whom an altered level of consciousness, other abnormal neurologic finding, or vital sign anomalies, such as tachycardia or hypertension, are thought to be caused by a harmful drug or chemical. Chapter 20 discusses toxicologic emergencies.

Axonal Transport

The intracellular transport of protein and organelle materials is essential for all mammalian cells, especially the neuron. Most neuron cell bodies have dendrites on one end and an axon on the opposite

end. Because most neuronal proteins are synthesized in the cell body, mechanisms that direct transport along the axon or along the dendrite are required. Additionally, the distance over which materials have to be moved in the nervous system far exceeds the distances of other cell types because a human motor neuron can exceed a length of 1 m. Finally, even within an individual axon, materials being transported must be targeted to specific compartments.

The feature of neurons that allows the delivery of cellular components to the appropriate site of action is a long-range microtubule-based transport system, which is composed of two key components: the molecular motors that drive the sending of cellular components and microtubules that direct cellular components to the correct site. The type of molecular motor present determines the direction of transport —either away from the cell body and toward the synapse or toward the cell body.

Axonal transport is commonly divided into fast and slow axonal transport based on the bulk speeds of cargo movement. Cellular components such as vesicles and mitochondria move by fast axonal transport speeds, whereas cytoskeleton components move by slow axonal transport speeds.

When transport of materials becomes disrupted, disease can result. Axonal and cell body accumulations of organelles and other proteins are hallmark pathologies for many human neurodegenerative diseases such as Alzheimer's and related diseases, Parkinson's disease, and amyotrophic lateral sclerosis.

Local Anesthetics

Local anesthetics are medications that cause reversible local anesthesia and loss of nociception. They can also be used on specific nerve pathways (nerve block) to produce analgesia and paralysis. Local anesthetics belong to one of two drug classes: aminoamide and aminoester local anesthetics, the difference being in the chemical linkage (amide-linkage or ester-linkage) between the lipophilic part and the intermediate chain of the molecule. Local anesthetics act mainly by inhibiting sodium influx through sodium-specific ion channels in the neuronal cell membrane. When this occurs, an action potential cannot be generated and signal conduction is inhibited.

Seizures and Epilepsy

When a nerve cell is stimulated, its action is transmitted to other cells by excitatory neurotransmitters and balanced by inhibitory neurotransmitters that slow communications. Overactivity by excitatory transmitters or underactivity by inhibitory transmitters can result in a seizure.

Seizure activity can be caused by different factors, such as fever, glucose imbalance, electrolyte imbalances, head injuries and hemorrhagic strokes, and toxins. Epilepsy is a recurrent seizure disorder caused by abnormal discharges in the brain cells. It is often idiopathic. Petite mal seizures should be monitored, but grand mal and focal seizures that may compromise the airway require immediate action.

Management of seizure patients should initially center on maintaining an airway, protecting the patient, and providing oxygen. If the patient is actively seizing, no attempt should be made to restrain him or her. An IV should be established, and the patient's cardiac status should be monitored. Also, the patient's glucose level should be checked, and glucose should be administered if indicated. The patient's temperature should be checked to ensure that fever is not an issue. Body temperature can be lowered by running normal saline, giving acetaminophen (Tylenol), and cooling down without causing shivering. If the patient is actively seizing, an anticonvulsant must be administered. The most common drugs administered during transports are lorazepam (Ativan), diazepam (Valium), and midazolam (Versed). If the patient is seizing and IV access is not possible, rectal diazepam (Diastat) can be administered.

Transport Management

Seizures

- Manage the patient's airway.
- Provide oxygen.
- Do not attempt to restrain the patient if the patient is actively seizing.
- Establish an IV.
- Monitor the patient's cardiac status. Check the patient's blood glucose level and administer glucose if indicated.
- Check the patient's temperature.
- To lower the patient's body temperature, run normal saline, give acetaminophen (Tylenol), and cool down the patient without causing shivering.
- If the patient is actively seizing, administer an anticonvulsant (eg, lorazepam [Ativan], diazepam [Valium], or midazolam [Versed]).
- If the patient is seizing and IV access is not possible, give intranasal, intramuscular, or rectal diazepam (Diastat).
- If the patient has epilepsy, give phenytoin (Dilantin), fosphenytoin sodium (Cerebryx), or, alternatively, carbamazine (Tegretol) or phenobarbital.
- If the patient has a focal seizure, administer etomidate as an induction agent to relax the muscles and allow tracheal access if intubation is necessary.

Patients with a history of epilepsy are most commonly taking phenytoin (Dilantin), which can be given orally or intravenously. If phenytoin is administered IV, it must be given slowly. The maximum rate for phenytoin IV is 50 mg/min (for a 500 mg load, this is 10 minutes; for a 1,000 mg load, this is 20 minutes). If phenytoin is given as a rapid push, it will create severe bradycardia and hypotension. An alternative is fosphenytoin sodium (Cerebryx), which can be given intravenously or intramuscularly. Other first-generation anticonvulsants that may be given are carbamazine (Tegretol) and phenobarbital.

A focal seizure that leaves the patient rigid from the neck up but flaccid from the neck down may compromise the airway in an unconscious patient. The rigidity makes endotracheal intubation impossible, but administering etomidate as an induction agent relaxes the muscles and allows tracheal access.

Transport Considerations

Prior to Transport

Before considering transport, it is imperative that potential complications be anticipated. To this end, it would be advisable to ensure that all diagnostic tests and surgical procedures are completed when at all possible prior to transport.

Although patient-related risk factors are difficult to identify, equipment-related complications (which occur in up to one third of transports) may be controlled more easily. During critical care transport, the following types of equipment may be present:

1. Cardiac monitor, to monitor cardiac status.
2. Ventilator with waveform capnography, to monitor and control ventilation.
3. Infusion pumps, to administer medications.

4. ICP monitoring, to measure and control ICP.
5. Cerebral function monitor, to assess sedation, monitor brain wave activity, predict and monitor seizure activity, and monitor the onset and effectiveness of paralytics.

Complications with equipment would relate to the specific equipment used. For the previously mentioned devices, the following measures can be taken if a malfunction occurs:

1. **Cardiac monitor:** Check for sufficient batteries, charging, general check, and waveform capnography.
2. **Ventilator:** Check for sufficient oxygen and gas supply, ventilator tubing, oxygen and circuit connections, and capnography hookup.
3. **Infusion pumps:** These are usually specific to manufacturers. See the manufacturer's troubleshooting chart.
4. **CFM:** These are usually specific to manufacturers. See the manufacturer's troubleshooting chart.
5. **ICP monitor:** Check for sterility, keep the fluid level, and refer to the manufacturer's troubleshooting manual.

Most interfacility transports do not involve ICP monitoring or CFM monitoring. When ICP or CFM monitoring is utilized, the patients are usually managed with a ventilator, cardiac monitor, and medications administered by IV pump.

Scene and Interhospital Transport Considerations

Apart from intracranial hemorrhage, the major early risks to the patient with head injury are hypoxia and hypertension. The ABCs are always the first priority. The patient with a severe closed head injury should be presumed to have an elevated ICP, and factors that may further increase ICP should be avoided. Some critical care transport systems advocate the use of ear and eye protection for the patient to prevent spikes in ICP as a result of outside stimulus from aircraft or sirens (mostly applicable in seizure patients). Urgent attention to the ABCs will help reduce cerebral hypoxia and hypoperfusion. Normoxia and normocarbia should be achieved as soon as practically possible. ICP can be increased by overperfusion, hypertension, inadequate sedation, hyperpyrexia, and inappropriate anesthetic agents. Intubation and endotracheal suction cause sharp, albeit transitory, spikes in ICP. In patients with reduced volume reserve (hypovolemic), however, these stimuli may initiate a prolonged increase in ICP. Pressure waves may be avoided by adequate fluid resuscitation to maintain CPP. It is important to consider administering sedation at a level that is sufficient to prevent coughing or gagging during transport.

Minimum requirements for monitoring patients during transport are continuous ECG, pulse oximetry, and the intermittent measurement of blood pressure, respiratory rate, and pulse rate. In specific patients, capnometry, continuous blood pressure reading, and further monitoring (such as of ICP and cardiac output and filling pressures) may be beneficial. Unfortunately, many of the complications reported during transport are actually caused by equipment malfunction. It is imperative that the CCTP be familiar with all equipment and monitoring devices in use to ensure optimal patient outcomes and avoid complications from equipment misuse or malfunction.

Of particular importance is the possibility of measuring the major ventilation parameters, such as tidal volume or minute ventilation. Although many transport ventilators do not display actual tidal volumes delivered, judicious use of $ETCO_2$ monitoring allows the CCTP to observe the effects of mechanical ventilation on the patient during transport, and make appropriate adjustments. One possibility to reduce inadvertent ventilation problems may be the use of improved monitoring equipment, particularly equipment that focuses on tidal or minute ventilation. Bearing in mind the limitations of many portable

ventilators, the use of sophisticated transport carts equipped with a standard ICU ventilator and the necessary gas supply should be considered. Such carts can be hooked to the patient bed and moved fairly easily. Monitoring devices and infusion pumps can be implemented into the cart with its battery. Such equipment is being increasingly used during interhospital transport.

In some cases, the hazards of transporting a patient could be prevented by performing diagnostic or therapeutic procedures within the ICU or choosing alternative (albeit equivalently effective) procedures that may render a transport of the patient unnecessary. Such interventions may include the following options:

- Use of chest ultrasound in detecting intrathoracic pathologies
- Introduction of mobile CT scanners that can be used in the ICU
- Application of conventional or dilatational percutaneous tracheostomy in the ICU, instead of transferring the patient to the operating room, for the placement of percutaneous endoscopic gastrostomy tubes and of inferior vena cava filters
- Use of fiber optic pressure monitoring instead of surgical ventriculostomy
- Scheduled reoperations for peritonitis with open abdomen in the ICU

Adverse effects during and after transport of critically ill patients occur all too frequently. Nevertheless, a change in patient management results from about half of the procedures that necessitate transport, indicating good efficiency. Although a few patient-related risk factors can be identified, the rate of equipment-related adverse events may be as high as one third of all transports. Thus, particular attention has to be focused on the personnel, equipment, and monitoring in use.

Table 11-27 lists specific neurologic disorders and conditions, and their treatment during transport.

Flight Considerations

Most fixed-wing aircraft used for medical transport have the ability to pressurize the cabin as the altitude increases and to depressurize the cabin as the altitude decreases. These aircraft have a chart indicating cabin pressure as a function of altitude. It is desirable to come as close to the original ground pressure as possible. If possible, have the pilot fly at altitudes where cabin pressure can be maintained as close to the original departure pressure as possible.

If this is impossible in flight, adjustments must be made. If the patient is intubated, the endotracheal cuff pressure should be decreased with altitude to prevent damage to the trachea and reinflated during descent to prevent leakage. The pressure inside indwelling catheters and colostomy bags must be adjusted to prevent breakage. If the patient has a drain and the ICP is monitored, the ICP must be maintained to less than 20 cm H_2O by draining off excess fluids. If the patient does not have a drain, then the patient must be monitored for any changes in status. If the patient shows changes in respiratory rate or pattern, or develops any changes in cardiac function (ie, arrhythmias or significant changes in heart rate), these signs may indicate herniation and the aircraft must immediately descend to an altitude at which the condition is manageable.

Summary

Management of neurologic emergencies requires an in-depth understanding of brain, spinal, and cranial nerve anatomy and physiology. In addition, the CCTP must be familiar with the complexity of severe TBI, the early recognition of signs and symptoms of TBI, and the associated impact early intervention has on the outcome of these patients.

The CCTP should make every effort to preserve cerebral perfusion and oxygenation as the primary goal in the initial management of neurologic patients. Current guidelines call for careful monitoring of oxygenation and preventing hypoxia by keeping Pa_{O_2} above 60 mm Hg and/or oxygen saturation greater than 90% at all times. Patients who, during field management or transport, exhibit signs and symptoms of cerebral herniation should be initially managed with hyperventilation. Mannitol may be helpful in treating ICP and cerebral herniation, but should be used judiciously given its other physiologic effects. The use of analgesia, sedation, and neuromuscular blockade is critical during transport to minimize the risks associated with the increase in ICP and to maximize safety in the transport setting. Hypoglycemia is a condition of which the CCTP should be mindful when treating neurologic patients, because it may mimic TBI or stroke, and should be considered in all patients with an altered LOC until ruled out by applicable diagnostic and laboratory confirmations.

The treatment of neurologic emergencies is also based on the ability to distinguish between primary and secondary injuries. A large part of managing secondary injuries centers on managing the effects of the injury—namely, CPP and ICP.

A CCTP who is involved in the transport of neurologic patients should be mindful of the complexities. If possible, the majority of diagnostics and/or treatments should be performed prior to transport. Although a number of patient-related risk factors can be identified in any transport involving neurologic transport, the rate of equipment-related adverse events in transport is substantially higher with these types of injuries. This requires careful attention to the personnel, equipment, and monitoring in use at any given time.

TABLE 11-27 Specific Neurologic Disorders and Conditions

Disease	Description	Symptoms and Key Issues	Treatment During Transport
Neuritis	General inflammation of the peripheral nervous system	Can cause pain, paresthesia, paresis, numbness, anesthesia, paralysis, wasting, and disappearance of reflexes	Supportive care and management of pain
Poliomyelitis	Viral disease that can destroy motor neurons (<1% of cases) or cause no symptoms at all (90% of cases)	Transmitted primarily by fecal-oral route; post-polio syndrome can affect polio survivors years after initial infection	Support respiratory effort and body substance isolation
Parkinson's disease	Degenerative disorder of the nervous system normally caused by insufficient formation and action of dopamine	Can cause muscle rigidity, tremor, a slowing of physical movement (bradykinesia) and, in extreme cases, a loss of physical movement (akinesia)	Supportive care
Multiple sclerosis	Autoimmune condition in which the immune system attacks the central nervous system and results in demyelination of neurons	Can cause hypoesthesias and paraesthesias, muscle weakness, muscle spasms, difficulty moving, ataxia, dysarthria, dysphagia, visual problems, fatigue, and chronic pain	Supportive care
Meningitis	Inflammation of the meninges caused by an infection of CSF by bacteria, viruses, fungi, or parasites; non-infectious causes include cancer, SLE, and certain drugs	Can cause headache, nuchal rigidity, photophobia, positive Kernig's and/or Brudzinski's signs, seizures, petechial rash	Supportive care, body substance isolation, and possible monitoring of antimicrobial agent
Encephalitis	Acute inflammation of the brain	Most commonly a viral infection but can be bacterial or parasitic; causes fever, headache, photophobia, weakness, seizure	Supportive care; antiviral agents are not effective (except for herpes origin); and possible monitoring of antimicrobial agent
Bacterial infection	Bacterial infection of brain tissue or CSF	Can lead to encephalitis or meningitis	Possible monitoring of antimicrobial agent
CNS tumors	Intracranial or intravertebral tumors	May be either primary (originating from within the CNS) or secondary (orginating from other organs)	Supportive care
Alzheimer's disease	Most common form of dementia; incurable, degenerative, and terminal disease; characterized by loss of neurons and gross atrophy	Staged according to clinical manifestations -Predementia -Early dementia -Moderate dementia -Advanced dementia	Supportive care

Amyotrophic lateral sclerosis	Progressive, usually fatal, neurodegenerative disease caused by degeneration of motor neurons	Causes muscle weakness and atrophy as motor neurons degenerate	Supportive care; may require ventilatory support
Guillain-Barre syndrome	An acute inflammatory autoimmune disease that results in demyelination	Marked by ascending paralysis	Supportive care; may require ventilatory support; treatment with immunoglobulins or plasmapheresis
Myasthenia gravis	Autoimmune disorder in which antibodies attack acetylcholine receptors at the post-synaptic neuromuscular junction	Causes fluctuating muscle weakness and fatiguability	Supportive care; treatment with cholinesterase inhibitors or immunosuppressants
Neuroleptic malignant syndrome	Rare, but life-threatening idiosyncratic reaction to a neuroleptic medication	All antipsychotic agents, typical or atypical, may be causes; Causes fever, muscle rigidity, altered mental status, and autonomic dysfunction	Supportive care; cooling measures; benzodiazepines may be used
Malignant hypertension syndrome	Acute or ongoing vital target organ damage in the setting of severe hypertension	Hypertension, severe headache, altered mental status, confusion, seizures, nausea/vomiting	Supportive care; anti-hypertensive agent that does not cause CNS effects
Creutzfeldt-Jakob disease	Rare, rapidly progressing, degenerative brain disorder that leads to spongy degeneration of the brain and dementia	Thought to be caused by prions Causes personality changes, anxiety, depression, memory loss, impaired thinking, impaired muscle coordination, blurred vision, insomnia, speech impairment	Supportive care
Bovine spongiform encephalopathy	Transmissible, neurodegenerative, fatal brain disease in cattle	Causes spongy degeneration of brain with severe and fatal neurologic signs and symptoms	No treatment exists
Neurotoxic poisoning	Exposure to a natural or artificial toxic substance that affects the normal activity of the nervous system in such a way as to cause damage to nervous tissue	Can be caused by exposure to chemotherapeutic agents, illicit drugs, heavy metals, and pesticides	Supportive care; specific treatment depends on nature of exposure

Abbreviations: CNS, central nervous system; CSF, cerebrospinal fluid; SLE, systemic lopus erythematosus.

Case Study

YOU AND YOUR CRITICAL CARE TRANSPORT TEAM ARE REQUESTED at the three-bed emergency department of a small community hospital to transfer a 60-year-old woman who was brought in moments ago by local EMS complaining of a severe headache.

As part of your history taking, you interview the patient's husband who is present at her bedside. He reports that his wife had just buried her mother yesterday and has been under a lot of stress dealing with her mother's affairs. He states that she has been complaining of a headache for the past few days, but it has become worse, increasing in severity.

When he arrived home after work, he found her lying on the couch. He states that he dimmed the lights, put a cool washcloth on her forehead, and gave her two aspirins. He went into the kitchen to start preparing dinner when he heard her vomiting. Upon entering the room, he saw that his wife was projectile vomiting and called 9-1-1. On your interview with the patient, she confirms what her husband reported and adds that she has been having blurred vision today as well. She states that she attributed the headache and nausea to stress over her mother's death. While you are obtaining information on her past medical conditions, she reports that she has a history of high blood pressure and has not been taking her medication for the past week, trying to save on money to pay for her mother's funeral. She denies having

any other medical problems or taking any other medications.

Your initial physical exam of the patient reveals a normal GCS score of 15. She is able to follow complex commands and move all extremities. She has no decreased level of consciousness and has equal strength bilaterally in all extremities, and all of her cranial nerves are intact. Her blood pressure is 192/110 mm Hg, consistent with the emergency department measurements. She is on a cardiac monitor, which reveals a heart rate of 95 beats/min, with normal sinus rhythm. Her respiratory rate is 18 breaths/min, and her lungs are clear to auscultation in all lung fields. Oral temperature is 99.6°F. She rates her headache a 10 on a scale of 1 to 10, with 10 being the worst pain in her life.

The patient is prepared for transport. The emergency department staff has placed the patient on 2 L of oxygen via nasal cannula. An IV has been placed in the patient's right antecubital vein, infusing normal saline at a keep-the-vein-open rate. She is placed on the stretcher with the head of the bed elevated at 45°. You and your team load the patient into your critical care ambulance and initiate seizure precautions during the transport. She is transported to the referral hospital.

1. What is going on with this patient?
2. Why is the head of the bed elevated at a 45° angle?
3. Why are seizure precautions initiated and what does the seizure precaution entail on the ambulance?
4. What are some possible complications that could occur during the transport of this patient and how would the critical care transport team handle them?

On arrival to the emergency department of the referral center, the patient has a grand mal seizure. An emergency CT scan of her head was ordered, which revealed a small, right subarachnoid hemorrhage with bleeding into the suprasellar cistern, indicating a grade 2 subarachnoid hemorrhage. (A grading scale for the classification of cerebral aneurysm was developed in 1968 by Drs Hunt and Hess. The grading scale ranges from grade 1, an asymptomatic or minor headache, to grade 5, a deep coma.) Neurosurgery was consulted and a cerebral angiogram was ordered. The cerebral angiogram revealed a 4-mm, berry-shaped, right middle cerebral aneurysm located at a bifurcation.

The patient was admitted to the neurosurgery intensive care unit and was scheduled for surgery the next morning. A clipping of the right middle cerebral artery was performed without complication. An intraventricular catheter was placed to monitor the patient's ICP. The surgeon's postoperative order stated to maintain a systolic blood pressure of 140 mm Hg or lower, to perform hourly neurologic exams, and to maintain an ICP of less than 15 mm Hg and a CPP of greater than 80 mm Hg. The patient remained on the ventilator until the following morning when a postoperative CT scan was performed. The scan revealed no complications and no recurrence of bleeding. She was extubated that day and was discharged home 6 days later without further complications.

5. What are some of the common reasons for an individual to develop a cerebral aneurysm?
6. List the three major postoperative complications that may occur following a cerebral aneurysm.

Analysis

The patient has a cerebral aneurysm. Elevating the head of the bed will help reduce increased ICP.

Seizure precautions include the following: Avoiding the use of lights and sirens; keeping lights dimmed in the compartment; padding side rails of the patient's stretcher; verifying that airway equipment is readily available, such as oxygen delivery systems, nasal and oral airways, Ambu-bag and mask, suction, catheters, and suction equipment; and being prepared to administer diazepam or lorazepam.

Possible complications in this patient include increased ICP, hypertension, respiratory depression and failure, and brain herniation. Hypertension should not be treated during transfer unless directed by the

receiving facility or neurosurgeon. The reason for this hypertension state is to provide increased cerebral perfusion to any potentially vasospastic areas. In general, patients should be maintained normotensive with a systolic blood pressure of 110 to 160 mm Hg. Keeping the head of the bed elevated and avoiding overstimulation of the patient will help in the reduction of ICP.

Patients with respiratory depression and failure may require rapid-sequence intubation. Patients with a potential increased ICP should be premedicated with a 1 mg/kg IV push of lidocaine and a defasciculating dose of vecuronium at 0.01 mg/kg IV push at least 2 minutes prior to intubation (doses may vary based on local protocol). (Defasciculating doses of nondepolarizing neuromuscular blockers consist of roughly one tenth the full intubating dose and provide enough blockade to prevent muscle fasciculations.) Lidocaine will blunt sympathetic responses to intubation and limit the associated increase in ICP, but it does not sedate the patient; it is cruel to paralyze and intubate an awake patient. A sedative should always be used unless the patient is comatose. Both lidocaine and etomidate can be used together, if necessary. Vecuronium, unlike succinylcholine, does not cause fasciculation, which is the involuntary contraction or twitching of muscle fibers that increases ICP. Etomidate may be used as an alternate induction agent. It is a hypnotic and will not cause an increase in ICP, but it is contraindicated in patients younger than 10 years. If etomidate is used, lidocaine will not be required.

Levels of $PaCO_2$ should be maintained between 36 and 42 mm Hg. Hyperventilation should be avoided unless the patient shows signs of herniation.

The causes of cerebral aneurysms include neoplastic disease, atherosclerosis, brain trauma, infection, and congenital defects. Cerebral aneurysms are usually surgically repaired by blocking the aneurysm off from the circulation with a clip, a procedure called clip ligation.

Potential postoperative complications of a cerebral aneurysm include recurrence of bleeding (especially in the first 24 hours), cerebral vasospasm, and electrolyte disturbances, such as hypernatremia. Vasospasm decreases blood flow to the brain and causes the death of nerve cells, so medication may be administered to prevent it. The patient may be given medications to address other potential complications as well.

Prep Kit

Ready for Review

- A large majority of neurologic patients who require transport will have traumatic brain injury (TBI).
- The nervous system can be divided into the CNS, which consists of the brain and spinal cord, and the PNS, which consists of the spinal and cranial nerves.
- Afferent pathways (ascending pathways) carry sensory impulses toward the CNS; efferent pathways (descending pathways) carry motor impulses away from the CNS to effector organs, such as muscles (smooth and skeletal) or glands.
- The voluntary (somatic) nervous system is composed of nervous system fibers that connect the structures of the CNS with skeletal muscles and the integument; the involuntary (autonomic) nervous system is divided into the sympathetic and parasympathetic branches.
- The brain is a very delicate substance that requires the protection afforded by the skull. Although the skull is primarily protective, excessive forces can cause a fracture to the adult skull and force bone fragments into the vulnerable brain tissue.
- Openings at the base of the skull (foramina) allow cranial nerves and blood vessels to enter and exit the cranial cavity. These openings also weaken the area, leaving it susceptible to fracture.

- The cranial meninges consist of three layers: the dura mater (a two-layer structure adherent to the internal surface of the cranium), the arachnoid mater (a thin layer loosely enclosing the brain), and the pia mater (a vascular membrane that adheres to the surface of the brain and follows its contours).
- The lateral ventricles extend from the frontal lobe to the occipital lobe of the brain. They are important structures for ICP monitoring, CSF drainage, or placement of a CSF shunt.
- CSF fills the ventricular system and surrounds the brain and spinal cord in the subarachnoid space. It protects the CNS by acting as a shock absorber for minor acceleration and deceleration and helping to remove waste products from cerebral tissue.
- Any obstruction to the normal flow of CSF may produce high CSF pressure and lead to brain damage.
- The cerebrum consists of two cerebral hemispheres, which are themselves divided into four paired lobes: the frontal, parietal, temporal, and occipital lobes.
- The brain stem acts as a bridge between the cerebral hemispheres and the spinal cord; all motor and sensory fibers travel through this structure. The brain stem consists of three parts: the midbrain, the pons, and the medulla oblongata.
- The cerebellum, which coordinates voluntary movement, consists of three major parts: the cortex, the white matter, and four pairs of deep cerebellar nuclei.
- The reticular formation, which is composed of both motor and sensory tracts, includes centers for blood pressure, respiration, and heart rate function.
- The overall goal of cerebral circulation is to provide enough blood to supply oxygen, glucose, and nutrients so that oxidation of glucose can take place.
- The blood-brain barrier regulates the transport of nutrients, ions, water, drugs, and waste products to and from the brain through the process of selective permeability.
- The spine usually consists of 33 irregular bones (vertebrae) articulating to form the vertebral column; these skeletal components are stabilized by both ligaments and muscle.
- Flexion of the vertebral column beyond the range of 60% to 70% may damage structural ligaments and allow excess vertebral movement that could expose the spinal cord to injury.
- The spinal cord transmits nerve impulses between the brain and the rest of the body. The 31 pairs of spinal nerves emerge from each side of the spinal cord and are named for the vertebral region and level from which they arise.
- The sympathetic nervous system is controlled by the brain's hypothalamus. A spinal cord injury at or above the level of T6 may disrupt the flow of sympathetic communication.
- When the sympathetic nerves are stimulated and produce autonomic dysreflexia, the parasympathetic nerves attempt to control the rapidly increasing blood pressure by slowing the heart rate.
- The foundation of neurologic care is a thorough serial assessment, which not only allows the tracking of patient trends, but also may allow the CCTP to identify which parts of the system are damaged.
- The neurologic field diagnosis of the impaired patient is divided into four main segments: mental status, cranial nerves, motor and cerebellar, and sensory.
- The history focuses on the patient's chief complaint. If taking a history from the critically ill patient is not possible, the CCTP must obtain information from secondary sources—other EMS reports, emergency department charts, medical records from previous admissions, and family and friends.
- The mental/emotional component of the neurologic examination assesses the patient's ability to understand and interact with the environment by addressing the level of consciousness; general

behavior; and thought process, including memory, attention and concentration, abstract thought, and judgment.

- Awareness and arousal are the fundamental constituents of consciousness and should be evaluated and documented repeatedly.
- Arousal is assessed initially by the AVPU scale. The GCS is then commonly used to document and trend a patient's level of arousal over time.
- In evaluating the patient's awareness, the CCTP should assess the patient's orientation to time, place, and person.
- A variety of neurologic pathologies—including pain, hypoxia, anxiety, and expanding intracranial lesions—may initially manifest themselves through changes in general behavior.
- The Mini-Mental State Examination is a simple, easily applied test of higher cognitive functions that consists of a series of questions testing orientation, registration, attention and calculation, recall, and language.
- Abnormalities of speech can reflect dysfunction in any component or process of communication. For example, aphasia encompasses any loss or impairment of language function as a result of brain damage.
- In some cases, the CCTP may perform assessment of cranial nerves, motor function, and sensory function; reflex testing; and evaluation of meningeal irritation if there was not much time for initial assessment or if there is time during transport.
- Certain nerves are more commonly tested in patients who are critically ill or injured: optic (CN II), oculomotor (CN III), trochlear (CN IV), trigeminal (CN V), abducens (CN VI), facial (CN VII), acoustic (CN VIII), glossopharyngeal (CN IX), vagus (CN X), and hypoglossal (CN XII).
- Cranial nerve I, which is responsible for olfaction, should be assessed in cases in which head trauma has occurred, when pathology at the base of the skull is suspected, and in patients who exhibit an altered mental status.
- Each eye should be evaluated individually to assess the optic nerve. The tests should include evaluation of visual acuity, visual fields, and the fundi.
- The two parameters that should be closely observed by the CCTP in relation to the oculomotor, trochlear, and abducens nerves are the position of the eyeball and the position of the upper eyelid.
- The motor portion of the facial nerve is tested by instructing the patient to wrinkle the forehead, to close the eyelids tightly, to smile or grimace showing the teeth, and to whistle.
- The vestibular division of the acoustic nerve is assessed using rotational and caloric stimuli; this evaluation is performed to determine the brain stem function of unconscious patients.
- To test the glossopharyngeal and vagus nerves, the CCTP should direct the patient to open his or her mouth and say, "Ah."
- To assess the hypoglossal nerve, the CCTP observes the tongue for fasciculations and atrophy, tests for muscle strength by having the patient push the tongue against the inside wall of the cheek, and checks for tongue protrusion.
- The motor examination includes a consideration of muscle tone and strength, during which the CCTP puts the muscles through the normal range of motion.
- As part of the assessment of sensory function, pain, temperature, and touch should be examined and documented.

- The most common pathological reflexes, all of which indicate pyramidal tract disease, include the plantar response, Babinski's sign (the most important), Oppenheim's sign, Gordon's sign, and Hoffmann's sign.
- Signs of meningeal irritation include generalized throbbing headache progressing in severity, progressive photophobia, and nuchal rigidity.
- The centers for control of vital functions are found within the brain stem, so changes in vital signs can signal changes in the patient's overall neurologic status.
- The CCTP should repeatedly check the following physiological variables: blood pressure, heart rate and rhythm, respiratory rate, pulse oximetry, end-tidal carbon dioxide, central venous pressure, temperature, and ICP.
- When assessing the critical care patient's respirations, pay particular attention to the rate, rhythm, and characteristics of the inspiratory and expiratory phases.
- The patient's heart rate may indicate not only probable neurologic injury, but also the presence of other pathologic processes. The CCTP should be especially mindful of the presence of tachycardia, bradycardia, or arrhythmias.
- Hypertension is the effect on blood pressure most often seen as a result of neurologic injury, especially in case of increased ICP.
- Alterations in body temperature—hypothermia or hyperthermia—are possible in the presence of neurologic injury, especially if the hypothalamus is involved.
- Two types of TBI are distinguished: primary (direct) injury, in which traumatic forces cause physical or functional disruption of brain tissue; and secondary (indirect) processes, which occur after injury and lead to brain dysfunction or cellular damage (eg, hypoxia and ischemia).
- Primary brain injury can occur as a result of two mechanisms: contact phenomena injuries (direct trauma to the head) and acceleration-deceleration injuries (sudden changes in velocity).
- Secondary injury involves more delayed mechanisms of brain damage and, if left uncorrected, will adversely affect brain function integrity.
- Cerebral perfusion pressure (CPP) is the primary determinant of blood flow to injured brain tissue when an injury has impaired the ability of the cerebral circulation to autoregulate in response to fluctuations in systemic blood pressure.
- Common injuries to the scalp include abrasions, contusions, lacerations, and avulsions. Treatment usually focuses on controlling bleeding, cleaning and dressing the area, and, in case of avulsions, evaluating for fractures.
- Skull fractures occur as a result of a direct, and often highforce, blow to the cranium; they are present in more than 50% of patients with a severe TBI.
- Fractures of the bones of the face may indicate possible underlying brain injury.
- To perform the halo test for leaking CSF, collect escaping fluid from the nose, mouth, or ears onto a gauze pad. The appearance of a "target" or "halo" symbol consisting of a dark red circle surrounded by a lighter yellowish one is considered a positive halo sign.
- The development of raccoon eyes (periorbital ecchymosis) and Battle's sign (retroauricular ecchymosis) may take several hours to develop post-injury.
- Many facial injuries have very grotesque presentations accompanied by copious bleeding and may potentially distract the CCTP from other, more serious injuries.

- Mandible fractures can cause life-threatening airway compromise if the patient is in the supine position.
- Airway control is the first assessment priority with facial injuries. After the airway has been secured, the CCTP can assess for the presence of facial fractures by grasping the hard palate and rocking it back and forth. The CCTP should also check for malocclusion and dental trauma, test extraocular muscles to rule out orbital fractures, and look for facial elongation and periorbital ecchymosis.
- Injuries to the brain can be either focal (lesions large enough to be observed directly) or diffuse (eg, concussions and diffuse axonal injuries).
- Types of focal injuries include contusion (bruising of the brain tissue), hematoma (epidural, subdural, or intracerebral), swelling, infarct, pressure necrosis, and abscess.
- Types of diffuse injuries include axonal injury, hypoxia/ischemia, diffuse vascular, fat embolism, subarachnoid, and meningitis.
- Treatment for patients with cerebral contusions and lacerations is largely supportive and includes monitoring for—and aggressively treating—evidence of rising ICP as a result of hematoma formation.
- An epidural hematoma (EDH) is an injury resulting in the accumulation of blood between the inner periosteum and the dura mater. The patient with an EDH may present with a history of a brief period of unconsciousness, followed by a lucid period lasting for minutes to several hours, which ends with a rapid deterioration of consciousness from drowsiness, to lethargy, and to coma as a mass effect and herniation develop.
- Mannitol is an osmotic diuretic that can reduce the increased ICP resulting from an increase in CSF and reduce intraocular pressure. It does not reduce the volume of blood accumulated by the bleed in the case of an EDH.
- High-energy impacts may result in a subdural hematoma (SDH)—bleeding that accumulates between the dura mater and arachnoid mater. These injuries are six times more common than EDHs and have a higher mortality rate.
- Intracerebral hemorrhages often occur in the same areas as cerebral contusions. They produce headaches, deteriorating levels of consciousness, hemiplegia on the contralateral side, and dilated ipsilateral pupils.
- Patients with a subarachnoid hemorrhage (SAH) often describe their pain as “the worst headache in my life” and are likely to have hypoxia, hypotension, and higher ICP. Signs and symptoms of SAH include altered mental status, coma, paralysis, slurred speech, mood changes, and seizures.
- Signs and symptoms of concussion (mild TBI) include loss of consciousness, loss of memory for events immediately before or the event that precipitated the injury, altered mental status, and focal neurologic deficits.
- The clinical presentation of diffuse axonal injury includes a sequence of events beginning with immediate unconsciousness, leading to a longer period of confusion and associated posttraumatic amnesia, and followed by a protracted recovery time. Treatment is supportive.
- In adults, the normal ICP is 0 to 15 mm Hg. Increased ICP may be precipitated by intracranial bleeding, cerebral edema, mass formation, or inability to regulate the production and removal of CSF.
- Many physiologic factors may alter cerebral blood flow (CBF). Increases in CBF as a result of hypoxia or hypercapnia (increased levels of CO_2 in blood), for example, will cause an increase in ICP once the normal compensating mechanisms have been exhausted.
- Poor airway and ventilatory control contribute to hypoxia, hypercapnia, and hypotension, and will

further damage the already critically ill brain.

- CPP is responsible for the movement of blood through the brain. The homeostatic mechanisms that manage CPP are often lost after head trauma, such that the patient with TBI may have increased cerebral vascular resistance, making the brain more susceptible to changes in blood pressure.
- CPP should be maintained between 50 and 70 mm Hg—by using fluids and pressors if necessary—to reduce the risk of poor outcomes for patients with increased ICP.
- The CCTP should perform serial neurologic examinations to monitor for increased ICP, especially for patients with TBI.
- The CCTP may also be required to maintain invasive neurologic monitoring devices; it is the responsibility of each CCTP to become familiar with all aspects of each device prior to using it on a patient.
- Increased ICP is a sustained elevation in pressure above 20 mm Hg. The most common causes of increased ICP are cerebral bleeding, tumors, hydrocephalus, and trauma.
- Cushing's triad is the combination of progressively increasing systolic blood pressure with progressively decreasing diastolic blood pressure (widening pulse pressure) and bradycardia.
- Respiratory changes noted with increased ICP include Cheyne-Stokes respirations (with midbrain involvement), sustained hyperventilation (with midbrain and pons involvement), and rapid and shallow respirations followed by ataxic breathing (with upper medulla involvement).
- Increased ICP produces pulmonary edema as a result of increased sympathetic activity as a result of the effects of the raised ICP on the hypothalamus, medulla, or cervical spinal cord.
- Brain herniation is a condition in which a portion of the brain is displaced because of increased ICP, resulting in progressive damage to brain tissue that may include life-threatening damage to the brain stem. Transport management includes intubation, mechanical ventilation, administration of an osmotic diuretic (eg, mannitol), possibly corticosteroids, and rapid transport.
- The primary goals of ICP monitoring are identification of ICP trends and evaluation of therapeutic interventions to minimize ischemia in the brain-injured patient.
- Contraindications to ICP monitoring include CNS infection, coagulation defects, anticoagulant therapy, scalp infection, severe midline shift resulting in ventricular displacement, and cerebral edema resulting in ventricular collapse.
- Noninvasive ICP monitoring involves assessment for clinical deterioration in neurologic status, evidenced by signs such as bradycardia, increased blood pressure, and pupillary dilation.
- Intraventricular monitoring is one of the most popular techniques for invasive ICP monitoring. It is also therapeutic, because it provides the ability to drain CSF; any fluid drained must be monitored for amount, color, and clarity at hourly intervals.
- A subarachnoid screw ("bolt") may be used to monitor ICP, although its accuracy is less than that of the more direct ventriculostomy drain.
- Patients with TBI should be intubated to protect the airway and allow maximal oxygenation. Standard monitoring for all such patients is required, including oxygen saturation, ECG, mean arterial pressure, and urine output.
- Normocapnia (normal carbon dioxide tension) is vital for maintenance of ICP, and patients should have continuous measurement of $ETCO_2$ levels using a capnometer.
- Potential complications of ICP monitoring include intracranial infection, intracerebral hemorrhage, air

leakage into the ventricle or subarachnoid space, CSF leakage, overdrainage of CSF (leading to ventricular collapse and herniation), occlusion of the catheter with brain tissue or blood, and inappropriate therapy because of erroneous ICP readings.

- Brain tissue oxygen tension monitoring is accomplished by placement of a commercial probe (capable of measuring temperature and oxygenation) into the brain tissue through a bolt. This type of monitoring is relatively sensitive for monitoring focal disruption of blood flow to particular areas of the brain.
- To minimize the risk that an infectious agent will enter the CNS during ICP monitoring, aseptic technique must be used at all times when assembling, manipulating, or accessing the fluid-filled monitoring system.
- To ensure optimal accuracy, proper leveling and zeroing of the ICP monitoring system must be maintained.
- Avoid flexion and hyperextension of the neck and positioning the patient in a Trendelenburg position when conducting ICP monitoring. Simultaneous drainage and pressure monitoring are not recommended.
- Notify the physician immediately if the patient shows signs of decompensation—altered LOC, restlessness, agitation, lethargy, confusion, motor weakness, seizures, alteration in breathing pattern, increases in blood pressure, bradycardia, vomiting, decortication/decerebration, or coma.
- The best treatment for increased ICP is the removal of the causative lesion (eg, tumor, hydrocephalus, or hematoma). In the field, treatment is aimed at preventing the secondary events associated with increased ICP; it focuses on reducing ICP and providing rapid transport to a neurologic facility for definitive treatment.
- First-line management consists of general measures aimed at making the neurologic patient comfortable (including adequate analgesia), along with effective handling of the ABCs of trauma management.
- Second-line management of increased ICP involves induced cerebral vasoconstriction, which may take the form of hyperventilation, hyperbaric oxygen, or hypothermia. Osmotherapy consisting of mannitol, glycerol, urea, or furosemide may also be implemented.
- Anesthetic agents, including barbiturates, gamma-hydroxy-butyrate, and etomidate, and paralytic agents may also be used in the event of increased ICP.
- Hyperventilation aims at keeping the P_{CO_2} between 25 and 30 mm Hg, so that CBF falls and CBV is reduced, thereby reducing ICP. Prophylactic hyperventilation is not recommended.
- Acute injuries of the spine are classified according to the associated mechanism, location, and stability of the injury.
- Stable vertebral fractures do not involve the posterior column; therefore, they pose less risk to the spinal cord. Unstable injuries involve the posterior column of the spinal cord and carry a higher risk of complicating SCI and progression of injury without appropriate treatment.
- Flexion injuries result from forward movement of the head, typically as the result of rapid deceleration (eg, in a car crash), or from a direct blow to the occiput. Rotationflexion injuries, which often result from high acceleration forces, can produce a stable dislocation in the cervical spine. Hyperextension of the head and neck can result in fractures and ligamentous injury of variable stability.
- Vertical compression injuries typically result from a direct blow to the crown (parietal region) of the skull or rapid deceleration from a fall through the feet, legs, and pelvis. Forces transmitted through the vertebral body cause fractures, ultimately shattering and producing a “burst” or compression fracture with or without associated SCI; patients may also develop herniation of disks, subsequent compression

on the spinal cord and nerve roots, and fragmentation into the canal.

- Primary spinal cord injury—injury that occurs at the moment of impact—can involve either penetrating or blunt trauma. Penetrating trauma typically results in transection of nonregenerative neural elements and complete injuries; blunt trauma may displace ligaments and bone fragments, resulting in compression of points of the spinal cord or an incomplete dislocation of the vertebral body. Spinal cord concussion, spinal cord contusion, and cord laceration are all types of primary spinal cord injury.
- Secondary spinal cord injury occurs when multiple factors permit a progression of the primary SCI and result in further deterioration. These effects can be exacerbated by exposing neural elements to further hypoxemia, hypoglycemia, and hypothermia.
- The CCTP should minimize the chance of secondary SCI through stabilization (ie, spinal motion restriction and neutral alignment). Minimizing heat loss and maintaining oxygenation and perfusion are other key elements in the care of a patient with a possible SCI.
- Because many SCIs can be so severe that they threaten life and leave the patient with a lifelong disability, CCTPs must always take the necessary precautions and properly immobilize trauma patients as indicated.
- Immediate attention to manual stabilization of the cervical spine is imperative, until a cervical collar and backboard can be placed. In most cases, the patient will be transported to the accepting facility in a supine position on the backboard.
- In conjunction with manual stabilization of the cervical spine, evaluation of the patient's airway must be done. While maintaining in-line stabilization of the spine, any blood or secretions present in the airway must be suctioned, and the preparation for a definitive airway must be done.
- The management of a patient with SCI should be treated as a true neurologic emergency, and the CCTP should make every effort to rapidly assess, treat, and transport the patient to the closest appropriate facility while maintaining in-line stabilization of the spinal column.
- Short-acting, reversible sedatives are recommended for the acute agitated patient after a correctible cause of agitation (eg, hypoxia) has been excluded.
- Acute-phase complications of SCI include the potential for aspiration or respiratory arrest, especially with high cervical injuries.
- Autonomic dysreflexia is evidenced by hypertension, headache, blurred vision, anxiety, cool clammy skin, and sweating above the injured site. Treatment consists of identifying and treating the cause (ie, repositioning the patient and checking the bladder and bowels for irritation or obstructions) and reducing the blood pressure with a beta-blocker.
- Stroke occurs as a result of disruption of blood flow to the brain that results in a neurologic deficit persisting for more than 24 hours. When deficits completely resolve within 24 hours, the event is referred to as a transient ischemic attack.
- Types of stroke include ischemic (caused by occlusion of a blood vessel by a thrombus or embolus), thrombotic (caused by accumulation of atherosclerotic plaques inside cerebral blood vessels that block blood flow), embolic (occurring when an embolus lodges in a smaller cerebral vessel), focal ischemic (caused by ongoing reduction of blood flow to already ischemic cells), and global ischemic (caused by severe hypotension or cardiac arrest that produces a transient drop in blood flow to all areas of the brain).
- The characteristic sign of stroke is the sudden onset of focal neurologic signs that oftentimes occur in combination.

- The CCTP can use abbreviated prehospital tools to identify patients suffering from stroke, such as the Cincinnati Prehospital Stroke Scale or the Los Angeles Prehospital Stroke Screen (LAPSS).
- Once stroke is suspected, the CCTP should determine the time of symptom onset.
- The CCTP should support the stroke patient's cardiopulmonary function, provide continuous monitoring of neurologic function, and maintain normal blood glucose levels.
- Frequent reassessment of airway and respiratory function is needed to avoid hypoxemia in stroke patients; supplemental oxygen may be given to hypoxemic patients.
- If a stroke patient's symptoms do not resolve, a CT scan shows no hemorrhage, and the symptom onset is within 3 hours, fibrinolytic therapy (tPA) may be used to restore circulation to ischemic brain tissue.
- Intracerebral hemorrhage is bleeding directly into the cerebral tissue. The most common cause of spontaneous intracerebral hemorrhage is hypertension-induced vessel rupture. Symptoms include local neurologic dysfunction, severe headache, nausea, and vomiting. As the ICP continues to rise, the patient may lose consciousness and require ventilatory support. Intubation may be necessary, and orders may be given to reduce blood pressure so as to decrease ongoing bleeding.
- The patient with a subarachnoid hemorrhage (typically caused by either an aneurysm or an arteriovenous malformation) characteristically has an abrupt onset of pain, sometimes accompanied by loss of consciousness, nausea, vomiting, focal neurologic deficits, photophobia, and nuchal rigidity. As the ICP increases, the patient may become comatose or die.
- Management of the patient with a subarachnoid hemorrhage requires measures to maintain cardiopulmonary function, manage the airway, provide ventilatory support (if necessary), and limit movement of the patient's head.
- Seizure activity can be caused by fever, glucose imbalance, electrolyte imbalances, head injuries and hemorrhagic strokes, and toxins.
- Epilepsy is a recurrent seizure disorder caused by abnormal discharges in the brain cells.
- Management of seizure patients focuses on the ABCs and administration of an anticonvulsant, if the patient is actively seizing.
- Minimum requirements for monitoring patients during transport are continuous ECG, pulse oximetry, and the intermittent measurement of blood pressure, respiratory rate, and pulse rate.
- Other pieces of equipment that should be available—and working properly—to monitor the neurologic patient during transport include a cardiac monitor, a ventilator with waveform capnography, infusion pumps, an ICP monitor, and a cerebral function monitor.
- Apart from intracranial hemorrhage, the major early risks to the patient with head injury are hypoxia and hypertension. For this reason, management of the ABCs is always the first priority.
- The biggest challenge when transporting patients by aircraft is dealing with pressure variations and oxygen availability at increasing altitudes. Oxygen content in pressurized cabin air is less than that at normal ground level, so be prepared to administer supplemental oxygen to nonintubated patients.

Vital Vocabulary

afferent pathways Ascending pathways that carry sensory impulses toward the central nervous system.

aneurysm A weakened portion of the wall of an artery where the blood creates a localized dilation or bulge; can involve the wall intact or be classified as dissecting, in which the artery wall ruptures and blood pools between the inner and outer artery wall.

anterior cord syndrome Displacement of bony fragments into the anterior portion of the spinal cord, often as the result of flexion injuries or fractures, that disrupts blood flow in the anterior spinal artery.

aphasia Any loss or impairment of language function as a result of brain damage.

arachnoid mater The middle layer of the meninges, which contains blood vessels that give it the appearance of a spider web.

arteriovenous malformation (AVM) Abnormally formed blood vessels that have a higher rate of bleeding than average vessels.

autonomic dysreflexia A potentially life-threatening complication of spinal cord injury that results from the loss of parasympathetic stimulation. It is characterized by a massive, uninhibited, uncompensated cardiovascular response as the result of some stimulation of the sympathetic nervous system below the level of injury. Also called autonomic hyperreflexia.

axonal transport The movement of organelles and proteins along a nerve cell axon into and out of the cell body.

basal ganglia Masses of nuclei located deep in the cerebral hemispheres that play a major role in fine motor function.

basilar skull fracture A fracture along the base of the skull.

Battle's sign Migration of blood to the mastoid region, posterior and slightly inferior to the ear, resulting in discoloration; also called retroauricular ecchymosis.

blood–brain barrier A network of endothelial cells and astrocytes (neuroglia) in the brain that regulate the transport of nutrients, ions, water, drugs, and waste products to and from the brain through the process of selective permeability.

brain herniation Displacement of a portion of the brain from its correct location within the cranial cavity to a different location.

brain tissue Compliance The change in brain volume resulting from a change in pressure.

brain tissue oxygen tension ($P_{br}o_2$) A method of monitoring temperature and oxygenation via placement of a commercial probe into the brain tissue through a bolt; can be done simultaneously with ICP monitoring placement.

Broca's area Part of the frontal lobe that is located at the inferior frontal gyrus and that participates in the formulation of words.

Brown-Sequard syndrome Loss of function as a result of penetrating trauma accompanied by hemisection of the spinal cord and complete damage to all spinal tracts on the involved side; characterized by loss of motor function and sensation of light touch, proprioception, and vibration on the ipsilateral side and temperature and pain sense on the contralateral side.

caloric test A method for assessing vestibular function that involves the raising and lowering of the temperature in the external auditory canal; also called Bárány test.

cauda equina The collection of individual nerve roots into which the spinal cord separates at the L2 vertebra.

central cord syndrome A syndrome in which cavities form in the central portions of the spinal cord, usually in the cervical area; may be due to a tumor, genetic origin, or trauma; presents along with hemorrhage or edema to the central cervical segments.

cerebral angiography A procedure that uses imaging and a contrast material or dye to view the blood

vessels in the brain and is used to find abnormalities.

cerebral aqueduct The narrowest portion of the brain's ventricular system; it provides communication with the fourth ventricle, which lies between the brain stem and the cerebellum.

cerebral blood flow (CBF) The amount of blood flow the brain requires to maintain homeostasis. In a 24-hour period, the brain requires 1,000 L of blood to obtain 71 L of oxygen and 100 g of glucose.

cerebral cortex The outermost layer of the cerebrum.

cerebral function analysis monitor (CFAM) A device that can provide summed, averaged, and analyzed outputs of the general state of brain activity.

cerebral function monitor (CFM) A device that can provide summed and averaged outputs, but not analysis, of the general state of brain activity.

cerebral hemispheres The name for each half of the brain (right or left), each of which contains one of the paired lobes (occipital, parietal, temporal, and frontal).

cerebral metabolic rate for oxygen ($CMRO_2$) A measurement used to determine neuronal demand for oxygen. Neurons with high activity rates require greater amounts of oxygen.

cerebral perfusion pressure (CPP) The pressure gradient across the brain; it provides an estimate of perfusion adequacy: CPP = MAP – ICP.

choroid plexus A cluster of nerve roots at the lateral and the third and fourth ventricles of the brain that produce cerebrospinal fluid.

circle of Willis A system of arteries located at the base of the skull that (in most people) is able to compensate for reduced blood flow from any one of the major contributors to cerebral circulation.

comminuted fracture A type of skull fracture in which the skull is splintered or shattered into many pieces.

complete spinal cord injury A complete disruption of all tracts of the spinal cord, with permanent loss of all cord-mediated functions below the level of transaction.

computed tomography (CT) A type of scan that provides a mathematically reconstructed view of multiple cross-sections of the body, including the brain; performed on almost every patient with abnormal neurologic findings.

conjugate movement Movement of both eyes together in the same direction.

contact phenomena injuries Injuries that occur as the direct result of trauma to the head, including local effects such as scalp laceration, skull fracture, hematoma, and intracerebral hemorrhage.

contrecoup injury A situation in which an impact occurs on one side of the head, causing the brain to move within the cranial vault and forcibly contact the opposite side of the skull, resulting in damage on that side of the brain; also called transitional injury.

corpus callosum A large tract of transverse fibers that provides a communication link between the two cerebral hemispheres.

cranial nerves (CN) The 12 nerves arising directly from the brain that govern many of the senses and the functions of muscles in the eyes, face, and pharynx.

Cushing's triad A cascade of events provoked when intracranial pressure rises to the level of the arterial pressure, vasoconstriction occurs in an effort to shift fluid volumes in the cranium, and the ensuing brain displacement puts pressure on the medulla oblongata by pushing it into the foramen magnum, resulting in disturbances in breathing, heart rate, and blood pressure.

delayed traumatic intracranial hemorrhage (DTICH) Hemorrhage that occurs within the first 3 to 10 days following an injury to the occipital-parietal region via a coup-contrecoup mechanism.

depressed skull fracture A type of skull fracture in which a portion of the skull is depressed; the scalp and/or dura may or may not be torn.

diaphragma sellae An extension of the dura mater that forms a roof over the sella turica, which contains the pituitary gland.

diastatic stellate fracture A fracture involving injury to a bone with separation of an epiphysis; prevalent in abused children.

diencephalon Portion of the cerebrum consisting of the thalamus, the hypothalamus, the subthalamus, and the epithalamus.

diffuse axonal injury (DAI) A deep-brain injury in which shearing forces damage the integrity of the axon at the node of Ranvier, which consequently alters the axoplasmic flow.

diplopia Double vision.

doll's eye test An oculocephalic reflex test that is performed on the unconscious patient by rapidly rotating the head from side to side and observing the eye movement.

dorsal Toward the back surface of an object; posterior.

dural venous sinuses Endothelial-lined spaces between the periosteal and meningeal layers of the dura mater.

dura mater The outer membrane of the meninges.

dysconjugate movement Lack of symmetric movement between the two visual axes.

Edinger-Westphal nucleus Part of the midbrain that is responsible for mediating the autonomic reflex centers for pupillary accommodation to light.

efferent pathways Descending pathways that carry motor impulses away from the central nervous system.

electroencephalography (EEG) A procedure that records the electrical activity of the brain by measuring brain waves.

embolic stroke Occurs when a blood clot, known as an embolus, forms in one part of the body and travels through the bloodstream to the brain or neck.

epidural hematoma (EDH) An injury resulting in the accumulation of blood between the inner periosteum and the dura mater; also called extradural hematoma.

epithalamus An area of the cerebrum that is located in the dorsal portion of the diencephalon and contains the pineal gland.

extinction A test of sensation discrimination in which the CCTP simultaneously touches opposite, corresponding areas of the body, and asks the patient where the touch is felt; it is intended to identify sensory inattention.

extradural space A potential space between the cranial bones and the periosteal layer of the dura that becomes a real space only when blood from torn vessels pushes the periosteum from the cranium and accumulates; also called epidural space.

falx cerebelli A fold of dura mater that forms the division between the two lateral lobes of the cerebellum.

falx cerebri A double fold of dura mater that divides the cerebrum into right and left hemispheres by descending vertically into the longitudinal fissure that extends from the frontal lobe to the occipital lobe.

Fick principle A method of indirectly determining cardiac output, in which the amount of oxygen uptake of blood as it passes through the lungs is equal to the oxygen concentration difference between mixed venous and arterial blood.

fissures Deep grooves between adjacent gyri of the brain.

flat fracture The least common type of depressed skull fracture, in which the depressed segment does not have any connection with the cranial vault.

flexion-extension injury A spinal cord injury that results from forward movement of the head, typically as the result of rapid deceleration, or from a direct blow to the occiput.

focal ischemic stroke Occurs when an area of marginally perfused tissue, the ischemic penumbra, surrounds a core of ischemic cells, and the cells will eventually die without medical intervention.

foramen of Luschka An opening at the base of the fourth ventricle that leads to the subarachnoid space and is essential for the normal flow of cerebrospinal fluid; part of the brain's ventricular system.

foramen of Magendie An opening at the base of the fourth ventricle that leads to the subarachnoid space and is essential for the normal flow of cerebrospinal fluid; part of the brain's ventricular system.

foramen magnum The opening at the base of the skull through which the bundle of nerve fibers constituting the spinal cord exits.

foramen of Monro An opening in the skull that connects the two lateral ventricles with the third ventricle, a central cavity; part of the brain's ventricular system.

Foster–Kennedy syndrome A tumor or abscess at the base of the frontal lobe that impacts the olfactory nerve.

frontal lobe The largest of the four lobes of the brain; it lies underneath the frontal bone of the skull and is separated posteriorly from the parietal lobe by the central fissure and inferiorly from the temporal lobe by the lateral fissure; responsible for a variety of cognitive and motor functions.

fundus The optic disk, macula, and blood vessels on the back wall of the internal eyeball.

global ischemic stroke Results when severe hypotension or cardiac arrest produces a transient drop in blood flow to all areas of the brain.

gyri Convolutions on the surface of the cerebrum that functionally increase the cortical surface area.

halo test A test for leaking CSF that is accomplished by collecting and assessing fluid that drains from the nose, mouth, or ears; a "halo" consisting of a dark red circle surrounded by a lighter yellowish one is a positive halo sign.

hemorrhagic conversion After the brain tissue surrounding the stroke has died, renewed blood flow to the region, for example as triggered by medication, is no longer held in place by the tissue, resulting in hemorrhage.

hypothalamus An area of the cerebrum located below the thalamus that forms the floor and the anterior walls of the third ventricle. It is responsible for the maintenance of homeostasis and the implementation of behavioral patterns.

incomplete spinal cord injury A disruption of the tracts of the spinal cord in which the patient retains some degree of cordmediated function.

internal capsule A massive bundle of efferent and afferent fibers connecting the various subdivisions of the brain and spinal cord.

intervertebral foramen A space in the middle of the vertebra that allows the exit of a peripheral nerve root and spinal vein as well as the entrance of a spinal artery on both sides at each vertebral junction.

intracranial pressure (ICP) The pressure exerted by brain tissue, intracranial vascular contents, and cerebrospinal fluid in the closed, nondistensible cranial cavity.

intracranial temperature (ICT) Core brain temperature or homeostatic mean gradient temperature of 38.4°C.

intracerebral hemorrhage Direct bleeding into the brain parenchyma.

involuntary (autonomic) nervous system The sympathetic and parasympathetic branches of the nervous system, whose fibers connect the structures of the CNS with smooth muscle, cardiac muscle, and glands.

ischemic stroke Occurs when an artery to the brain has been blocked by a thrombus, embolus, trauma, or vasospasm (often due to drugs).

jugular venous bulb oximetry A technique in which a sampling catheter is placed in the internal jugular vein and directed upward so that its tip rests in the jugular venous bulb at the base of the brain; samples of blood can then be drawn to measure mixed venous oxygen saturation (SvO_2).

Le Fort criteria A categorization of facial fractures involving the maxilla that are differentiated based on the location of fracture lines and the extent of mobility of facial structures on physical examination.

Lhermitte phenomenon A condition in which forward flexion of the neck produces an electric shock feeling, usually running down the back.

limbic lobe (rhinencephalon) Part of the temporal lobe that is the seat of emotions and instincts.

linear skull fracture A type of skull fracture characterized by a single fracture line.

linear stellate fracture A fracture with multiple linear fractures radiating from the site of impact.

lumbar puncture A procedure in which a needle is inserted into the lumbar portion of the back and into the subarachnoid space to obtain spinal fluid for testing or to administer drugs.

magnetic resonance imaging (MRI) A noninvasive procedure that provides computer images of internal organs and the body through the use of radio frequency pulses and a magnetic field.

mean arterial pressure (MAP) The mean between the systolic and diastolic blood pressures (SBP and DBP): MAP = DBP + 1/3(SBP – DBP).

medulla oblongata The lowermost portion of the brain stem.

midbrain A small area of the brain stem extending between the diencephalon, the pons, and the third ventricle.

mild TBI Concussion; a traumatically induced physiologic disruption of brain function that occurs without structural damage.

Mini-Mental State Examination A simple, easily applied test of higher cognitive functions.

mixed venous oxygen saturation (SvO_2) A measurement of the oxygenation of blood leaving the brain, which is normally in the range of 50% to 75%.

Monro–Kellie doctrine A theory developed by two Scottish anatomists, who stated that the central nervous system is enclosed in a rigid compartment along with cerebrospinal fluid, whose total volume

tends to remain constant; an increase in any component—whether brain, blood, or CSF—will cause an increase in pressure and decrease the volume of one of the other elements.

motor area Part of the frontal lobe that contains pyramidal cells that control voluntary motor function on the opposite side of the body.

neurocranium The part of the skull that encloses and protects the brain.

neurogenic shock A temporary loss of autonomic function, which controls cardiovascular function, at the level of a spinal cord injury; it is characterized by marked hemodynamic and systemic effects.

normal pressure hydrocephalus (NPH) An accumulation of CSF that causes the ventricles of the brain to enlarge. The enlarged ventricles of a patient with this condition may not cause increased intracranial pressure.

nuchal rigidity Marked resistance to head movement in any direction that is suggestive of meningeal irritation.

occipital lobe The lobe of the brain that occupies the most posterior portion of the cerebrum; it is the primary receptive area for vision, specifically the interpretation of visual stimuli.

optic disk The most prominent structure visible in the eye; it represents the termination of the optic nerve.

oxygen extraction fraction (OEF) The fraction of oxygen extracted from the blood as it passes by to maintain normal oxygen delivery and, consequently, normal brain functions.

parietal lobe The lobe of the brain situated directly posterior to the frontal lobe on the other side of the central fissure; it is largely responsible for sensory functions.

pia mater The innermost layer of the meninges, which rests directly on the brain or spinal cord.

ping-pong ball fracture A pediatric greenstick fracture of the skull.

plexus A cluster of nerve roots that permits peripheral nerve roots to function as a group.

pons Part of the brain stem located between the midbrain and the medulla oblongata; it relays information to and from the brain and spinal cord along fiber tracts.

posterior cord syndrome Extension injury that produces dysfunction of the dorsal columns, presenting as decreased sensation to light touch, proprioception, and vibration.

prefrontal area Part of the frontal lobe that provides control of thought, concentration, depth and ability to think abstractly, memory, and autonomic nervous system response, concomitant to emotional change.

premotor area Part of the frontal lobe that is adjacent to the motor area and helps coordinate certain movements.

primary spinal cord injury Spinal cord injury that occurs at the moment of impact.

proprioception The ability to perceive the position and movement of one's own body or limbs.

ptosis Drooping of an eyelid.

raccoon eyes Orbital fractures and hemorrhage into the surrounding tissue; also called periorbital ecchymosis.

reticular activating system (RAS) A diffuse system that extends from the lower brain stem to the cerebral cortex; it controls the sleep-wakefulness cycle, consciousness, the ability to direct attention to a specific task, and the perception of sensory input that might alter behavior.

reticular formation (RF) A set of neurons that extends from the upper level of the spinal cord, through

the medulla, pons, and midbrain, and into the thalamus and cerebral cortex. It has both excitatory and some inhibitory capabilities, and can enhance, suppress, or modify impulse transmission.

reverse Trendelenburg Supine position with head higher than feet.

rotational force An injury-producing force in which the head moves around its center of gravity.

rotation–flexion injury A spinal cord injury to C1-C2, the only area of the spine that allows for significant rotation, in which rotation with abrupt flexion produces a stable dislocation in the cervical spine. In the thoracolumbar spine, rotation-flexion forces typically cause fracture rather than dislocation.

rubrospinal tract Part of the midbrain that controls the tone of flexor muscles.

secondary spinal cord injury Spinal cord injury in which multiple factors permit a progression of the primary spinal cord injury; the ensuing cascade of inflammatory responses may result in further deterioration.

spinal shock The temporary local neurologic condition that occurs immediately after spinal trauma; it is characterized by swelling and edema of the spinal cord and can lead to a physiologic transection, mechanically disrupting all nerve conduction distal to the injury.

stereognosis The ability to sense an object's form through touch.

stroke Results from a disruption of blood flow to the brain that results in neurologic deficit persisting for more than 24 hours.

subarachnoid hemorrhage (SAH) Bleeding between the arachnoid mater and dura mater of the brain.

subdural hematoma (SDH) Bleeding that accumulates between the dura mater and arachnoid mater.

subdural space The dura-arachnoid junction; this potential space may develop into a real one if a blow to the head causes a loss of blood into the cranial meninges.

subthalamus An area of the cerebrum that is located below the thalamus and is closely related to the basal ganglia in function.

sulci Grooves between adjacent gyri.

tectospinal tract Part of the midbrain that controls reflex motor movements in response to visual and auditory stimuli.

temporal lobe The lobe of the brain that is located beneath the temporal bone of the cranium; its primary functions relate to hearing, speech, behavior, and memory.

tentorium cerebelli A fold of the dura mater that separates the occipital lobes of the cerebrum from the cerebellum and brain stem, thereby dividing the brain into upper and lower compartments.

thalamus The largest portion of the diencephalons; it acts as a relay station for motor and sensory activity; basic neuronal activity; and memory, thought, emotion, and complex behavior.

thrombotic stroke Occurs when the blood supply to part of the brain is disrupted by a thrombus, or blood clot.

transcranial Doppler (TCD) ultrasound A noninvasive method of assessing the state of intracranial perfusion and monitoring of cerebral blood flow velocity through thinner areas of the skull; used in patients following rupture of an intracranial aneurysm to assess for vasospasm, used to identify intracranial lesions following a stroke, and used for the detection of cerebral blood flow changes associated with increased ICP.

transient ischemic attack (TIA) A temporary disruption in the blood flow to the brain that lasts less

than 24 hours with temporary side effects.

translational force An injury-producing force in which the head's center of gravity moves along a linear path.

true fracture The most common type of closed skull fracture, in which the depressed segment has contact with the cranial vault.

two-point discrimination A test of sensation discrimination that measures the shortest distance at which the sides of two separate points of a compass or calipers can be distinguished from one another.

uncal herniation The most common type of brain herniation, in which a portion of the temporal lobe is displaced, resulting in compression of cranial nerve III, the midbrain, and the posterior cerebral artery.

uncus The medially curved anterior part of the hippocampal gyrus.

ventral Toward the abdomen; anterior.

vertebral body The anterior weight-bearing structure within the spine.

vertical compression Forces transmitted through vertebral bodies and directed either inferiorly through the skull or superiorly through the pelvis or feet (eg, from a direct blow to the parietal region of the skull or rapid deceleration from a fall through the feet, legs, and pelvis).

viscerocranium The bones making up the facial skeleton.

voluntary (somatic) nervous system The nervous system fibers that connect the structures of the CNS with skeletal muscles and the integument.

Wernicke's area Part of the temporal lobe that is responsible for comprehension of both written and spoken words.

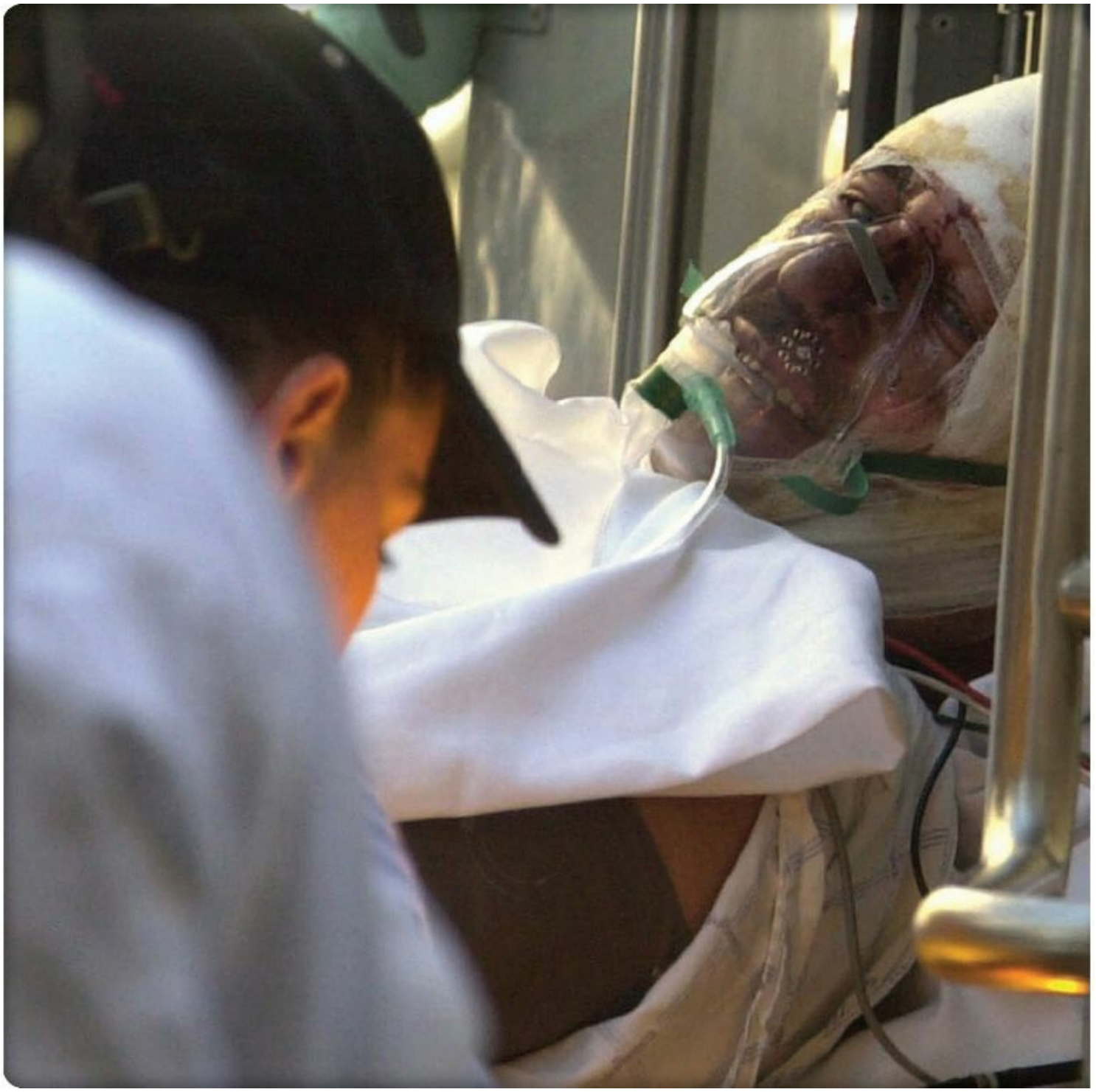
Bibliography

Adams HP, Brott TA, Crowell RM, et al. Guidelines for the management of patients with acute ischemic stroke: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Circulation*. 1994;90(3):1588. Available at: http://www.strokecenter.org/education/ais_evaluation/brain_stem.htm. Accessed March 9, 2009.

Fuller G. *Neurological Examination Made Easy*. Edinburgh, Scotland: Churchill Livingstone; 1999.

Kinney MR, Dunbar SB, Brooks-Brunn JA, Molter N, Vitello-Ciccu JM. *AACN Clinical Reference for Critical Care Nursing*. St Louis, MO: Mosby; 1998.

McCance K, Huether S. *Pathophysiology—The Biological Basis for Disease in Adults and Children*. St Louis, MO: Mosby; 2002.



Burns

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Objectives

1. Describe the layers and functions of the skin (p 444).
2. List the major causes of burn injury (p 445).
3. Describe the anatomy of a burn (p 446).
4. Explain the process of the body's systemic inflammatory response to a burn (p 446).
5. Explain the factors that determine the classification of burn injury, including body surface area and burn depth (p 446).
6. List the classifications of burn injuries, including superficial burns, partial thickness burns, deep partial thickness burns, full-thickness burns, subdermal burns, as well as minor burns, moderate burns, and major burns (p 446–447).
7. Identify the methods for calculating the total body surface area burned, including the rule of nines and the Lund and Browder chart (p 448).
8. Describe how to evaluate a burn patient and what constitutes a pertinent history (p 449).
9. Discuss assessment considerations for a burn patient's airway, breathing, and circulation (p 449–452).
10. List situations in which the CCTP should suspect an inhalation injury (p 451).
11. Discuss the roles of edema and compartment syndrome in relation to a burn injury (p 451–452).
12. Describe the process of stopping a burn, including irrigation, cooling, decontamination, and special considerations (p 453).
13. Discuss management of a burn patient's airway, breathing, and circulation (p 453–455).
14. Discuss special considerations for patients who may have carbon monoxide poisoning or cyanide poisoning in conjunction with a burn injury (p 451).
15. Explain various fluid resuscitation formulas, including the Parkland formula and the Galveston formula, and describe parameters for adjusting the infusion rate (p 455).
16. Describe how to manage and dress burn wounds (p 455–456).
17. Discuss pain management of burn patients (p 456–457).
18. Recognize which patients require transport to a burn center or unit (p 449, 456, 457, 458–464).
19. Discuss special situations relating to burn injuries such as hypothermia, the need for gastric decompression, renal failure, and rhabdomyolysis (p 457–458).
20. Discuss management of specific burns, including ocular burns, facial burns, ear burns,

circumferential burns, hand and foot burns, genitalia burns, pediatric burns, electrical burns, and chemical burns (p 458–463).

21. List the types of burns that may suggest potential child maltreatment (p 460).
22. Discuss toxic epidermal necrolysis (TENS) and Stevens-Johnson syndrome (SJS) and the similarity between their management and the management of severe burn patients (p 464).

Introduction

According to the American Burn Association there are approximately 1 million burn injuries per year in the United States resulting in approximately 500,000 emergency department visits, 40,000 hospitalizations, and 4,000 fire and burn-related deaths. Approximately 25,000 of the admissions per year are to specialized burn units, with the majority of these patients requiring transfer to receive their specialized care. In the year 2000, there were 125 burn centers in the United States with an average of 200 admissions per year. The average size of a burn injury that requires admission is 14% total body surface area (TBSA). Six percent of burn center admissions do not survive. The highest incidence of burns occurs in the first few years of life and between 20 and 29 years of age. Serious burns occur most often in males (67%), with the majority of these burns resulting from flame.

Most of the approximately 25,000 admissions to burn units require transfer via EMS to specialized care. Specialized burn care may be provided at burn centers or burn units. Burn centers are large facilities that admit more than 100 patients per year, whereas burn units are specialized facilities that have fewer than 100 admissions per year. The principles of acute burn management taught in basic paramedic and nursing courses are the foundation for caring for these patients. However, the first 24 hours of care after a burn are crucial. Factors such as unrecognized injuries from associated trauma, complications of treatment such as fluid overload, and comorbidities such as diabetes or chronic obstructive pulmonary disease may complicate care, and the conditions of patients who appear stable may acutely worsen. Given that complications of burns may develop during the transport of the patient to a burn facility, the CCTP must ensure that adequate resuscitation and stabilization continue during transport. Knowledge of common complications and the timing of their treatment are important for safe and effective transport to specialty care centers.

Anatomy and Function of the Skin

The skin is the largest organ of the body and its function is as vital to survival as any other organ. A basic understanding of skin anatomy and function will help the CCTP understand burn treatment and potential complications. The skin is a multilayered structure composed of the epidermis and dermis as the principle components **Figure 12-1**. The epidermis and dermis are divided into additional levels that have specific functions.

The Epidermis

The epidermis generally consists of four layers (thin skin) except on the palms, fingertips, and soles of the feet, where five layers exist (thick skin). Each layer has a different composition.

The layers of the epidermis from deep to superficial are the stratum basale, which is attached to the underlying dermis, the stratum spinosum (prickly layer), stratum granulosum, stratum lucidum (thick skin only), and most superficially, the stratum corneum.

The epidermis contains the pigment cells known as melanocytes, immune cells known as Langerhans cells, as well as capillaries in the lower layers. It is important to note that while the epidermis does

contain some receptors for touch, the nerve receptors that transmit pain are located in the deeper dermis layer. The outer layer of the epidermis, the stratum corneum, provides a barrier to prevent water loss from deeper structures, and is constantly losing cells as a result of sloughing. It takes approximately 2 to 3 weeks for a cell to make its way from the lowest layer to become a keratinocyte and eventually be sloughed off. As the epidermis is the first barrier to injury and infection, it is injured more often than other layers and is constantly repairing itself.

The Dermis

The dermis lies beneath the epidermis and is a dynamic layer of thick connective tissue that, like the epidermis, is in constant turnover. The cell types found here are fibroblasts, macrophages, white blood cells, and the occasional mast cell. The dermis is also richly supplied with nerve fibers, blood vessels, and lymphatic vessels. The major portions of the hair follicles and the oil and sweat glands are also found in the dermis.

The dermis is divided into two layers: the papillary and reticular layers. The thin superficial papillary layer is composed of connective tissue forming the anchoring system for the epidermis. It is also heavily invested with blood vessels yielding the highest blood flow of the dermal layers, which is important for temperature regulation. These blood vessels are found in nipple-like projections called dermal papillae that indent the overlying epidermis. These dermal papillae also house free nerve endings (pain receptors) and touch receptors.

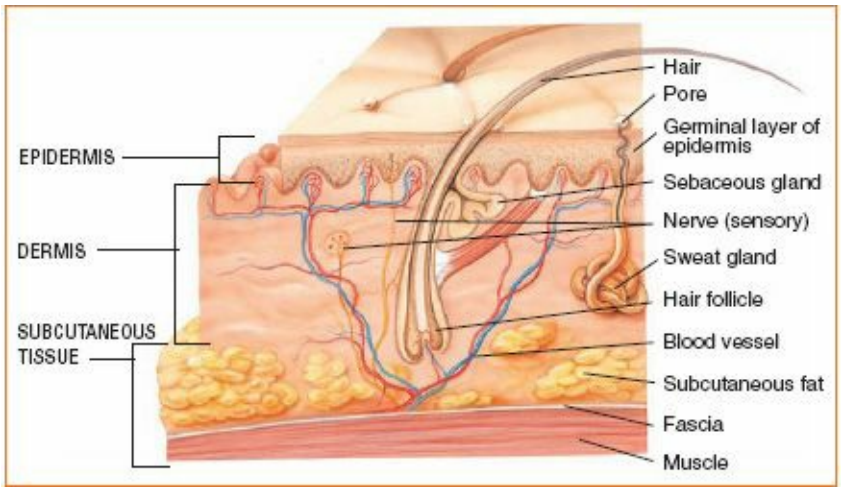


Figure 12-1 The skin has two principal layers: the epidermis and the dermis.

The reticular layer accounts for 80% of the dermis. Its dense irregular connective tissue is responsible for the durability of the skin and anchors skin appendages. Interlocking collagen fibers run in various planes, usually parallel to the skin. Regions between these bundles form *lines of cleavage* in the skin **Figure 12-2**. These cleavage lines run longitudinally in the skin of the head and limbs and in a circular pattern around the neck and truck. This becomes important when considering where to make incisions for escharotomies and fasciotomies because incisions made parallel to these lines cause the skin to gape less and heal more readily than incisions made across cleavage lines.

Collagen fibers give the skin strength and resiliency against injury. Collagen also helps maintain hydration of the skin, an important concern after a major burn. Another concern is the adverse effect burns have on the elastin fibers, which provide the stretch-recoil properties of the skin.

Deep in the dermis lies the hypodermis, which is composed of subcutaneous fat, connective tissue, sweat glands, muscle, and bone.

The Process of Healing

Proteins that are produced in the dermis, such as collagen and fibronectin, support the continuously reproducing epidermis and play a major role in wound healing. Multiple cell types are contained in the dermis when undamaged, but many more are attracted to the area when it is wounded.

Fibroblasts produce proteins such as collagen, fibronectin, and others responsible for skin repair. Macrophages are normally present in the tissues, but increase in numbers after an injury. Macrophages release chemical messages that help attract other cells and orchestrate organized healing. Endothelial cells produce capillaries and restore blood flow after an injury. Fibroblasts, macrophages, platelets, and endothelial cells play a vital role in the wound healing process of the skin. The inflammatory response that is responsible for burn healing is also responsible for multiple complications that will be discussed later.

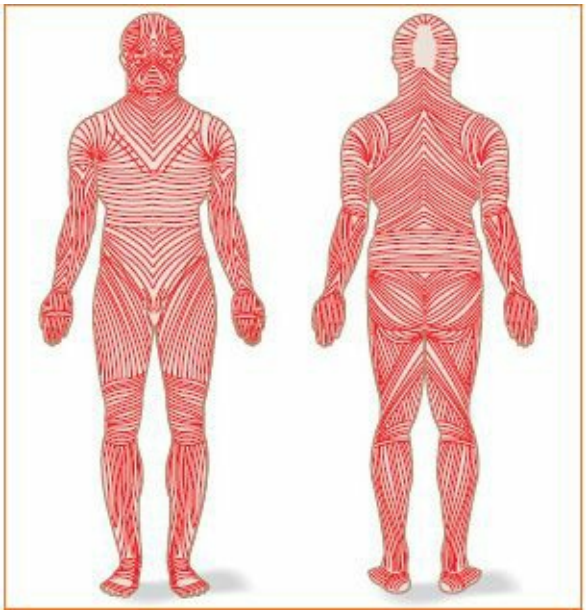


Figure 12-2 The skin's lines of cleavage. Incisions made parallel to these lines heal more easily than incisions made perpendicular to these lines.

Functions of the Skin

The skin has multiple functions including protection, immunologic, thermo-regulation, maintenance of fluid and electrolyte balance, metabolism, neurosensory, and social. All of these functions of the skin are essential to the normal functioning of the body, and any or all of these functions can be impaired in a burn injury. The epidermis serves as a barrier that protects the body from desiccation (drying out) and maintains fluid balance by preventing excess loss of fluid through evaporation. An intact epidermis is also relatively effective at preventing the entry of toxins and bacteria. Burn injuries result in damage to the epidermis, increasing the risk of fluid loss through evaporation from the skin and allowing the direct entry of bacteria through the compromised skin.

The neurosensory function of skin is also important for social and interactive functioning. This ability to sense temperature and touch may be impaired or absent in the burned patient.

The skin has a complex mechanism by which wound healing and skin repair occur. In severe burns this intrinsic healing mechanism may be irreparably damaged, necessitating skin grafting for wound closure.

Because of its elastic properties, the dermis protects a person from minor trauma and assists in the regulation of fluid balance through its regulation of blood flow. This regulation of blood flow is also a major mechanism by which the body regulates temperature. Because the skin is responsible for fluid and

temperature regulation, large burns (greater than 20% in adults and 15% to 18% in children) can cause massive fluid loss, shock, and hypothermia, even on a hot day. The skin also serves as a barrier from infection, and burn damage gives bacteria easier access to the body. Damaged skin is also more susceptible to pressure injuries such as bed sores. Finally, the skin is a sensory organ that can cause intense pain and jeopardize the patient's ability to recognize touch after a burn. Any burn, regardless of the cause, can alter these vital functions; the degree of dysfunction is determined by the extent and depth of the burn.

Physiology of Burns

Causes of Burns

Burns occur from a variety of mechanisms: flame/flash, scald (for example, water or grease), contact, electrical, chemical inhalation, radiation, or any thermal source. In addition, some medical conditions such as **Stevens-Johnson syndrome** and **toxic epidermal necrolysis syndrome** cause burnlike injuries and are treated similar to heat-related burns. Regardless of the mechanism of injury, all burns share common features—the destruction of skin and an impairment of its ability to perform its functions. The size and depth of the burn are determined by the agent, temperature, and duration of exposure. **Thermal burns** having temperatures greater than 113°F can cause cell damage and denaturing of cellular proteins. This denaturing of proteins eventually causes cell death. At temperatures of 120°F, an exposure for 5 minutes is enough to cause a full-thickness burn, and at temperatures above 159°F, it only takes 1 second to create a full-thickness burn in a healthy adult. These temperatures and times may vary when children and the elderly are affected.

Anatomy of a Burn

The most damaged area of a burn is referred to as the **zone of coagulation** and is the area of skin that came into direct contact with the source of the heat. In the zone of coagulation, the tissue is destroyed and clotting results in impairment of circulation to the surrounding tissues. The surrounding areas of injury are referred to as the zone of stasis and the zone of erythema. The **zone of stasis** has injured tissue and stagnant blood flow resulting in ischemic tissues. It may appear red and **hyperemic** (filled with an increased amount of blood) initially, but within 24 hours after injury, blood flow will cease in the area and ischemia results. By the third day after the injury the zone of stasis becomes white because of avascular necrosis. The area surrounding the zone of stasis is the outermost area known as the **zone of erythema** owing to its increased blood flow. This area has sustained minimal damage and will recover from the injury.

Systemic Inflammatory Response of the Burned Patient

Although inflammation is necessary for the normal healing process to occur, excessive inflammation results in undesirable effects on other tissues and organs. Burns of greater than 25% TBSA result in a systemic inflammatory response. Cells in the body migrate to areas of injury to affect repairs at the injury site. A blood-rich environment is necessary for these cells to function properly. In larger areas of injury, however, the vascular endothelium becomes "leaky" in order for "repairing" cells to make their way from the vasculature into the wound. When this occurs at noninjured sites in the body, undesirable results occur, such as those that follow:

- Proteins may leak into subcutaneous tissues at uninjured sites, or the pressure at which proteins and fluids may be exuded into the lungs may be lowered, making pulmonary edema more likely.

- The mediators can suppress the immune system, adding insult to injury.
- The leaky endothelial cells of the intestinal vasculature may allow bacteria normally found in the bowel to make its way into the bloodstream (a process known as translocation), causing sepsis, even in the absence of a burn wound infection.
- Cardiac output may also be impaired after a burn injury. Although the mechanism is unclear, myocardial contractility is suppressed after a significant burn injury.

Regardless of the cause, the inflammatory response from a burn injury, although necessary to promote burn healing, may also result in untoward effects.

Classification of Burn Injury

The severity of a burn depends on (1) extent, depth, and location of the burn injury; (2) age of the patient; (3) etiologic agents involved; (4) presence of inhalation injury; and (5) coexisting injuries or preexisting illnesses.

Two classification systems for the depth of burns exist. The system describing first-through fourth-degree burns is widely known and used, but is less predictive of the need for surgical intervention than the simple partial- or full-thickness descriptions **Table 12-1**. Both systems are discussed here.

Burn Extent and Depth

Superficial burns, or first-degree burns, involve only the epidermis and are usually the result of ultraviolet light exposures (sunburn), very minor scald injuries, or flash burns. The skin is hyperemic but not blistered, with healing occurring without scarring in approximately seven days.

TABLE 12-1 Classification of Burns

Depth	Color and Vascularity	Surface Appearance and Pain	Swelling, Healing, and Scarring
Superficial (first degree)	Erythematous, pink and red	No blisters Dry and tender	Slight edema Heals easily without scarring
Superficial to partial thickness (second degree)	Erythematous, bright pink/red, mottled Blanches with brisk capillary refill	Intact blisters, moist when removed Weeping wounds and extremely painful	Moderate edema Easily heals but with skin discoloration
Deep to partial thickness (second degree)	Red, waxy-white Blanches with slow capillary refill	Broken blisters, wet Sensitive to pressure, but not to light touch	Marked edema Heals slowly with hypertrophic scars
Full thickness (third degree)	White, black to red/tan No blanching, vessels thrombosed Poor distal circulation	Dry, leathery Anesthetic Hairs pull out easily	Skin grafting required Scarring likely after healing
Subdermal (fourth degree)	Charred	Obvious subcutaneous tissue involvement Anesthetic	Skin grafting and/or flap required Scarring after healing

Partial-thickness burns, or second-degree burns, are subdivided into superficial and deep burns. In **superficial partial-thickness burns** (superficial second-degree burns) the epidermis and part of the dermis are involved, but the deeper layers of the dermis are spared, including the sweat glands, sebaceous glands, and hair follicles. These burns are typically caused by hot liquids or minimal contact with flame. Blisters form, and the injured skin is red and painful to touch. These burns do not typically require skin grafting unless they are extensive and occur on areas of the body with thin skin such as the face, hands, genitals, and feet. Superficial partial-thickness burns will heal in 14 to 21 days. The extent of the scarring depends on the extent of the burn. **Deep partial-thickness burns** (deep second-degree burns) are most often the result of steam, oil, or flames, and involve the deeper layers of the dermis. They may be difficult to distinguish from full-thickness burns. The skin is blistered but not charred and is painful to touch. Healing takes 21 days or more and may require surgical grafts. Scarring may be moderate and depends on the extent and location of the burn.

Full-thickness burns, or third-degree burns, involve the entire thickness of the dermis down to the subcutaneous fat. All epidermal and dermal structures, including nerve endings in the burned area, are destroyed, resulting in a pale, painless, leathery charred area of skin. Full-thickness burns will not heal spontaneously and surgical skin grafting is necessary. Full-thickness burns result in significant scarring.

Subdermal burns, or fourth-degree burns, involve the deep structures of muscle and bone, larger blood vessels, and nerves. These injuries are severe and life threatening. Like full-thickness burns, they will not heal spontaneously and require surgical intervention.

Superficial, partial-thickness, full-thickness, and subdermal burns are shown in **Figure 12-3**.

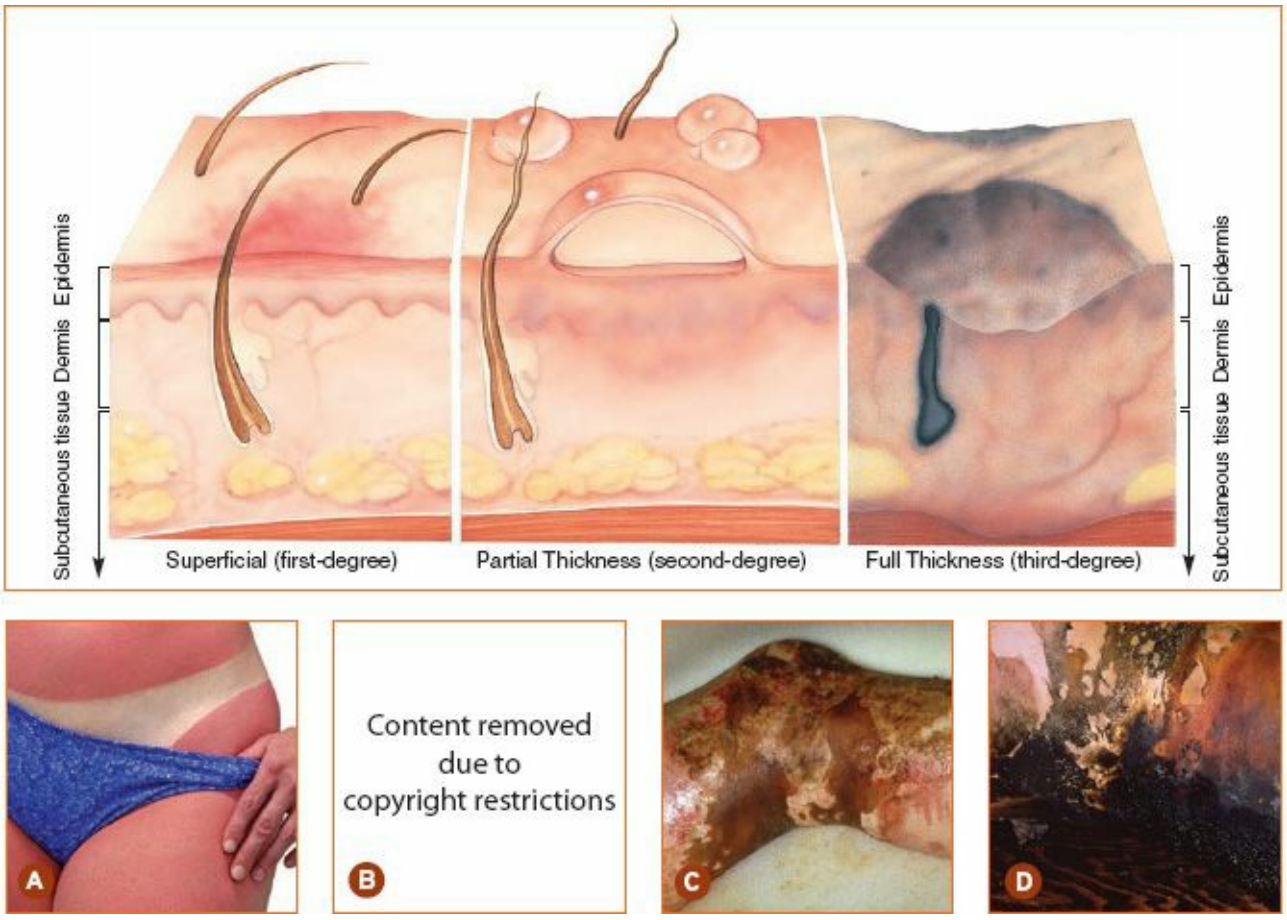


Figure 12-3 Classification of burns. **A.** Superficial (first-degree) burns involve only the epidermis. **B.** Partial-thickness (second-degree) burns involve some of the dermis but do not destroy the entire thickness of the skin. The skin is mottled, white to red, and often blistered. **C.** Full-thickness (third-degree) burns extend through all layers of the skin and may involve subcutaneous tissue and muscle. The skin is dry, leathery, and often white or charred. **D.** Subdermal (fourth-degree) burns involve deep structures such as

muscle, bone, large blood vessels, and nerves.

Burn Size

As stated previously, the depth of the burn is only one factor determining its severity; the size of the burn is also a determining factor. Numerous methods are available to estimate the body surface area involved in a burn. The **rule of nines** is the most common formula and is based on the fact that large regions of the adult body can be divided into areas that represent roughly 9% of the total body surface area or multiples of 9% **Figure 12-4**. For infants and children an amended rule is used that adjusts the rule of nines to account for the relatively large head and small lower extremities. For patients with scattered irregular burns or small burns, the patient's palm may be used to estimate the burn area. In children and adults, the palm (not including fingers) represents 0.5% of the patient's total body surface area. Previously the palm was thought to represent 1% of the body surface area. However, in 1995 Sheridan and coworkers demonstrated that 0.5% is more accurate. Data also demonstrate that the **Lund and Browder chart** **Figure 12-5** is more accurate than the rule of nines for calculating the body surface area burned and is the preferred method in most burn centers. However, the chart is difficult to memorize, making the rule of nines the most common method in the prehospital environment. Regardless of the method used, accuracy improves with experience.

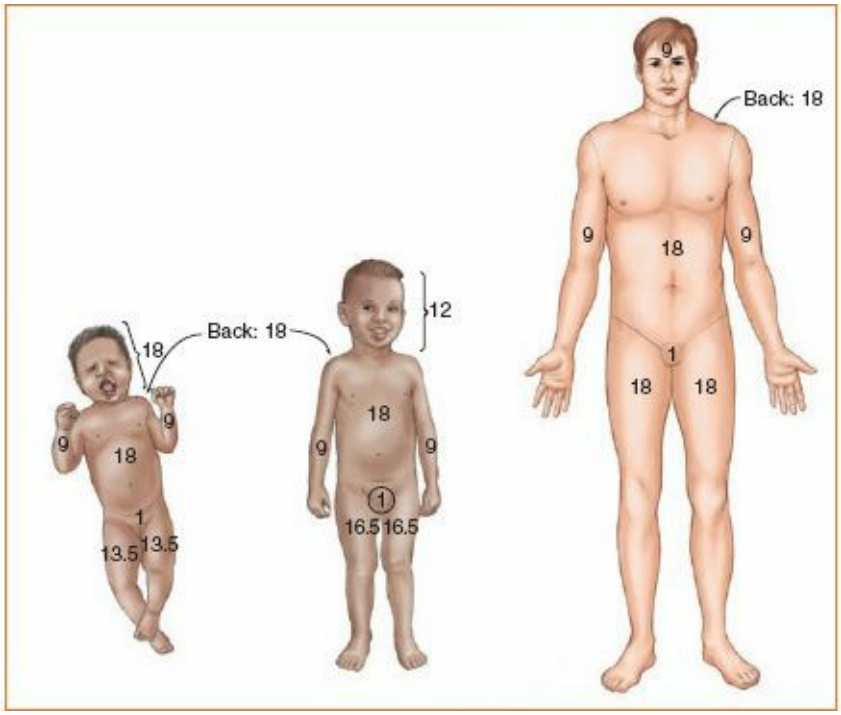
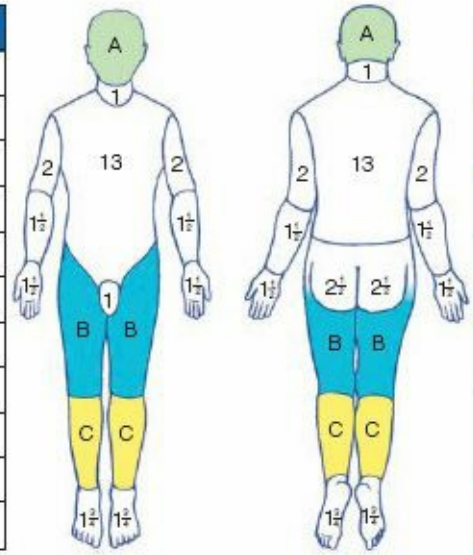


Figure 12-4 The rule of nines. The body is divided into sections, each representing approximately 9% of the total body surface area. The proportions differ for infants, children, and adults.

Region	%
Head	
Neck	
Ant. Trunk	
Post. Trunk	
Right arm	
Left arm	
Buttocks	
Genitalia	
Right leg	
Left leg	
Total burn	



Relative percentages of body surface area affected by growth

Age (years)	A (½ of head)	B (½ of one thigh)	C (½ of one leg)
0	9½	2¾	2½
1	8½	3¼	2½
5	6½	4	2¾
10	5½	4¼	3
15	4½	4½	3¼
Adult	3½	4¾	3

Figure 12-5 The Lund and Browder chart.
Adapted from: Lund CC, Browder NC. *Surg Gynecol Obstet*. 1944;79:352–358.

Special Populations

Fetal mortality is closely linked to maternal mortality when considering burn victims. Generally speaking, a maternal patient with greater than 60% total body surface area burns has a high rate of fetal mortality.

Burn Severity

The American Burn Association classifies burns into three major categories—major, moderate, and minor.

Major Burn Injury

Major burn injuries include:

1. Partial-thickness burns involving more than 25% of BSA in adults or 20% of BSA in children younger than 10 years and adults older than 50 years.
2. Full-thickness burns involving more than 10% of BSA.
3. Burns involving the face, eyes, ears, hands, feet, or perineum that may result in functional or cosmetic impairment.
4. Burns caused by caustic chemical agents.
5. High-voltage electrical injury.
6. Burns complicated by inhalation injury or major trauma.
7. Burns sustained by high-risk patients (eg, patients with debilitating diseases such as diabetes or congestive heart failure).

Moderate Burn Injury

Moderate burn injuries include the following (note that this list excludes high-voltage injury):

1. Partial-thickness burns of 15% to 25% of BSA in adults or more than 10% to 20% of BSA in children and older adults.
2. Full-thickness burns involving 2% to 10% of BSA that do not present a serious threat to functional or cosmetic impairment of the eyes, ears, face, hands, feet, or perineum.

Minor Burn Injury

Minor burn injuries include:

1. Partial-thickness burns involving less that 15% of BSA in adults or 10% of BSA in children and older adults.
2. Full-thickness burns involving less that 2% of BSA that do not present a serious threat of functional or cosmetic risk to the eyes, ears, face, hands, feet, or perineum.

Patients who meet the criteria for major burn injury should be transferred to a specialized burn center to ensure appropriate acute care, rehabilitation, and long-term care. Patients who meet the criteria for moderate burn injury should be hospitalized and consideration given to transferring care to a specialized burn center. Patients with minor burn injuries can usually be managed in the outpatient setting.

Assessment

Assessment for burn patients should be similar to any other patient. However, there are particular complications and findings that must be detected in order to take corrective action **Figure 12-6**.

A complete initial assessment and focused history and physical exam should be performed, always using caution whenever a c-spine injury is suspected. Keep in mind that explosions may produce c-spine injuries that may not be as obvious as the visible multiple burns. The CCTP should not rely on the history and exam of others because their level of training may not be equivalent to that of the CCTP. In addition, the patient's condition may have changed since the last exam. As always during interfacility transfers, an initial evaluation of the patient for life-threatening problems should be conducted and then a history obtained from the patient and/or family members and a report from the nursing staff. In order to avoid excessive delay in initiating transfer, the CCTP should review the chart for the most relevant information. The CCTP can then fill in remaining details by asking the patient, staff, or family. A focused history and physical exam of the patient should be performed to assess for complications that need corrective action and to document the patient's condition. Vital signs, including temperature and oxygen saturation, should also be obtained again prior to transporting the patient. The initial assessment should include the ABCs, and the physical exam should follow the head-to-toe format, with attention paid to each organ system.

Initial Assessment

Airway

The CCTP should pay special attention to the airway of the burned patient, particularly if the patient was burned as the result of a confined space fire. Inhaling heated gases from combustion damages the respiratory tract. Inhaled gases, particles, and other debris damage the respiratory epithelium resulting in increased mucus production, impairment of mucociliary function, and eventual cell death. Inflammatory changes in the airway occur *within 2 hours of injury*. Bacteria colonize the damaged airway and infection is present microscopically within 72 hours. The extent of the inflammatory response is directly proportional to the dose of smoke inhaled and length of exposure.

The CCTP should inspect the airway and face for any evidence of inhalation injury and, if present, consider performing endotracheal intubation after rapid sequence intubation (RSI) if the transport time is

prolonged or if there is any deterioration in the patient's condition. Preemptive airway control is necessary because of the worsening edema that occurs after fluid resuscitation, potentially causing airway obstruction and necessitating a surgical airway. If stridor, progressive hoarseness, or any change in the patient's voice is present, the patient should be intubated *immediately using RSI*, with instruments for performing a surgical airway on hand.

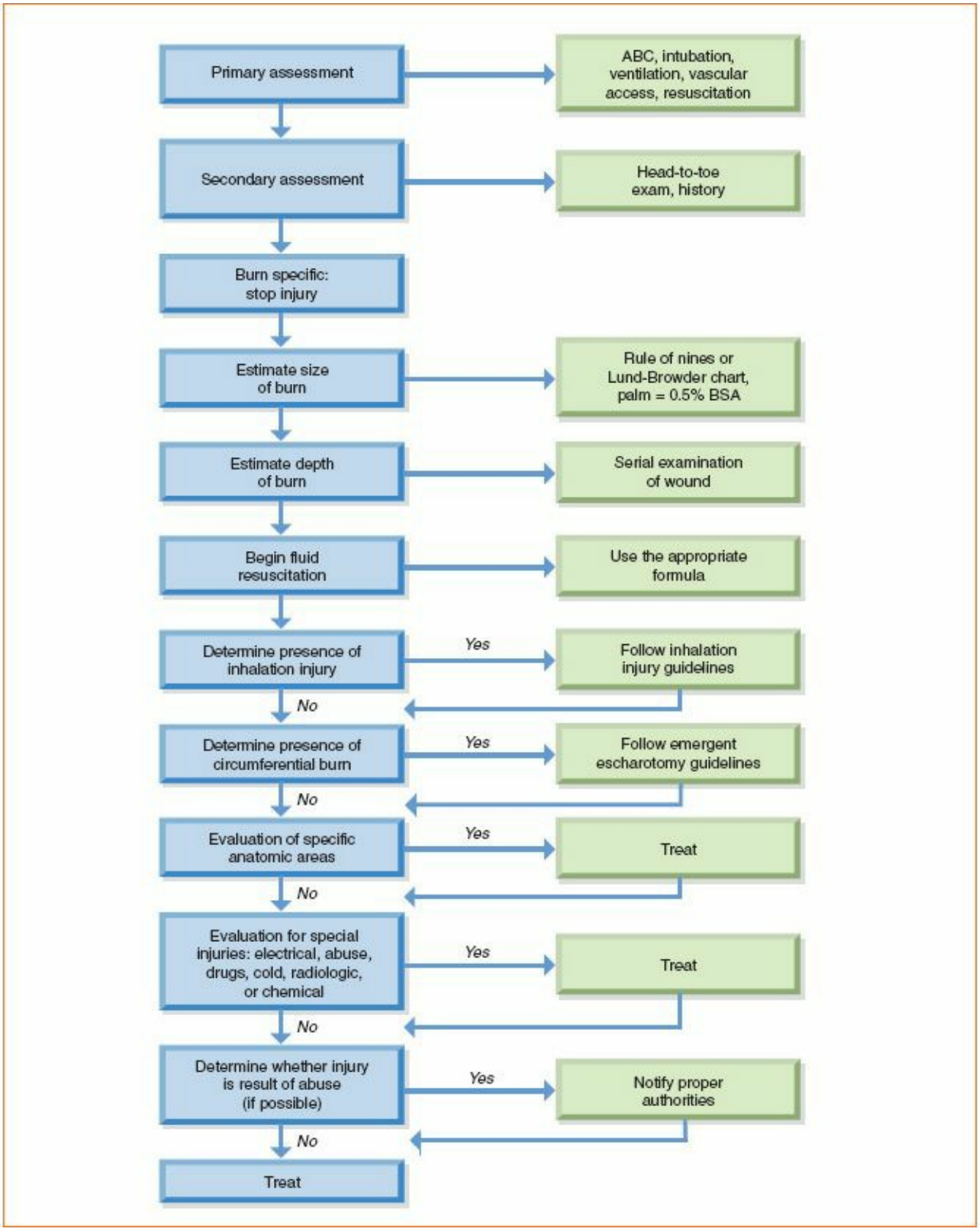


Figure 12-6 Algorithm for initial assessment of a burn injury.

Inhalation injury should be suspected if the patient has any of the following:

1. Facial burns
2. Stridor or progressive hoarseness
3. Singeing of the eyebrows and nasal vibrissae (hairs)
4. Carbon deposits and acute inflammatory changes in the oropharynx
5. Carbonaceous (black) sputum
6. History of impaired mentation and/or confinement in a burning environment
7. Explosion with burns to the head and torso
8. Circumferential neck burns
9. Carboxyhemoglobin level greater than 10% if the patient is involved in a confined space fire
10. Soot around the nose and mouth

The patient's airway should be reevaluated frequently to assess for potential compromise due to edema. In addition, patients with an altered mental status or a Glasgow Coma Scale score of 8 or less after a major burn or any injury should have their airway controlled. If the patient is to be intubated for an elevated carboxyhemoglobin level, water should be used to fill the endotracheal tube cuff in preparation for hyperbaric therapy. However, it is often difficult for the CCTP to predict who will need hyperbaric oxygen, and an air-filled cuff can always be changed to water prior to initiating hyperbaric treatment. It is important to point out that if RSI is not in the scope of practice for the CCTP in your jurisdiction, attempted intubation in the setting of an inhalation injury is likely to result in worsened edema and potentially a failed airway. In this situation a risk/benefit assessment of airway management without RSI must be undertaken. Consideration should be given to delaying the transfer of the patient until the airway can be managed by someone skilled in airway management and capable of RSI.

Breathing

The patient's breathing rate and depth should be assessed along with the lung sounds. Noncardiogenic pulmonary edema may develop as a result of inhalation injury or fluid overload from aggressive fluid replacement, but needed fluids should never be withheld. High-flow oxygen should be started immediately in the patient with major burns, using caution in patients with chronic obstructive pulmonary disease (COPD) due to suppression of the hypoxic drive. Although COPD often causes concern regarding oxygen therapy, a hypoxic patient should never have high-flow oxygen withheld, and the CCTP should be prepared to assist ventilation if needed. Examination of the chest should include a search for full-thickness circumferential burns, which may significantly impair ventilation by impairing expansion of the chest. A field escharotomy is rarely, if ever, needed. Although it takes time for tissues to dessicate (dry out) and a constrictive **eschar** (the leathery covering of a burn injury) to develop, the CCTP will encounter burn patients at varying times after injury and an escharotomy may be necessary. For example, the CCTP may need to perform chest wall escharotomies in extreme cases with extended transport times. Emergent chest wall escharotomy may be performed (if scope of practice allows) to allow for adequate movement of the chest wall. If available, the arterial blood gas levels should be reviewed, paying particular attention to the oxygenation and carboxyhemoglobin levels. Wheezing should be treated with bronchodilators, as is standard. Another therapy to consider includes humidified oxygen for inhalation injuries (in the absence of wheezing) using a nebulizer with normal saline only and no bronchodilators.

Burn patients may have experienced significant trauma from explosions or motor vehicle collisions resulting in fires. In such situations it is easy to overlook conditions such as a pneumothorax or hemothorax while treating the obvious burn. Repeated patient examinations should be aimed at identifying these conditions.

Patients with carbon monoxide poisoning may present with symptoms of hypoxia, but without

cyanosis. The classical teaching is that patients present with cherry red skin, although this is not often identified until postmortem. The CCTP should be aware of the limitations of pulse oximetry in the patient with carbon monoxide poisoning. Carbon monoxide poisoning will be covered in Chapter 20.

Cyanide poisoning should be considered if the assessment of the burn patient reveals intense air hunger, metabolic acidosis, or sudden cardiovascular collapse and the patient has been the victim of a confined space fire. Begin appropriate therapy such as hydroxocobalamin.

Circulation

Burn patients may have impaired cardiac output. The etiology of decreased cardiac output is unclear, but may be from a variety of causes, including high levels of circulating inflammatory mediators, carbon monoxide poisoning, or cyanide poisoning. Assessment of the burned patient should include an evaluation of cardiac output, capillary refill, neck vein distension, lung sounds, pulse rate, and blood pressure. Burn patients who have been victims of other trauma such as motor vehicle collisions or explosions may have myocardial contusions, cardiac tamponade, or abdominal or solid organ injuries. The CCTP should review the lab values to ensure that the hemoglobin level is adequate—a value of more than 10 g/dL is preferred for flight. Consider administering blood transfusions to patients with hemoglobin levels of less than 10 g/dL, patients with major burns, patients with significant comorbidities (such as coronary artery disease), or elderly patients.

Assessment of the patient's circulation should include evaluation of the extremities and digits for circumferential burns that have impaired circulation. Edema in the arms and legs may also impair circulation. Circumferential deep partial-thickness burns cause impairment of circulation because of their tough leathery nature. Impaired circulation may result in an absence of pulses, delayed capillary refill, or mottling and discoloration of the extremity. Early identification of circulatory impairment in the extremities is crucial to prevent tissue necrosis and amputation. Patients with circumferential burns and a lack of distal circulation should have an escharotomy performed if local scope of practice allows.

In the arms and legs, edema may also develop deep in compartments created by the tough nonelastic fascia that surrounds and separates the muscles. Edema can compress the blood vessels and nerves, which impairs blood flow and sensation and results in **compartment syndrome**. The symptoms of compartment syndrome are known as the Five Ps: Pain, Pallor, Paralysis, Paresthesias, and Pulselessness. The affected extremities may also be cold (poikilothermia, sometimes called the sixth P). Pulselessness is a late finding in compartment syndrome. Pain is considered the hallmark of compartment syndrome and is typically out of proportion to the injury or physical findings. Compartment syndrome therefore may be difficult to detect in burn patients because burns are expected to be painful. Patients with compartment syndrome undergo **fasciotomy**, a surgical incision of the tough and fibrous fasciae that separate the compartments to relieve the pressure. Fasciotomies are typically not performed by prehospital providers and should only be performed by properly trained personnel.

The assessment of a patient's circulation should also include determining the adequacy of fluid resuscitation in the burn patient. Fluid resuscitation in burn patients should be guided by formulas such as the Galveston and Parkland formulas, which are discussed later; however, in patients with significant comorbid conditions such as congestive heart failure or renal failure, the assessment of the patient's fluid status may play a greater role in guiding fluid replacement. Pulse rate, oxygen saturation, and lung sounds must all be considered in determining the patient's fluid balance. In patients with adequate renal function, urine output should be monitored closely to ensure an output of at least 50 mL/h (0.5 to 1.0 mL/kg/h) for the adult. Pulse rate is not a good indicator of the adequacy of fluid resuscitation and may remain elevated because of pain and the release of catecholamine. If **myoglobinuria** develops (an accumulation of myoglobin in the renal tubules), additional fluids should be given to reach a urine output of at least 100 mL/h. Myoglobinuria should be suspected if the urine becomes dark, brown, or bloody.

Signs and Symptoms

Compartment Syndrome

- The Five Ps: Pain, Pallor, Paralysis, Paresthesias, and Pulselessness
- The affected extremities may also be cold (poikilothermia)

Transport Management

Compartment Syndrome

- Keep the involved extremity elevated.
- Monitor pulses, sensation, and motor function.
- Do not place anything on the extremity that is compressive, such as splints or bandages.
- Monitor for increased pain (remember pain out of proportion is the hallmark of this syndrome).
- Provide supportive care (ie, maintain body temperature, analgesics, IV fluids, and oxygen).

Signs and Symptoms

Myoglobinuria

- Dark, brown, or bloody urine

Transport Management

Myoglobinuria

- Administer additional fluids so that urine output is at least 100 mL/h.

Focused History and Physical Exam

Disability

A brief neurologic exam is part of the initial assessment, but a more detailed neurologic assessment is necessary. Burn patients are generally awake and alert. Unconscious or confused patients may have other issues affecting their mental status, such as hypoxia, carbon monoxide poisoning, cyanide poisoning (from burning plastics), head injury, or substance abuse. Alcohol is commonly present in burn victims. Patients with carbon monoxide poisoning may exhibit subtle cognitive impairment that may not be detected during initial assessment. The CCTP therefore should be alert to changes in mental status. A patient with altered mental status should have aggressive support of the ABCs while the cause is investigated. The neurologic exam should include pupillary response to light and gross motor activity in the extremities. Patients with additional trauma (for example, injuries from explosions or motor vehicle collisions) should undergo c-spine immobilization until evaluated and cleared by radiograph, or clinical criteria such as the NEXUS or the Canadian c-spine rule, which are the clinical rules used in emergency departments throughout the country.

Exposure

A head-to-toe examination of the patient should be undertaken, noting the depth and size of the burns and other associated injuries. If the patient's wounds have been dressed prior to transport, within reason these should be removed to inspect the wounds and injuries (use your clinical judgment regarding the feasibility of doing this prior to transport). Care should be taken to identify and correct any inappropriate dressings, such as wet dressings. Assess the patient for hypothermia, and take precautions to prevent it.

History

In addition to the initial assessment and physical exam, a detailed history should be obtained. Information about the patient, the incident, and what treatment has been provided must be included, especially the amount of fluids delivered prior to arriving at the hospital and at the referring hospital. This is particularly important for patients who will be transported a long distance from home. If the patient is unconscious or becomes unconscious en route, vital information may not be available to the team that assumes care. The following information should be obtained:

- Patient information (SAMPLE)
  - Signs/symptoms
  - Allergies
  - Medications
  - Past medical and surgical history, and tetanus status
  - Last meal or drink
  - Events leading to injury
- Mechanism of injury
  - Source of burn
  - Closed space and duration of exposure
  - Chemical exposure
  - Related trauma (ie, fall, blast, motor vehicle crash)
- Prior interventions
  - Scene
    - Resuscitation required and fluid administered to patient
    - Field decontamination (ie, method of cooling, eye irrigation)
  - Hospital
    - Tetanus booster
    - Fluids given (amount and type)
    - Other treatments (ie, antibiotics, nebulized medications, diuretics, sedation, and pain medications)
    - Procedures (ie, central line placement, endoscopy, bronchoscopy, fasciotomy, and escharotomy)
- Bring copies of all records and radiographs from the transferring facility

Transport Management

General Burn Management

- Remove the patient from the source of the burn.
- Cool the burn within the first 2 minutes, and no longer than 20 to 30 minutes.

- Irrigate. This should not be done for longer than 30 minutes.
- Administer high-flow oxygen.
- Remove wet clothing, dressings, and linens.
- Provide early, aggressive, sustained fluid management; obtain IV access through unburned skin if possible.
- Consider using warmed IV fluids, especially if large volumes are required.
- Monitor the patient's overall perfusion status, mental status, urine output, and lung sounds.
- Leave blisters intact.
- Administer pain medication. Assess and treat for pain every 10 minutes until the patient is comfortable.
- Check fluid and infusion rates when accepting a transfer.
- Inventory the fluid given already.
- Reconcile urine output since injury.
- Empty the Foley bag at the time of assuming care.
- Keep the patient warm and place him or her in clean, dry linens prior to transport.

Management

Stopping the Burn

Removing the patient from the source of the burn is an obvious first step. Cooling is only effective if performed within the first 2 minutes and should not be continued longer than 20 to 30 minutes. Irrigation of a thermal burn that is older than 30 minutes has little to no benefit. Copious irrigation is indicated for chemical burns but may also be beneficial if molten metals, synthetic materials, or tar adhere to the skin. Irrigation dissipates heat and therefore should be continued until the burning has stopped and the material is cool **Figure 12-7**. In addition, cold inhibits lactate production and acidosis, thereby promoting catecholamine function and cardiovascular homeostasis in the wound. Cooling of the burn also limits the release of histamine, which results in increased vasculature permeability and **thromboxane**, which promotes coagulation in the wound contributing to ischemia. In chemical burns, the irrigation length should be guided by the pH of the wound assessed in the base of the wound. If the CCTP is unable to measure pH using standard litmus paper, copious irrigation should continue for at least 20 minutes. Blisters should be left intact unless they are the result of a chemical burn, in which case they should be broken because they may trap chemicals in the blisters. All clothing and jewelry should be removed because they may also trap chemicals. Wet clothing may contribute to heat loss and cause hypothermia. After cooling or decontamination, the patient should be kept warm and placed in clean, dry linens prior to transport.

Airway and Breathing

Airway management for the burn patient is a difficult challenge. Inhalation injury, c-spine injury, circumferential chest burns, and poisoning from noxious fumes are challenging scenarios. As stated previously, the patient must be assessed for the possibility of inhalation injury and the airway managed with endotracheal intubation if stridor, progressive hoarseness, or any other evidence of airway compromise is present. Because inhalation injuries result in airway edema, the CCTP should anticipate a difficult airway and be prepared to perform a surgical airway intervention should endotracheal intubation fail.

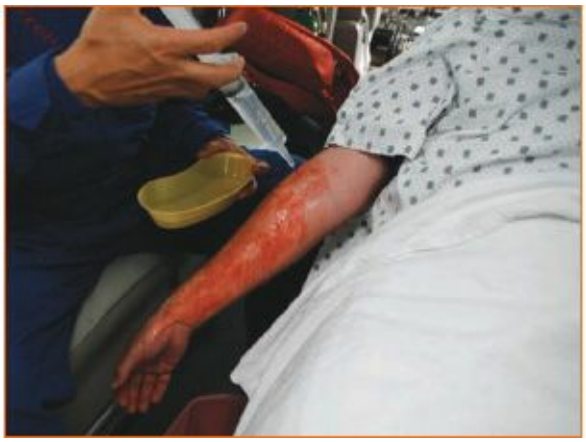


Figure 12-7 Management of the burn patient includes irrigation.

Signs and Symptoms

Inhalation Injury

- Facial burns
- Stridor or progressive hoarseness
- Singeing of the eyebrows and hairs
- Carbon deposits and acute inflammatory changes in the oropharynx
- Carbonaceous (black) sputum
- History of impaired mentation and/or confinement in a burning environment
- Explosion with burns to head and torso
- Circumferential neck burns
- Carboxyhemoglobin level greater than 10% if patient is involved in a confined space fire
- Soot around the nose and mouth
- Increased mucus production
- Impairment of mucociliary function

Transport Management

Inhalation Injury

- Consider performing endotracheal intubation after RSI if the transport time is prolonged; if there is stridor, progressive hoarseness, or any change in the patient's voice; or for any deterioration in the patient's condition.
- Be prepared to perform surgical intervention should intubation fail.
- Consider administering humidified oxygen (in the absence of wheezing) using a nebulizer with normal saline only and no bronchodilators.

For patients who require endotracheal intubation, RSI should be performed per local protocol.

If the patient's oxygen saturation drops at any point, you have no choice except to ventilate (slowly). If the patient is hemodynamically unstable, you must judge whether sedation is appropriate or whether the risk of profound hypotension is too great to sedate the patient.

Although succinylcholine is the drug of choice in RSI, it is contraindicated in patients with burn injuries greater than 48 hours old or in patients with known rhabdomyolysis secondary to their burn injuries because it may cause an extremely dangerous rise in potassium level. In these situations, a nondepolarizing agent such as vecuronium or rocuronium should be used. Because of the extended time of paralysis with this agent, the CCTP should carefully weigh the risk of being unable to intubate the patient with the benefit of intubation. Most often the benefits of intubation outweigh the risk associated with paralytic-assisted intubation with a nondepolarizing agent. The CCTP should also consider sedation-only assisted intubation (with a medication such as etomidate) or awake intubation as an alternative in a patient in whom the administration of succinylcholine is contraindicated.

Airway management in the burn patient can be difficult. Not only can airway edema make intubation difficult but it can foil most airway devices designed for use with failed intubation. Most of these devices rely on the ability to ventilate the patient through a patent glottic opening, which may not be present in the burn patient with inhalation injury. It is imperative therefore that the CCTP be prepared to surgically manage the airway if orotracheal intubation has failed.

Initial ventilator settings on the burn patient should be standard with the exception of the addition of positive end-expiratory pressure (PEEP) to aid in oxygenation and to treat pulmonary edema.

Carbon monoxide poisoning, asphyxiation, and cyanide poisoning are a major concern; most early fatalities are a result of carbon monoxide poisoning. Poisoning may be suspected from historical information such as burn injury in an enclosed space. An elevated carboxyhemoglobin level may be diagnosed via an arterial or venous blood gas determination or by using an oximeter capable of measuring carboxyhemoglobin (SpCO). Chapter 20 covers the treatment of carbon monoxide poisoning and cyanide poisoning.

Cyanide can be produced from the burning of any substance that contains carbon and nitrogen. The treatment for cyanide poisoning is described further in Chapter 20, but good supportive care in the absence of antidote can save many cyanide poisoning victims.

If any of these conditions are suspected, the patient should be treated with 100% oxygen via nonrebreathing mask. In most burn patients, oxygen, preferably humidified, should be administered immediately. High-flow oxygen may be used in patients with chronic pulmonary disease, but the patient's condition should be monitored closely for evidence of hypoventilation and the CCTP should be prepared to manage the airway. Patients who are known to have COPD and carbon dioxide retention should receive enough supplemental oxygen to maintain their baseline oxygenation or a saturation of 90% to 95%. Providing the patient supplemental oxygen prevents hypoventilation and respiratory acidosis, which occur with oxygen saturations that are above the patient's baseline in COPD. Attempts to oxygenate the patient to supra-baseline oxygenation levels (his or her normal saturation level) can result in carbon dioxide retention, acidosis, arrhythmias, and respiratory arrest; however, oxygen should never be withheld from a hypoxic patient.

Patients with inhalation injury from cyanide or carbon dioxide exposure require immediate intubation. Cricothyroidotomy should be performed in the event of an unsuccessful intubation because the use of airway rescue devices such as laryngeal mask airway, King, and Combitube are not likely to be successful secondary to airway edema. An unsuccessful intubation is defined as three attempts by the most experienced person.

Circulation

The essentials of circulatory management include early, aggressive, sustained fluid management. This management is unique to burn patients. Burns can result in large intravascular fluid losses as a result of the loss of the protection against desiccation and evaporation. In addition, circulating inflammatory mediators cause fluid from capillaries to leak into the interstitial space (neither in the vasculature nor the

cell). The inflammatory process at the site of injury results in plasma "weeping" into the burned area. The larger the surface area involved, the greater the fluid loss. Burns that are deep second degree or larger can cause hypotension and therefore fluid resuscitation should begin immediately via large-bore IV catheter. If possible, obtain IV access through unburned skin; however, access through burned skin is acceptable if necessary.

There are numerous fluid resuscitation formulas for the first 24 hours after a burn. The most commonly used is the **Parkland formula**, which recommends resuscitation with lactated Ringer's solution (normal saline is an acceptable substitute) of 4 mL/kg/%BSA burned; half this amount should be given over the first 8 hours (from the time of injury), one fourth of the total amount should be given over the second 8 hours, and one fourth of the total amount should be given over the third 8 hours. All calculations are determined from the time of the injury, and therefore fluid boluses may be necessary to correct deficits in fluid resuscitation becasue of either a delay in presentation or underresuscitation. It may also be necessary to administer fluids more aggressively if the patient is in shock with unstable vital signs. The goal for all fluid replacement is to maintain a urine output of 0.5 to

For children, the calculation of fluid resuscitation becomes a little more complex. The **Galveston formula** **Table 12-2** uses BSA burned and TBSA of the child (for maintenance fluids) as opposed to weight to calculate fluid needs. The Galveston formula is as follows: 5,000 mL/m^2 burned + 2,000 mL/TBSA m^2, with half of the amount of fluid given in the first 8 hours after injury and the second half over the next 16 hours. The problem with this approach, however, is that calculating m^2 burned while preparing to transport a critically burned child is not easily done, making this approach somewhat impractical. Therefore, for the acute phase and immediate transport, the Parkland formula is adequate until conditions allow the calculation of more precise fluid resuscitation rates.

The formulas used to calculate a burn patient's fluid requirements should serve only as a guide. The patient's overall perfusion status, mental status, urine output, and lung sounds should be monitored. If the patient is too aggressively hydrated, pulmonary or cerebral edema may result. If the patient is not adequately resuscitated, inadequate perfusion of the kidneys or mesentery may worsen organ system damage. The best monitor of fluid replacement is urine output with a goal of a minimum of 30 mL/h (0.5 mL/kg/h) for adults, 1 mL/kg/h in children, and 1 to 2 mL/kg/h for neonates. The urine output should serve to guide the subsequent fluid replacement rates beyond the initial 24 hours after injury. If urine output is above or below the normal range by one third for more than 3 hours, the hourly infusion rate is increased or decreased by one third. If the urine is dark brown or red, which means it contains hemochromogens, titrate the fluids to maintain a urine output of 1.0 to 2.0 mL/kg/h in adults and 2 mL/kg/h in children, until the pigments have cleared and the urine is straw colored. Burn centers often want to participate in the calculation of fluid requirements for children. Know the preference of the receiving facility or contact them prior to transport to discuss fluid resuscitation strategies. When assuming responsibility for a patient, the CCTP should check all fluids and infusion rates to make sure they are appropriate, inventory the fluid given already, and reconcile urine output since injury. It is a good idea for the CCTP to empty the Foley bag at the time you assume care to allow for accurate measurement of urine output during transport **Figure 12-8**.

TABLE 12-2 Galveston Resuscitation Formula

Lactated Ringer's solution (5% Dextrose in children < 2 years)	
First 24 hours	**Second 24 hours**
5,000 mL/m^2 burned +	3,750 mL/m^2 burned

2,000 mL/m^2 TBSA m^2	1,500 mL/m^2 TBSA
Provide half this amount in the first 8 hours, and the second half in the next 16 hours.	
Abbreviation: TBSA, total body surface area.	

Special Populations

Children are much more sensitive to fluid shifts than adults. The CCTP must be very careful with fluid administration in children, who have a much larger surface area relative to body mass and can lose body heat much more easily.

Wound Management and Dressings

Wound management is critical, especially when burn injury compromises the skin as a barrier to infection and regulator of the body's temperature. Proper dressings help reduce infection, decrease pain, and prevent heat loss. After cooling, irrigating, and decontaminating, the wounds should be dried and dressed with *dry*, clean dressings **Figure 12-9**. A common mistake when caring for burned patients is using wet dressings, which contribute to heat loss and are ineffective barriers to infection. Bulky dressings may be used to pad pressure points and sensitive areas. No ointments or creams are necessary, but if wounds are already dressed with ointments or creams they may be left in place. All wounds should be examined to confirm that the BSA and the fluid resuscitation calculation are accurate.

Figure 12-8 Empty the indwelling catheter bag at the time you assume care to allow for accurate measurement of urine output during transport.

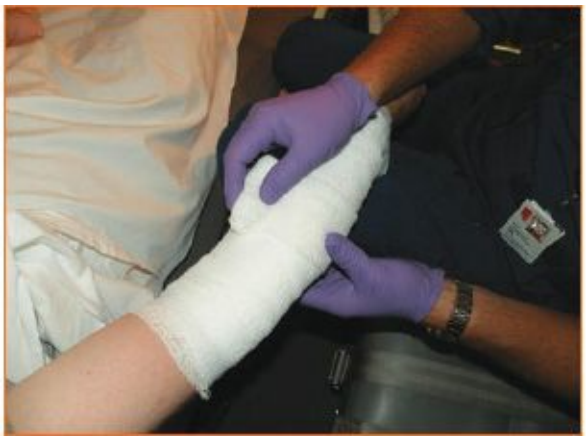


Figure 12-9 Wounds should be dressed with dry, clean dressings. Do not use wet dressings.

The debridement of most wounds will be performed at the receiving burn unit. As a general rule, blisters are left intact unless they are caused by a chemical burn. Blisters from chemical burns should be broken during decontamination as they may trap the causative agent and worsen the extent of injury.

Tar and asphalt burns can represent a challenge in the area of burn wound care. When tar that is heated to form a liquid comes in contact with skin, heat transfer occurs, causing a burn injury. Asphalt cements become a liquid at lower temperatures (275°F to 300°F) compared with tar, which requires a higher temperature to remain in a liquid state (450°F to 500°F). Therefore, more serious burns occur with tar than asphalt. As tar or asphalt cool, they solidify and adhere to hair. There is little direct bonding between the tar and skin. The tar or asphalt should be cooled with water until hardened and cool. After the tar is cooled, the patient may be transported to the burn center. If an attempt at removal is made prior to transfer to the burn unit, several ointments and solvents may assist in the removal process. Tar must be removed because it would serve as an occlusive dressing and cause infection. Naphthalene, hexane, and other carbon solvents may be useful for removing asphalts, whereas coal tars respond to aromatic hydrocarbons (benzene, toluene, or naphthalene) only. Unfortunately, these solvents can be absorbed, producing toxic side effects; therefore, a specific type called long-chain aliphatic hydrocarbons (hexanes or pentanes) should be used, or removal of coal tars may need to be delayed until reaching the burn center. Sunflower oil, butter, baby oil, and commercial surface active agents such as polysorbate 80 have been shown to be highly effective and inexpensive for tar and asphalt removal.

Transport Management

Burn Wounds

- Dry and dress wounds with dry, clean dressings.
- Use bulky dressings to pad pressure points and sensitive areas.
- Do not use ointments or creams, but if wounds are already dressed with ointments or creams, leave them in place.
- Examine all wounds to confirm that the body surface area and the fluid resuscitation calculation are accurate.

As a general rule, wound debridement should not be performed by the CCTP except in the setting of chemical exposure and blisters formed by chemical agents. Wounds must generally be debrided to remove dead skin, which can serve as a source for infection and prevent healthy tissue from repairing the burned area. Wound debridement consists of removing any dead or nonviable tissue from the wound. The treatment of blisters is somewhat controversial. On the one side, exposing a broken blister can result in local wound infection, and on the other side, fluid confined by necrotic skin can result in a closed-space infection. The debridement of burned skin is best left to the burn specialist.

Wound Infections and Treatment

Wound infections do not occur during the first few hours after a burn and therefore there is no role for antibiotic prophylaxis by the CCTP. Early antibiotic administration without proven infection may select for resistant bacteria, which makes fighting future infections difficult. Topical antibiotic preparations are generally used to dress wounds. Agents such as bacitracin or a combination preparation of neomycin

sulfate and polymyxin B sulfate (Neosporin) are generally used on the face and areas of thin skin, whereas silver sulfadiazine (Silvadene) is commonly used on other parts of the body. Silvadene should never be used on the face as it can cause skin discoloration. The exception to early wound infection is with tetanus. Patients who have not had a tetanus booster in 5 years require 0.5 mL of diphtheria and tetanus toxoids intramuscularly. Patients who have not had the primary series of three immunizations (more commonly found in immigrants or visitors from developing countries) will require tetanus immunoglobulin in addition to starting the primary immunization series. Patients at highest risk for tetanus are immigrants from developing countries and older patients who have not had a booster in many years.

Pain Control

Burns can be excruciatingly painful, and pain control in the burn patient must be taken seriously. An opioid such as morphine sulfate, in doses of 4 to 8 mg IV (0.1 mg/kg), is the agent of choice in patients who are not allergic, and may be repeated until adequate pain control is achieved. Alternative agents such as fentanyl (Sublimaze) or hydromorphone (Dilaudid) may also be used. Pain should be assessed and treated every 10 minutes until the patient is comfortable. Burn victims may require large doses of narcotics for adequate pain control, so if patients say they are in pain, the pain should be treated. Titration of narcotics should be based on the patient's perception of pain, with additional doses withheld in cases of altered level of consciousness, respiratory depression, or hypotension. The patient should be reassessed often and treated as needed. Morphine sulfate may also be used for children. An initial dose of 0.1 mg/kg up to a maximum of 5 mg/kg per dose should be given. Individual requirements for narcotics are different, and the CCTP should take care to ensure that the pain is not under-medicated or overmedicated.

CCTPs should also be careful not to confuse sedation and analgesia. Sedatives such as lorazepam (Ativan) do not have analgesic properties, and patients who are receiving ventilation will likely require sedation in addition to analgesia. The patient's condition should be monitored closely to prevent oversedation. The CCTP should remember that oxygenation does not equal ventilation, and patients who are oversedated will have a rise in the partial pressure of carbon dioxide long before there is any drop in the partial pressure of oxygen. Therefore, clinical assessment of the patient's ventilatory status or end-tidal carbon dioxide level must be used as the gauge to assess for oversedation. All medications should be given IV and never intramuscularly unless special circumstances exist. Long transports may require large quantities of medication, and the CCTP should make sure enough medication is available for sedation and pain control during transport.

Other Issues

Hypothermia

As mentioned earlier, every effort should be made to maintain a normal body temperature. The patient's temperature should be measured prior to transport. Removal of wet clothing, dressings, and linens is essential. Dry sheets and blankets are preferred. Warmed IV fluids should also be used, especially if large volumes are going to be infused.

Gastric Decompression

Patients with greater than 20% BSA burned are prone to ileus (loss of intestinal motility) because of the systemic inflammatory response that occurs with large burns. The administration of narcotic pain medications decreases gastric and intestinal motility and therefore increases the likelihood of an ileus developing. An ileus can cause abdominal distension, discomfort, and vomiting. Therefore, nasogastric tubes should be placed in these patients. Decompression of the stomach with low intermittent suction will

reduce distension, discomfort, and vomiting. Patients who have been endotracheally intubated also require orogastric tube placement to evacuate the stomach and prevent regurgitation.

Signs and Symptoms

Ileus

- Abdominal distention
- Discomfort
- Vomiting

Transport Management

Ileus

- Perform gastric decompression.
- Use nasogastric tubes.
- Place an orogastric tube in patients who have been intubated.

Figure 12-10 shows an algorithm for the management of a burn patient.

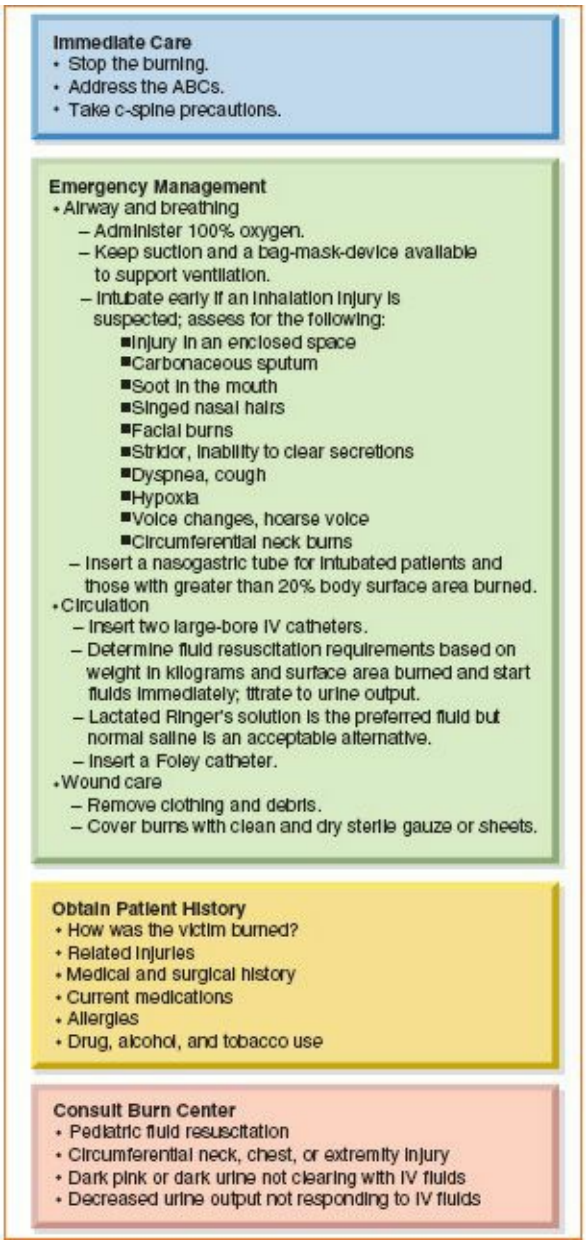


Figure 12-10 Algorithm for the management of a burn patient.

Special Situations

Burn centers provide care and expertise for many different types of injuries. A basic knowledge of these special situations is important to prevent further injury and provide appropriate treatment during transport.

Renal Failure and Rhabdomyolysis

Despite adequate fluid resuscitation, urine output may not be adequate. Given that urine output is the best indicator of adequate fluid resuscitation, patients with inadequate urine output can be extremely challenging for any health care provider. There are several common reasons for inadequate urine output. Severe shock may have caused kidney damage or the patient may have preexisting renal failure. The patient may also have renal dysfunction as a result of the mobilization of muscle proteins impairing filtration, a condition known as **rhabdomyolysis**. Rhabdomyolysis occurs most often with crush injuries, electrical burns, or large full-thickness burns. Specific proteins from the destruction of muscle may accumulate in the blood and urine as a result of tissue damage, causing the urine to become pink, dark red, or rust colored. This potential complication usually improves with appropriate fluid resuscitation, and

appropriate early resuscitation may prevent it from occurring. If urine output is inadequate or if there is urine discoloration that does not clear with the administration of IV fluids, increase the fluid rate, add sodium bicarbonate, and consider a diuretic such as mannitol. The dosage of mannitol and sodium bicarbonate varies somewhat based on physician preference, but typical orders are for three ampules of sodium bicarbonate in a liter of 5% dextrose, and 25 to 50 g of mannitol added to each liter of resuscitation fluid. For severe rhabdomyolysis, the patient may require emergent dialysis.

Patients with preexisting cardiac and renal dysfunction may develop pulmonary edema and congestive heart failure as a result of aggressive fluid resuscitation. In the absence of comorbidities, complications from fluid resuscitation are less likely than are complications from underresuscitation, and therefore it is better to err on the side of overresuscitation rather than underresuscitation. Patients may require ventilatory and inotropic support if fluid resuscitation is too aggressive. In addition to pulmonary edema, patients with renal failure are at increased risk of electrolyte abnormalities such as hyperkalemia. Potassium-containing fluids, including lactated Ringer's solution, should be avoided in the patient with renal failure or insufficiency and normal saline used instead. Always communicate closely with the burn center regarding fluid management in such patients.

Signs and Symptoms

Rhabdomyolysis

- Pink, dark red, or rust-colored urine that does not clear with IV fluids

Transport Management

Rhabdomyolysis

- Increase the fluid rate.
- Add sodium bicarbonate and consider a diuretic such as mannitol.
- When performing RSI, use a nondepolarizing agent such as vecuronium or rocuronium. Succinylcholine is contra–indicated in patients with known rhabdomyolysis secondary to burn injuries.
- Consider sedation-only assisted intubation with a medication such as etomidate or awake intubation as an alternative.
- Err on the side of fluid overresuscitation, but if fluid resuscitation is too aggressive, provide ventilatory and inotropic support.

Ocular Burns

Every burned patient should have a thorough eye exam. Corneal abrasions and corneal burns may occur from many mechanisms. Friction burns from auto airbags, blast injury, thermal burns, and chemical burns are common sources of injury. In awake and alert patients, eye injuries are rarely overlooked because the patient complains of pain or visual changes. If the patient is sedated, is unconscious, or has distracting injuries, clinical suspicion must remain high to avoid missing injuries. Because facial edema may develop quickly, early intervention is important. After the eyelids swell, it may be difficult to open the eyes for an exam or treatment **Figure 12-11**. Copious irrigation is indicated for chemical burns, and airbag injuries should also be irrigated. Airbags may cause friction burns because the bag impact deploys faster than the

eye can react and close to prevent the injury. In addition, the propellant that inflates the bag can release heat or chemicals that can burn the eyes. Corneal injuries can be very painful and should be treated with appropriate IV narcotics. Instillation of topical anesthetics such as tetracaine may alleviate the pain from corneal injuries and aid in irrigation, although topical anesthetic drops should not be used repeatedly.

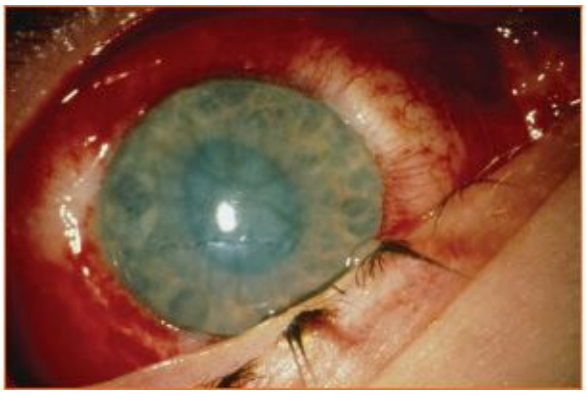


Figure 12-11 Examine the burn patient's eyes early; facial edema may develop quickly and make assessment difficult.

If chemical burns of the eyes are present, the eyes should be irrigated with copious amounts of water for at least 20 minutes or until the conjunctival sac pH has returned to 7. Irrigation of the eyes can be difficult at best if attempted manually. Morgan lenses—contact lenses with tubing connected to IV fluids—should be placed to facilitate irrigation. Lactated Ringer's solution is better tolerated as an irrigation fluid than normal saline, but either is acceptable. Topical anesthetics should be instilled prior to placement of the Morgan lenses.

For the critically burned patient with nonchemical burns to the eyes, application of topical ophthalmic preparations (if patient care demands allow) will suffice as temporizing treatment until more definitive examination and treatment at the receiving facility can be undertaken.

Transport Management

Ocular Burns

- Treat corneal injuries with appropriate IV narcotics.
- Chemical burns of the eyes: irrigate with copious amounts of water for at least 20 minutes or until the pH of the conjunctival sac has returned to 7.
- Nonchemical burns to the eyes: apply a topical ophthalmic preparation.

Facial Burns

Facial burns require special attention. Ocular and airway injuries are the major concerns and significant facial burns suggest airway injury. Rapid and repeated airway evaluation is crucial. Early intubation should be considered for any airway injury. As mentioned earlier, the face develops edema quickly. Therefore, an early eye exam is important. To help reduce facial edema, the head of the stretcher should be elevated 30° if spinal injury is not suspected.

Transport Management

Facial Burns

- If no spinal injury is suspected, elevate the head of the stretcher 30° to reduce facial edema.

Ear Burns

Ears are also prone to significant swelling. The ear canal and ear drum should be examined before edema develops. Discuss this with the transporting or receiving physician prior to departure. Prevent additional trauma by avoiding pressure dressings and do not use a pillow. Only large, dry, bulky dressings are required.

Transport Management

Ear Burns

- Use large, bulky dressings.
- Avoid pressure dressings.
- Do not use a pillow.

Circumferential Burns and Compartment Syndrome

Burns that encircle the chest, an extremity, or the penis present a difficult challenge. Chest burns can cause respiratory impairment because of eschar limitation of chest excursion. Edema in the extremities and penis may result in vascular compromise. These injuries may require an **escharotomy**, a procedure in which an incision is made through the stiff edematous burned skin to restore mobility to the chest or circulation to an extremity **Figure 12-12**. Again, most burn specialists discourage the use of field escharotomy unless absolutely and immediately necessary to preserve life or limb.

The equipment needed to perform an escharotomy includes the following:

- Scalpel
- Pain medication
- Sedation
- Sterile dressing
- 4″ × 4″ gauze pads

An escharotomy may be necessary in the following circumstances:

- Circumferential burns
- Full-thickness burns
- Compartment syndrome

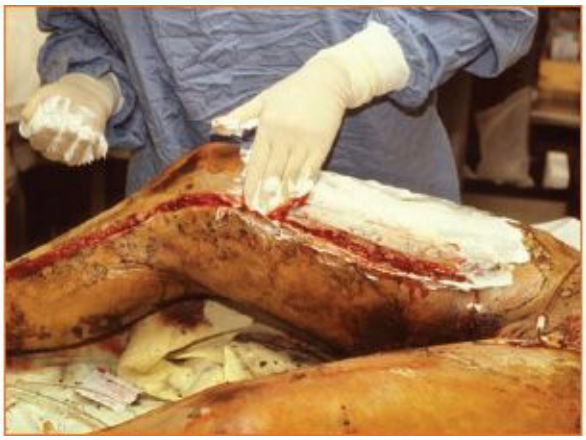


Figure 12-12 An escharotomy, in which an incision is made to restore circulation in the context of a circumferential extremity burn.

- Thermal burns
- High-voltage electrical burns
- Tissue pressure measurements greater than 35 mm Hg (obtained by placing a needle into the compartment in question, usually done by an orthopaedist)
- Complications of an escharotomy include:
- Bleeding
- Infection
- Nerve damage
- Limited range of motion due to improper technique

Skill Drill 12-1 describes the steps for performing an emergency escharotomy:

1. Prepare your equipment.
2. Administer sedation and pain medications. Burns that cause such injuries are deep and destroy nerve endings; therefore, this procedure can be performed without anesthesia.
3. Maintain an aseptic technique **Step 1**.
4. Determine a well-defined incision pattern. For the chest, an incision is made along both anterior axillary lines and may also be performed transversely. For an extremity, an incision is made parallel to the bone. A single incision may be sufficient for an extremity. The incision should be of sufficient depth for an obvious release in pressure on the skin and for fat to bulge through the incision. For the penis, a single dorsal incision is adequate (the dorsum of the penis is the anterior surface if the penis is pointing toward the feet). Hand escharotomies are performed with incisions in the medial and lateral surfaces of each finger and of the palm.
5. Incise the derma of the burned tissue **Step 2**. Built-up pressure should cause the tissue to open even more.
6. Prepare to manage bleeding **Step 3**.
7. Apply a sterile dressing over the incision.

The most qualified person should perform the procedure; however, situations may arise that make it necessary on an emergent basis to save life or limb. An escharotomy should only be performed after

discussion with the receiving burn center or transport service medical direction (if local scope of practice allows).

Signs and Symptoms

Circulation Problems Due to Circumferential Burns

- Absence of pulses
- Delayed capillary refill
- Mottling and discoloration of an extremity

Transport Management

Circulation Problems Due to Circumferential Burns

- Escharotomy, if allowed per protocol

Controversies

The necessity of prehospital escharotomies is a subject of debate. Many believe that the efficacy of field escharotomies is limited when given a risk-benefit analysis. With field escharotomies, there is the potential for blood loss, severe hypotension, contamination of the wound, and damage to the nerves. Because of the amount of time it takes for an eschar to form, the likelihood of needing to perform an escharotomy in the field is low. However, one must also consider potential benefit of performing escharotomies for interfacility transports of extended length. When performed in a facility as opposed to the field, the benefit may outweigh the risk of the procedure.

Hand and Foot Burns

Burns on the hands and feet can cause significant disability and require specialized care. Circulation is the main concern because edema may impair blood flow to the periphery of the extremity, causing extensive damage. Simple interventions such as elevating the injured extremity above the heart and avoiding constricting dressings are important. Loose, dry dressings that allow easy assessment of the patient's circulation may be used during transport; however, no dressing is required. Do not apply creams or ointments. Do not apply ice packs because they may cause frostbite.

Transport Management

Hand and Foot Burns

- Elevate the injured extremity above the heart.
- Avoid constricting dressings; use loose, dry dressings or no dressing.
- Do not apply creams or ointments.
- Do not apply ice packs.

Genitalia Burns

Burns on the genitalia and perineum should not distract health care providers from life-threatening injuries. However, certain details in the treatment of patients with such injuries require prompt attention. A Foley catheter should be placed immediately before edema develops. The penis should be examined for circumferential burns and monitored for impaired circulation. As mentioned previously, a dorsal escharotomy of the penis may be necessary, but is highly unlikely.

Transport Management

Genitalia Burns

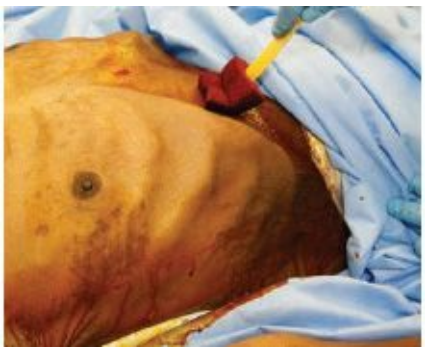
- Place a Foley catheter immediately.
- If a penis has a circumferential burn, monitor for impaired circulation.

Pediatric Burns and Child Abuse

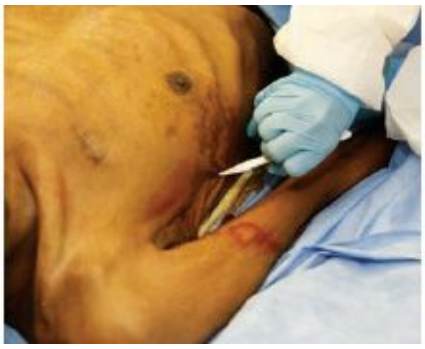
Children present special challenges when burned because their physiology and composition are different than those of adults. Children have relatively more surface area per kilogram, which necessitates higher rates of fluid resuscitation. Infants also have relatively less glycogen stores than adults, making them susceptible to hypoglycemia if deprived of oral intake for several hours. Consideration should be given to the addition of glucose to resuscitation fluids; however, this treatment should be guided by local burn center preference. Some strategies include frequently checking glucose levels and correcting any hypoglycemia as it occurs rather than administering glucose-containing fluids. The blood glucose level should be maintained between 60 and 120 mg/dL.

Skill Drill 12-1

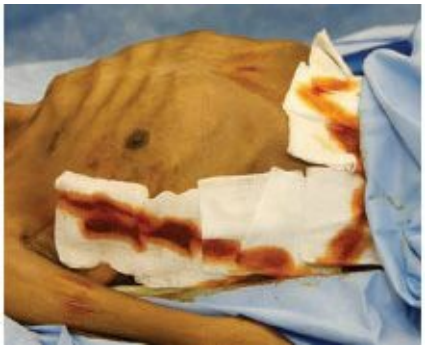
Performing an Emergency Escharotomy



1 Prepare your equipment. Administer sedation and pain medications. Maintain an aseptic technique. Determine a welldefined incision pattern.



2 Incise the derma of the burned tissue.



3 Prepare to manage bleeding. Apply a sterile dressing.

Approximately 25% of all childhood burns are from child abuse and therefore should be considered by the CCTP caring for the child. Children with burns who receive care more than 24 hours after the incident should be suspected of being maltreated and should be reported to child protection authorities. Accidental burns in children most often result from scalding and more commonly occur in the "spill area"—the area of the face and chest that is exposed from a child pulling a container of hot liquid onto himself **Figure 12-13**. Burns to other areas with well-demarcated edges and in a stocking glove distribution should raise the suspicion of abuse **Figure 12-14**. Any suspicion of child maltreatment should be reported to child protection authorities and the receiving facility.

Transport Management

Pediatric Burns

- Consult with a local burn center regarding adding glucose to resuscitation fluids.
- Check blood glucose levels frequently.
- Correct hypoglycemia.

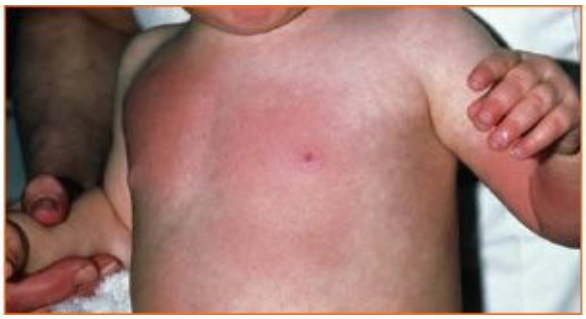


Figure 12-13 An accidental burn.

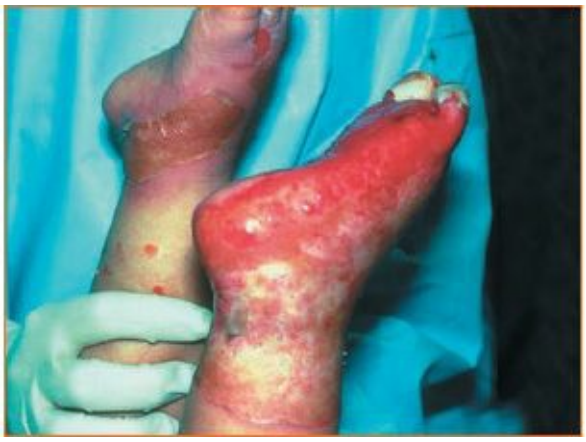


Figure 12-14 Stocking glove distribution.

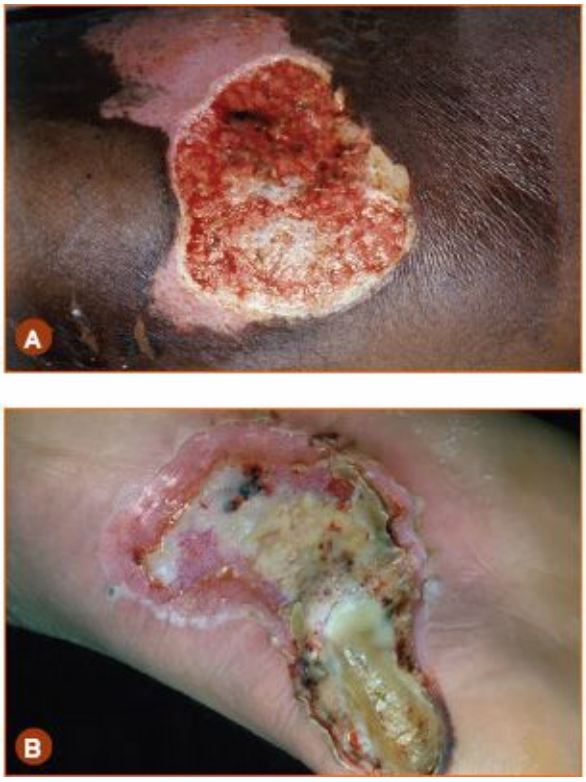


Figure 12-15 **A.** An electrical burn entry wound on the shoulder of a patient. **B.** An electrical burn exit wound on the foot of a patient.

Electrical Burns

Injuries from electricity may result from an alternating current or direct current such as lightning. An

alternating current is found in homes and businesses and is the most common source of electrical injury. A direct current is found in automobile electric systems and some lighting systems, and may be found in some industries. Lightning is also direct current.

Electricity releases heat as it travels through or across the body, causing extensive tissue damage. All tissues have a different resistance to current flow, with the nervous system being the least resistant. Bone is the most resistant, but all tissue is susceptible to damage. Electricity may enter and exit the body at any point. The hands are the most common point of entry **Figure 12-15A**, and the feet are the most common point of exit **Figure 12-15B**. The path electricity follows between entrance and exit is erratic and unpredictable. Severe charring frequently occurs at the point of entry and this may be the only obvious injury. Damage to deeper structures may not be evident if the superficial structure such as the skin is uninjured.

Early evaluation of the extent of electrical injury is difficult. There are common patterns of injury, however. Damage to the cardiac conduction system may present as cardiac arrest, and high voltages may lead to respiratory arrest by paralyzing the diaphragm. Alternating current typically causes ventricular fibrillation and is the most common cause of death. Direct current typically causes asystole. Cardiac arrest in the electrocuted victim is treated with standard ACLS protocol. Other arrhythmias may develop at any time after injury, necessitating continuous cardiac monitoring for the first 24 hours because a normal electrocardiogram or rhythm does not rule out injury. Skin burns sometimes occur without deeper structures being damaged. Clothing may ignite without current entering the body. These injuries are treated as other cutaneous burns. Because the electrical current can travel deep to the skin, deep structures such as muscle, bone, tendon, blood vessels, and nerves may be severely damaged, with little evidence on the skin. Massive muscle damage and cell death will release muscle proteins into the bloodstream. Untreated rhabdomyolysis can cause kidney damage and should be treated with increased IV fluids to maintain adequate urine output. As mentioned previously, rhabdomyolysis should be suspected with certain mechanisms of injury or by the development of pink or dark urine. The addition of sodium bicarbonate or mannitol may be necessary to maintain urine output and should be discussed with the burn center or medical direction. Compartment syndrome may develop in the extremities as a result of deep burns. Progressive pain, paresthesias, and decreased circulation are clues to this injury. Consultation with the transferring and receiving physician about fasciotomy should occur before transport. Electric current may also cause violent muscle contractions that break bones. The cervical spine and suspected fractures should be immobilized prior to transport. Care should be taken to protect open fractures from additional contamination.

Lightning injuries are a common source of burns. Patients with lightning injuries may experience any of the injuries already mentioned. They may also experience extensive superficial cutaneous burns in a fern or reticular pattern as electricity passes across the skin **Figure 12-16**. The ear drum may rupture as a result of the pressure wave created by the rapid heating of the surrounding air, and the patient may have displacement to the lens of the eye from the pressure gradient produced.

Chemical Burns

Common household, industrial, and farm products can be toxic and cause significant burns. The active ingredients responsible for injury are usually acids, alkalis, and a few other organic compounds. The extent of damage depends on the type of agent, its relative strength, and the duration of contact. It is crucial to decontaminate patients quickly and thoroughly. Chemicals can settle in clothing, shoes, linens, and jewelry. All patients should be completely disrobed and irrigated with copious amounts of water. Attempts to neutralize one chemical with another can produce an exothermic reaction (producing heat) and worsen the burn; therefore, this should not be attempted. After decontamination, the patient should be

placed on clean, dry linens. Cutaneous burns are not the only risk from chemical exposure because some chemicals may be absorbed systemically, causing organ damage or failure. Injuries may not be apparent for 6 to 24 hours after exposure. Patients with relatively minor-appearing injuries may require transport for observation at a center for specialized care.

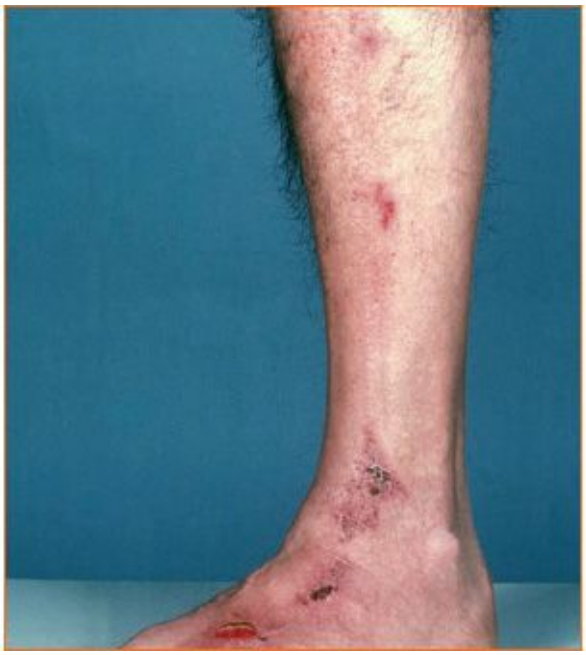


Figure 12-16 Fern pattern as a result of a lightning injury.

Transport Management

Electrical Burns

- Treat the same as other cutaneous burns.

Acids are found in drain opener, drain cleaner, bathroom cleaner, swimming pool products, and rust remover. They are also found in hair care products such as hair relaxer. Acids are used extensively in industry for production of other compounds and are found in potent industrial cleaning products. Duration of contact is the main determinant of damage from acids. Rapid, copious irrigation with water is the initial treatment. Acids tend to cause burns that are more self-limiting than alkalis. When acids come in contact with skin, the damage caused is in the form of coagulation necrosis. The coagulum, or scar, limits the penetration of the acid, and thereby limits the damage done. This results in a burn with well-demarcated edges and often in a pattern that suggests liquid spilling. Some acids such as hydrofluoric acid also have other mechanisms of injury and require special attention. Hydrofluoric acid is used in microelectronics, petroleum processing, glass and metal etching, wheel cleaners, rust removal, and many other applications. Hydrofluoric acid is capable of progressive injury despite irrigation. After penetrating the skin, fluoride ions are released and cause nerve damage and severe pain. Calcium binds to fluoride and reduces damage. Therefore, topical calcium gluconate gel should be applied immediately after irrigation. There are commercial preparations of calcium gluconate gels, but mixing calcium gluconate with a water-based, water-soluble lubricant (such as K-Y Jelly) will suffice if commercial preparations are not available. This mixture can be squirted into an exam glove and applied directly to the patient's hand. Intradermal injection of calcium gluconate and intra-arterial calcium gluconate may also be used to alleviate pain. Oxalic acid is not as toxic as hydrofluoric acid but may also require calcium gluconate

therapy. Calcium *chloride* should not be administered intradermally or intra-arterially.

Alkalis are found in many household and industrial products and are capable of producing significant burns. Oven cleaner, drain cleaner, lime, lye, and cement are common alkalis. These agents, when combined with fat, produce soap, and therefore penetrate deeply and rapidly into the tissues. They continue to burn for extended periods of time and require irrigation for more than 20 minutes. Because there is no coagulum to restrict the penetration of alkalis into the tissues, burns caused by alkalis typically have poorly defined edges and diffuse spreading of the injury. Special care must be taken during decontamination to avoid unnecessary heat loss. Because of deep penetration, systemic toxicity is also a significant concern. There are no antidotes. Care of systemic complications is supportive.

Dry lime is a common agricultural product. When combined with water it may also generate an exothermic reaction and cause burns. All dry powders should be brushed away to reduce the amount of chemical before irrigation is performed with flowing water. Fast-flowing water will help dissipate heat, but the higher pressure may cause or worsen tissue damage. When you are using water under pressure, avoid self-contamination from splashing.

Hydrocarbons such as petroleum fuels may cause burns after immersion or prolonged contact. They can also cause significant systemic toxicity if absorbed. Treatment is supportive with copious irrigation and observation. Hydrocarbons (most petroleum products such as gasoline) can cause myocardial depression leading to hypotension and cardiovascular collapse. Halogenated hydrocarbons (chlorofluorocarbons or Freon), such as those used in propellants, are known to cause central nervous system depression or excitability. In particular, the halogenated hydrocarbons (Freon or many solvents like oven cleaner) are best known for potentially creating a fatal arrhythmia.

Transport Management

Chemical Burns

- Decontaminate patients quickly and thoroughly.
- Copiously irrigate until the burning has stopped or the material is cool.
- Irrigation length should be guided by the pH of the wound assessed in the base of the wound.
- If you are unable to measure pH, continue copious irrigation for at least 20 minutes.
- After decontamination, place the patient on clean, dry linens.
- Break blisters.
- Remove all clothing and jewelry.
- Do not attempt to neutralize one chemical with another.
- For burns from hydrofluoric acid, apply topical calcium gluconate gel immediately after irrigation.

After decontamination, management of chemical wounds is the same as for thermal injuries with the exception of applying calcium gluconate gel to hydrofluoric acid burns. Clean, dry dressings are the only requirement.

Toxic Epidermal Necrolysis Syndrome and Stevens-Johnson Syndrome

Toxic epidermal necrolysis syndrome is the most severe form of skin reaction to certain medications, environmental allergies, and other unknown toxins. The medication phenytoin (Dilantin), among others, is well known for causing this reaction. The milder form is called Stevens-Johnson syndrome, which is believed to be an immune-mediated illness causing sloughing of the skin, mucous membranes, and the

cells lining the respiratory system. Stevens-Johnson syndrome and toxic epidermal necrolysis can present respiratory and wound care challenges that are as difficult to manage as severe burns of other types **Figure 12-17**.

The term Stevens-Johnson syndrome is used in conjunction with toxic epidermal necrolysis. The spectrum from Stevens-Johnson syndrome to toxic epidermal necrolysis is as follows:

- In Stevens-Johnson syndrome, epidermal detachment involves less than 10% of the total body skin area.
- Transitional Stevens-Johnson syndrome-toxic epidermal necrolysis is defined by an epidermal detachment between 10% and 30%.

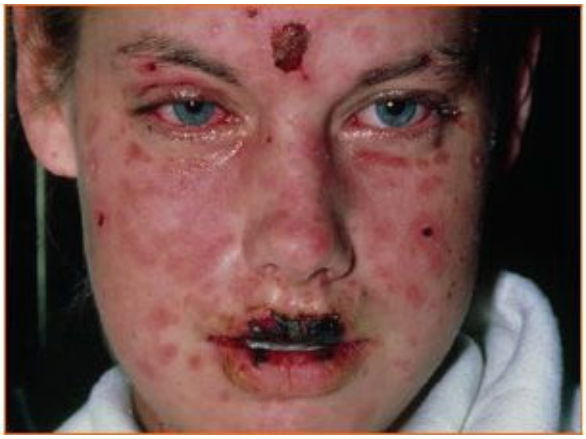


Figure 12-17 Patients with toxic epidermal necrolysis or Stevens-Johnson syndrome can present management challenges that are similar to those presented by patients with severe burns.

- Toxic epidermal necrolysis is defined by an epidermal detachment greater than 30%.

Patients with toxic epidermal necrolysis are frequently managed in burn centers because their wounds are analogous to burn injuries. Treatment is primarily supportive in addition to the skin care.

Summary

Mastering the care of burn patients is a continuous process. Research is ongoing to discover optimal wound management and resuscitation techniques. Additional training is available from various sources. Most burn centers and units also have education programs that are an invaluable resource to local providers.

Case Study

WHILE IN FLIGHT TO A RURAL COMMUNITY HOSPITAL for an interfacility transport, your MedEvac unit is diverted to a scene to care for a victim of a house explosion. While flying to the scene, your communication center advises you of the ground EMS report that states the victim is a 54-year-old woman who is experiencing dyspnea and pain.

During your final approach to the landing zone, you observe a house fully engulfed by flames. As you enter the awaiting ground ambulance to begin your assessment, you find a woman with multiple partial- and full-thickness burns to her arms, chest, abdomen, and face. She is experiencing severe dyspnea and crying out in pain. She is conscious, confused, and difficult to understand because of the hoarseness of her voice. You continue your assessment, while your partner sets up equipment for RSI. The patient is complaining of severe pain throughout the burned areas of her body.

Her vital signs include a blood pressure of 160/100 mm Hg; respirations, 38 breaths/min and labored with audible stridor; and a pulse rate of 150 beats/min. You note singed eyebrows and nasal hairs; soot around the nose and mouth; partial-thickness burns to the chest, abdomen, and arms; and full-thickness burns to the face. You estimate her TBSA of burns to be approximately 30%.

Your partner advises you that he is ready for RSI. You use an established IV line to administer 0.3 mg/kg of etomidate and 2 mg/kg of succinylcholine. You insert the laryngoscope and attempt to visualize the vocal cords; you are unable to visualize the vocal cords or pass the endotracheal tube because of edema. You direct your partner to ventilate the patient using a bag-mask device and 100% oxygen while you prepare the surgical airway kit. You are successful at securing a surgical airway. Upon confirmation of placement, you note decreased breath sounds on the right side as compared to the left side. You quickly cover all burns with dry, sterile dressings and prepare for transport by placing the patient onto your equipment and rapidly performing a hot load (with the engine running and rotors turning) for transport to the closest burn center.

In flight, your patient's vital signs remain stable; you administer 0.1 mg/kg of vecuronium and 1 μg/kg of fentanyl, and you start administering lactated Ringer's solution at 100 mL/h. Approximately 2 minutes from the burn center, your partner, who is ventilating the patient, reports that he is meeting resistance. You assess lung sounds and do not hear anything on the right although you are uncertain because of the amount of noise in the helicopter. Because of the burns and swelling, you are unable to determine whether jugular vein distension or tracheal deviation is present. You confirm that the surgical airway has not moved and then decide to do a needle decompression, which your partner reports makes ventilating much easier.

On arrival at the burn center, you do a hot off-load of the patient and transport the patient to the trauma bay, where you give a report to the trauma team. You and your partner clean your equipment and get the aircraft back in service. Once you return to base, you complete your report for the medical record and discover that you miscalculated the initial IV fluid rate according to the Parkland formula. You notify the nursing staff at the burn unit of the error and complete a protocol deviation form. Finally, you notify the referring EMS agency of the outcome.

1. What should the IV fluid rate have been?
2. What is the rationale for the resistance met during ventilation that led to a needle decompression?
3. According to the American Burn Association, what classification of burns did the patient in the case present?

Analysis

Although there are numerous fluid resuscitation formulas for the first 24 hours after a burn, the most commonly used method is the Parkland formula, which recommends resuscitation with lactated Ringer's solution of 4 mL/kg/% BSA burned. In this case, the initial fluid rate should have been 750 mL/h.

Burn patients may experience significant trauma from explosions or motor vehicle collisions resulting in fires. With the presence of such trauma, the possibility exists that you may overlook conditions such as a pneumothorax or hemothorax while treating the obvious burns. Careful repeated examinations should be aimed at identifying these conditions.

This patient met several criteria for major burn injuries, including partial-thickness burns involving more than 25% of BSA in an adult, burns involving the face, and burns complicated by inhalation injury or major trauma.

Caring for victims of burn injuries requires the CCTP to deploy aggressive critical thinking, assessment, and intervention skills. The CCTP must use an organized, rapid approach to assess,

prioritize, and treat both the burns and any secondary injuries found. Because of the early fluid losses associated with burn injuries, aggressive and sustained fluid management is essential. Determining the severity and extent of burns is important in order to arrange for transportation to the most appropriate treatment facility.

Prep Kit

Ready for Review

- The initial 24 hours of care after a burn are crucial. The CCTP must have an understanding of the common complications of burns that can develop during the transport of the patient to a burn facility.
- The skin, composed of the epidermis and the dermis, is the largest organ of the body and is vital to survival.
- The layers of the epidermis contain pigment cells, immune cells, and capillaries in the lower layers. The epidermis is the first barrier to injury and infection and is constantly repairing itself, as it is injured more often than the other layers, and the outer layer provides a barrier to prevent water loss from deeper structures.
- The dermis lies beneath the epidermis and contains fibroblasts, macrophages, white blood cells, nerve cells, lymphatic vessels, blood vessels, portions of the hair follicles, and oil and sweat glands.
- Proteins such as collagen and fibronectin, which are produced in the dermis, support the continuously reproducing epidermis and play a major role in wound healing, along with fibroblasts, macrophages, platelets, and endothelial cells. The inflammatory response, which is important in the burn healing process, is also responsible for multiple complications.
- The skin has multiple functions, which include protection from infection, maintaining fluid and electrolyte balance, thermoregulation, and neurosensory functioning, which is important for social interaction.
- Burns occur from a variety of mechanisms, including flame/flash, scald (water, grease, etc), contact, electrical, chemical inhalation, radiation, or any thermal source. The size and depth of a burn are determined by the agent, temperature, and duration of exposure.
- The zone of coagulation is the most damaged area of a burn and is the area of skin that came into direct contact with the source of the heat. The zone of stasis has injured tissue and may appear red and hyperemic (filled with an increased amount of blood) initially, but within 24 hours has a stagnant blood flow resulting in ischemic tissues, which by the third day becomes white because it is avascular and necrotic. The zone of erythema, which has increased blood flow, is the outermost area surrounding the zone of stasis, has minimal damage, and will recover.
- Burns of greater than 25% TBSA result in a systemic inflammatory response, which can result in undesirable effects on other tissues and organs, including pulmonary edema, suppression of the immune system, sepsis, and impaired cardiac output.
- The severity of a burn depends on the extent, depth, and location of the burn; the age of the patient; the etiologic agents involved; the presence of inhalation injury; and coexisting injuries or preexisting illnesses.
- The classification of burns includes superficial or first-degree burns, which involve only the epidermis; partial-thickness or second-degree burns, which involve some of the dermis but not the entire thickness of the skin; full-thickness or third-degree burns, which involve all layers of the skin

and perhaps some subcutaneous tissue and muscle; and subdermal or fourth-degree burns, which involve deep structures such as muscle, bone, large blood vessels, and nerves.

- Burn size is also a determining factor of burn severity. A common factor to determine severity is the rule of nines, which is the most common method in the prehospital environment.
- Assessment for burn patients should be similar to any other patient, with attention paid to each organ system, but with special attention paid to the patient's airway, breathing rate and depth, and decreased cardiac output.
- Burn management includes removing the patient from the source of the burn, cooling within the first 2 minutes but not for longer than 20 to 30 minutes, and irrigation but not for longer than 30 minutes.
- Airway management for a burn patient can present difficult challenges because of airway edema resulting from inhalation injuries.
- Aggressive and sustained fluid management is important, because burns can result in large intravascular fluid losses due to the loss of the protection against desiccation and evaporation.
- Wounds should be dressed with clean, dry dressings (never wet dressings), and blisters should be left intact. In general, wound debridement should not be performed by the CCTP.
- Burn victims may require large doses of narcotics for adequate pain control, and pain should be assessed and treated every 10 minutes until the patient is comfortable.
- Other issues of burn management include maintaining a normal body temperature in order to prevent hypothermia and performing gastric decompression.
- Renal dysfunction and rhabdomyolysis should be managed by an increase in the fluid rate.
- Ocular burns can occur from many different sources, including friction burns from auto airbags, blast injury, thermal burns, and chemical burns. A burn patient's eyes need to be examined early, because edema can develop quickly and make assessment difficult. Corneal injuries should be treated with appropriate intravenous narcotics, chemical burns of the eye should be treated with irrigation, and nonchemical eye burns should be treated with a topical ophthalmic preparation.
- Ocular and airway injuries are the major concerns of facial burns, and airway evaluation is important, knowing that early intubation may be necessary.
- Ear burns are prone to increased swelling, and an exam should be done before edema results.
- Circumferential burns (burns that encircle the chest, an extremity, or the penis) and the resultant compartment syndrome present a difficult challenge. These injuries may require an escharotomy, a procedure in which an incision is made through the stiff edematous burned skin, in order to restore mobility to the chest or circulation to an extremity as a result of a circumferential burn.
- Hand and foot burns can cause significant disability and should be treated by elevating the injured extremity above the heart and by using loose, dry dressings or no dressing.
- Genitalia burns require placement of a Foley catheter before edema develops, and if a penis has a circumferential burn, the CCTP should monitor for impaired circulation.
- Approximately 25% of all childhood burns are from child maltreatment, and the CCTP should consider this when caring for the burned child. Accidental burns in children often result from scalding and often occur in the "spill area" or the area of the face and chest that is exposed from a child pulling a container of hot liquids onto himself or herself.
- Early evaluation of the extent of injury from an electric burn is difficult. Damage to the cardiac conduction system may present as cardiac arrest, and high voltages may lead to respiratory arrest by

paralyzing the diaphragm. Alternating current typically causes ventricular fibrillation and is the most common cause of death, whereas direct current typically causes asystole. Lightning can cause extensive superficial burns in a fern pattern as electricity passes across the skin.

- Patients with chemical burns should be decontaminated quickly and thoroughly, irrigating until the burning has stopped and the material is cooled, or for at least 20 minutes, taking care to avoid unnecessary heat loss.
- Toxic epidermal necrolysis syndrome, the most severe form of skin reaction to certain medications, environmental allergies, or other unknown toxins, and a milder form, Steven-Johnson syndrome, can present management challenges similar to those presented by severe burn patients.

Vital Vocabulary

compartment syndrome A condition that develops when edema and swelling result in increased pressure within soft tissues, causing circulation to be compromised, possibly resulting in tissue necrosis.

deep partial-thickness burn A burn in which the skin is blistered but not charred and is painful to the touch, usually the result of steam, oil, or flames and involving the deeper layers of the dermis.

eschar The leathery covering of a burn injury, formed after the burned tissues dry out.

escharotomy A surgical incision in an eschar to lessen constriction; sometimes necessary (although rarely performed in a prehospital setting) to prevent edema from building up, impairing capillary filling, and causing ischemia.

fasciotomy A surgical incision into an area of fascia, for example to relieve pressure between two compartments; not usually performed in a prehospital setting.

full-thickness burn A burn that extends through the epidermis and dermis into the subcutaneous tissues beneath, in which skin is pale, painless, leathery, and charred; also called a third-degree burn.

Galveston formula A formula used to calculate fluid resuscitation for children, which takes into consideration the BSA burned and the TBSA of the child, with half of the amount given in the first 8 hours after injury and the second half over the next 16 hours.

hyperemic An increase in blood flow into a tissue or organ; congested with blood.

Lund and Browder chart A detailed version of the rule of nines chart that takes into consideration the changes in body surface area brought on by growth.

major burn A classification from the American Burn Association that includes: (1) partial-thickness burns involving more than 25% of BSA in adults or 20% of BSA in children younger than 10 years and adults older than 50 years; (2) full-thickness burns involving more than 10% of BSA; (3) burns involving the face, eyes, ears, hands, feet, or perineum that may result in functional or cosmetic impairment; (4) burns caused by caustic chemical agents; (5) high-voltage electrical injury; (6) burns complicated by inhalation injury or major trauma; and (7) burns sustained by high-risk patients.

minor burn A classification from the American Burn Association that includes: (1) partial-thickness burns involving less than 15% of BSA in adults or 10% of BSA in children and older adults; and (2) full-thickness burns involving less than 2% BSA that do not present a serious threat of functional or cosmetic risk to the eyes, ears, face, hands, feet, or perineum.

moderate burn A classification from the American Burn Association that includes: (1) partial-thickness burns of 15% to 25% of BSA in adults or over 10% to 20% of BSA in children and older adults; and

(2) full-thickness burns involving 2% to 10% of BSA that do not present a serious threat to functional or cosmetic impairment of the eyes, ears, face, hands, feet, or perineum.

myoglobinuria The presence of myoglobin, a respiratory pigment of muscle tissue, in the urine.

Parkland formula A formula that recommends giving 4 mL of lactated Ringer's solution or normal saline for each kilogram of body weight, multiplied by the percentage of body surface area burned; sometimes used to calculate fluid needs during lengthy transport times.

partial-thickness burn A burn that involves the epidermis and part of the dermis, characterized by pain and blistering; also called a second-degree burn.

rhabdomyolysis The destruction of muscle tissue leading to a release of potassium and myoglobin, which then accumulate in the blood and urine and impair filtration; occurs most often with crush injuries, electrical burns, or large full-thickness burns. The urine becomes pink, dark red, or rust colored.

rule of nines A system that assigns percentages to sections of the body, allowing calculation of the amount of skin surface involved in the burn area.

Stevens-Johnson syndrome A milder form of toxic epidermal necrolysis, in which epidermal detachment involves less than 10% of the total body surface area; causes sloughing of the skin, mucous membranes, and cells lining the respiratory system.

subdermal burn A severe, life-threatening burn involving the deep structures of muscle, bone, larger blood vessels, and nerves; also called a fourth-degree burn.

superficial burn A burn involving only the epidermis, producing very red, painful skin; also called a first-degree burn.

superficial partial-thickness burn A burn involving the epidermis and part of the dermis, but the deeper layers of the dermis are not involved; also called a second-degree burn.

thermal burn A burn caused by heat, contact with hot objects, ignited liquids, steam, or hot liquids.

thromboxane A type of substance that causes blood vessels and bronchial smooth muscles to constrict, and promotes blood coagulation by causing platelets to collect.

toxic epidermal necrolysis syndrome A severe skin reaction to certain medications, environmental allergies, and other unknown toxins, in which epidermal detachment is greater than 30%.

zone of coagulation The center of a burn, usually the deepest and most severe area.

zone of erythema The outermost area of a burn, representing the least severe burned area, usually an area of first-degree burn.

zone of stasis The area found just outside the zone of coagulation, representing a burned area that is less severe.

Bibliography

Advanced Trauma Life Support for Doctors Student Manual. Chicago, IL: American College of Surgeons; 2005:275.

American Burn Association. Burn Incidence and Treatment in the US: 2007 Fact Sheet. Available at: http://www.ameriburn.org. Accessed February 5, 2008.

Demling RH, Desanti L, Orgill DP. What are the properties of normal skin: Skin (Biologic Properties).

Available at: http://www.burnsurgery.org. Accessed February 5, 2008.

Ma OJ, Cline DM, Tintinalli JE, Kelen GD, Stapczynski JS. *Emergency Medicine Manual,* 6th ed. New York, NY: McGraw-Hill Medical Publishing Division; 2004:586–592.

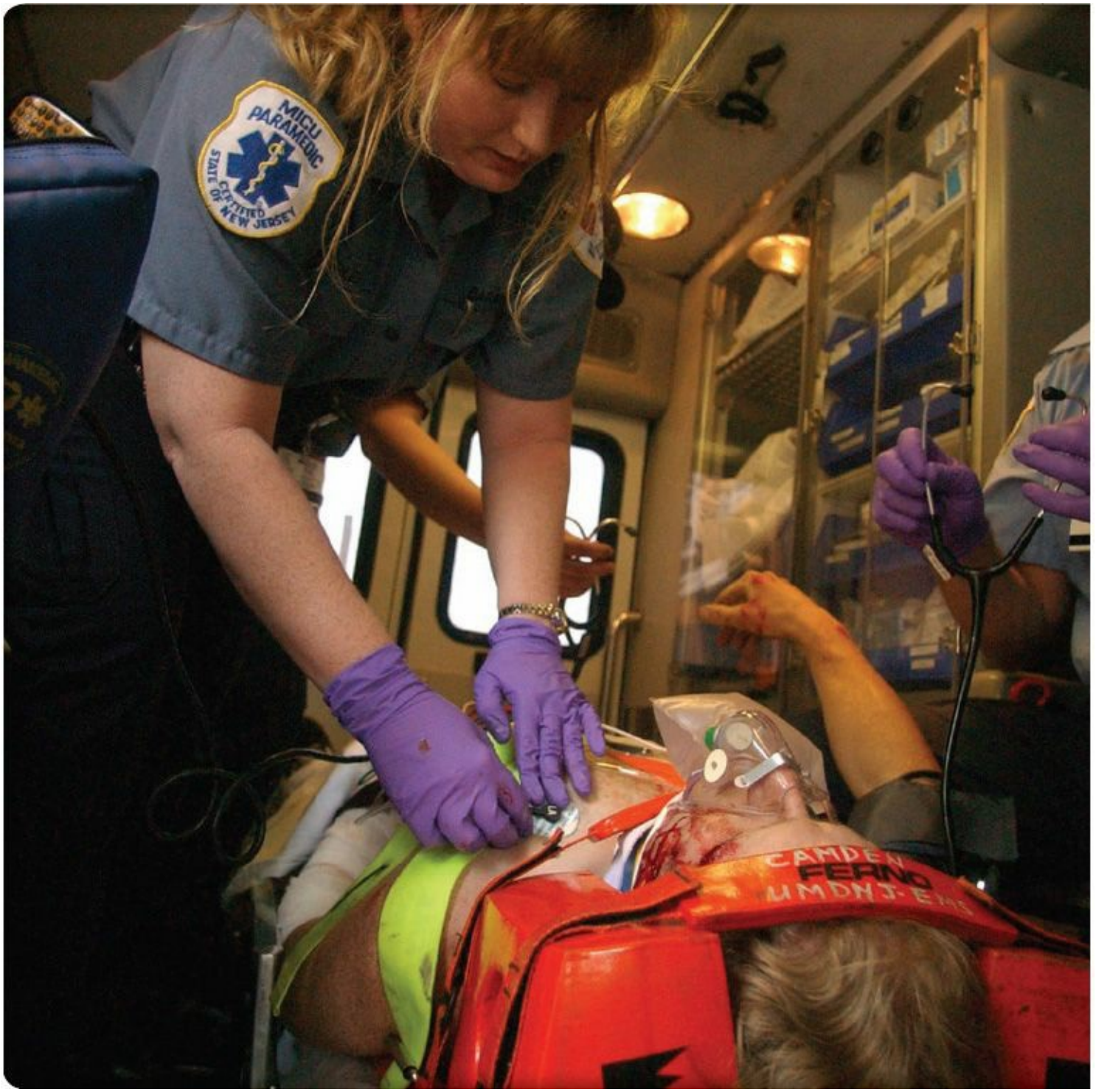
Marx JA, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice,* 5th ed. St Louis, MO: Mosby, Inc; 2002:801, 807, 808, 811, 812.

McLoughlin E, Crawford JD. Burns. *Pediatr Clin North Am* 1985;321:61–75.

Monteleone JA, Brodeur AE, eds. *Child Maltreatment: A Clinical Guide and Photographic Reference.* St Louis, MO: G. W. Medical Publishing; 2005:105, 107, 128.

Rockwell WB, Ehrlich HP. Should burn blister fluid be evacuated? *J Burn Care Rehab.* 1990;11:93.

Wolf SE, Herndon DN, eds. *Burn Care* (Vademecum series). Austin, TX: Landes Bioscience; 1999.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Electrophysiology, Cardiac Devices, and Transport Management

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Objectives

1. Explain how to correctly place leads from a 12-lead electrocardiographic (ECG) monitor (p 480–483).
2. Explain how to correctly place additional precordial leads for diagnosing right ventricular and posterior infarctions (p 483).
3. Explain how to determine the heart's electrical axis (p 486).
4. Discuss the step-by-step systematic approach that should be used when interpreting an ECG (p 484–486).
5. Describe how to monitor a patient by using an ECG machine during a critical care transport (p 543).
6. Explain the assessment process for a patient undergoing ECG monitoring during a critical care transport (p 544).
7. Describe how to identify a hemiblock on an ECG (p 489).
8. Explain the management of a patient with a hemiblock during critical care transport (p 494).
9. Describe how to identify a bundle branch block on an ECG (p 489–492).
10. Explain the management of a patient with a bundle branch block during critical care transport (p 489–492).
11. Describe the significance of ST- and T-wave changes and how they are identified on an ECG (p 496).
12. Describe complications that can occur with a cardiac patient during critical care transport (p 544).
13. Describe different criteria for determining the presence of left ventricular hypertrophy (LVH), right ventricular hypertrophy (RVH), and the presence of strain (p 498–499).
14. Using various criteria, identify cases of LVH and RVH on a 12-lead ECG (p 498–499).
15. Describe the clinical significance of LVH (p 498).
16. Identify criteria suggestive of right and left atrial enlargement, and describe the clinical implications of such enlargement (p 497–499).
17. Describe the ECG changes that indicate the presence of Wolff-Parkinson-White syndrome (p 500).
18. Identify ECG changes that could indicate conditions such as pericarditis, acute pulmonary embolism, and early repolarization variant (p 501).
19. Describe the potential implications of a prolonged QT interval (p 501–502).

20. Describe how to identify ventricular tachycardia on an ECG, including when it occurs in conjunction with wide complex tachycardia (p 502–504).
21. Explain the management of a patient with ventricular tachycardia, including when it occurs in conjunction with wide complex tachycardia (p 502–504).
22. Describe ECG changes associated with drug and electrolyte disturbances, hyperkalemia, hypokalemia, hypercalcemia, and hypocalcemia and the clinical implications of these conditions (p 504–507).
23. Generally describe the field of electrophysiology, including its purpose and capabilities (p 529).
24. Describe the spectrum of therapeutic options for patients with cardiac arrhythmias (p 529–530).
25. Briefly discuss the general categories of cardiovascular drugs and their actions and indications (p 531–532).
26. Understand the basic concepts underlying cardiac pacemaker technology, including the following:
    - Pacing circuits and impulses
    - Leads and electrodes
    - Single-chamber demand pacemakers and a dual-chamber pacemaker
    - NASPE/BPEG Generic (NBG) codes for describing pacemaker functions and capabilities (p 530–538).
27. Describe the general steps in pacemaker troubleshooting and the specifics of identifying and resolving problems with electromagnetic interference (p 536–537).
28. Recognize single- and dual-chamber pacing systems by their ECG characteristics (p 538–539).
29. Understand the purpose and function of the atriobiventricular pacing device (p 539–540).
30. Describe the components and function of implantable cardioverter defibrillators (ICDs) (p 540–541).
31. Understand the purpose of an atrial tachycardia ICD (p 543).
32. List the types of therapy an ICD can deliver and their indications (p 530, 540–541).
33. Discuss the general types of ICD malfunction, including the following:
    - Historical factors that may play a role or offer diagnostic clues
    - Common reasons for malfunction
    - The role of the "doughnut" magnet in managing ICD malfunction (p 532–533, 537)
34. Briefly list the special considerations related to external defibrillation when an ICD is present (p 542).

Introduction

On a routine basis, CCTPs encounter patients with cardiac arrhythmias and, when providing care, must be familiar with the various cardiac devices used to diagnose and treat such disorders. In particular, interpretation and monitoring of the 12-lead electrocardiogram (ECG)—including the understanding of the process behind the ECG changes and how they are depicted on the ECG monitor or printout—are key skills for CCTPs.

The 12-lead ECG graphically represents electrical activity in cardiac tissue. By using this information, CCTPs can interpret normal findings vs those found with changes in the function of cardiac tissue as a result of disease or injury. During the transport of a special care or critical care patient, there

are many modalities available to continuously monitor vital bodily functions. Cardiac monitoring in this setting typically includes a view of the heart's electrical rhythm in three or more leads and the serial evaluation of the 12-lead ECG. In some cases, additional precordial leads are placed for the diagnosis of right ventricular and posterior infarctions, which will be discussed later in this chapter. A key role of the critical care transport team is to continuously monitor the cardiac rhythm and serial 12-lead ECGs, responding to changes with rapid and appropriate intervention as indicated by local protocol and national guidelines, such as advanced cardiac life support (ACLS) and pediatric ALS. With that in mind, this chapter provides concise methods and patterns of interpretation to help CCTPs develop a deeper understanding of the causes and changes noted in electrical activity as a result of injury or illness.

Traditionally, patients with cardiac arrhythmias have most often been treated with medication therapy. These medications come with side effects that can reduce the patient's quality of life. However, patient adherence to the medication regimen is crucial to effectiveness. In some cases, medications are not always effective, even when patients use them correctly.

Technological developments that began with early ECG discoveries led to a cardiac subspecialty called electrophysiology. This field, in turn, has developed diagnostic and therapeutic equipment that vastly broadens the options for patients with cardiac arrhythmias—equipment that CCTPs will encounter routinely.

Implanted pacemakers and implantable cardioverter defibrillators (ICDs) offer solutions for patients with arrhythmias that might otherwise be disabling or lethal. These adjuncts also considerably raise the level of technological expertise required by the providers, including CCTPs, who care for patients with such devices. This chapter describes aspects of implantable devices and their electrical therapies that may be important during transport.

Cardiac Anatomy and Physiology

The Heart

Although CCTPs are already very familiar with cardiac anatomy and physiology, this section provides a brief review of some aspects. **Figure 13-1** shows the anatomy of the heart.

Myocardial cells require an uninterrupted supply of oxygen and nutrients. Indeed, the cardiac demand for oxygen is particularly unremitting because the heart never stops to rest (not without catastrophic consequences), so it is essential that the heart have an absolutely reliable blood supply. Oxygenated blood reaches the heart through the coronary arteries **Figure 13-2**, which branch off the aorta at the coronary ostia, just above the leaflets of the aortic valve. The numerous connections (anastomoses) between the arterioles of the various coronary arteries allow for the development of collateral circulation in case of blockage. Unfortunately, the coronary arteries are also vulnerable to narrowing in atherosclerotic heart disease. When the lumen of one of the arteries becomes so narrowed that blood flow through it is impeded, the symptoms of angina occur.

Heart Sounds

The purpose of listening to heart sounds is to identify the "lub-dub" that indicates the cardiac valves are operating properly. The major heart sounds are the two normal sounds, S_1 and S_2 **Figure 13-3**, and the two abnormal sounds, S_3 and S_4 **Figure 13-4**.

S_1 occurs near the beginning of ventricular contraction (systole), when the tricuspid and mitral valves close. The closing of these two valves should occur simultaneously as the pressure within the ventricles increases. This is the "lub" sound. Any delay in the closing of these two valves, heard as a split sound, is considered abnormal.

S_2 occurs near the end of ventricular contraction (systole), when the pulmonary and aortic valves close. As the ventricles relax, these valves close because of backward flow in the pulmonary artery and aorta. The two valves can close simultaneously or with a slight delay between them under normal physiologic circumstances. This is the "dub" sound.

S_3 is the result of the end of the rapid filling period of the ventricle during the beginning of diastole. An S_3 sound should occur 120 to 170 milliseconds after S_2, if it is heard at all. S_3 is generally heard in children and young adults. When it is heard in older adults, it often signifies heart failure. S_3 is sometimes called a Kentucky gallop, to help differentiate it from the S_4 sound, discussed next. When S_3 is present, the heartbeat sounds like dub-lub-dub (the second sound is the most pronounced, as in the word "Kentucky").

S_4, if heard, coincides with atrial contraction at the end of ventricular diastole. If heard at any other time, it usually occurs in patients who have resistance to ventricular filling, as with a weak left ventricle. S_4 is also normally heard in children and young adults. S_4 is sometimes called a Tennessee gallop, to help differentiate it from the S_3 sound. When S_4 is present, the heartbeat sounds like lub-dub-dub (the first sound is more pronounced than the latter two, as in the word "Tennessee").

Table 13-1 lists some of the most common heart sounds, where they are heard, and potential associated causes.

The Cardiac Cycle

The cardiac cycle comprises one complete phase of atrial and ventricular relaxation (diastole) followed by one atrial and ventricular contraction (systole).

During the relatively longer relaxation phase (normally 520 milliseconds), the left atrium fills passively with blood, under the influence of venous pressure. Approximately 80% of ventricular filling also occurs during this time as blood flows through the open tricuspid and mitral valves.

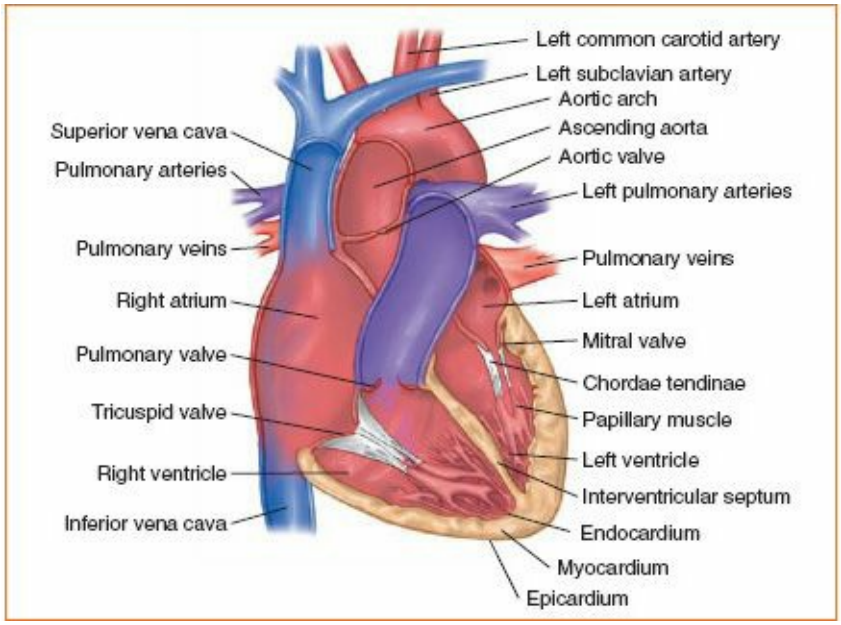


Figure 13-1 Anatomy of the heart.

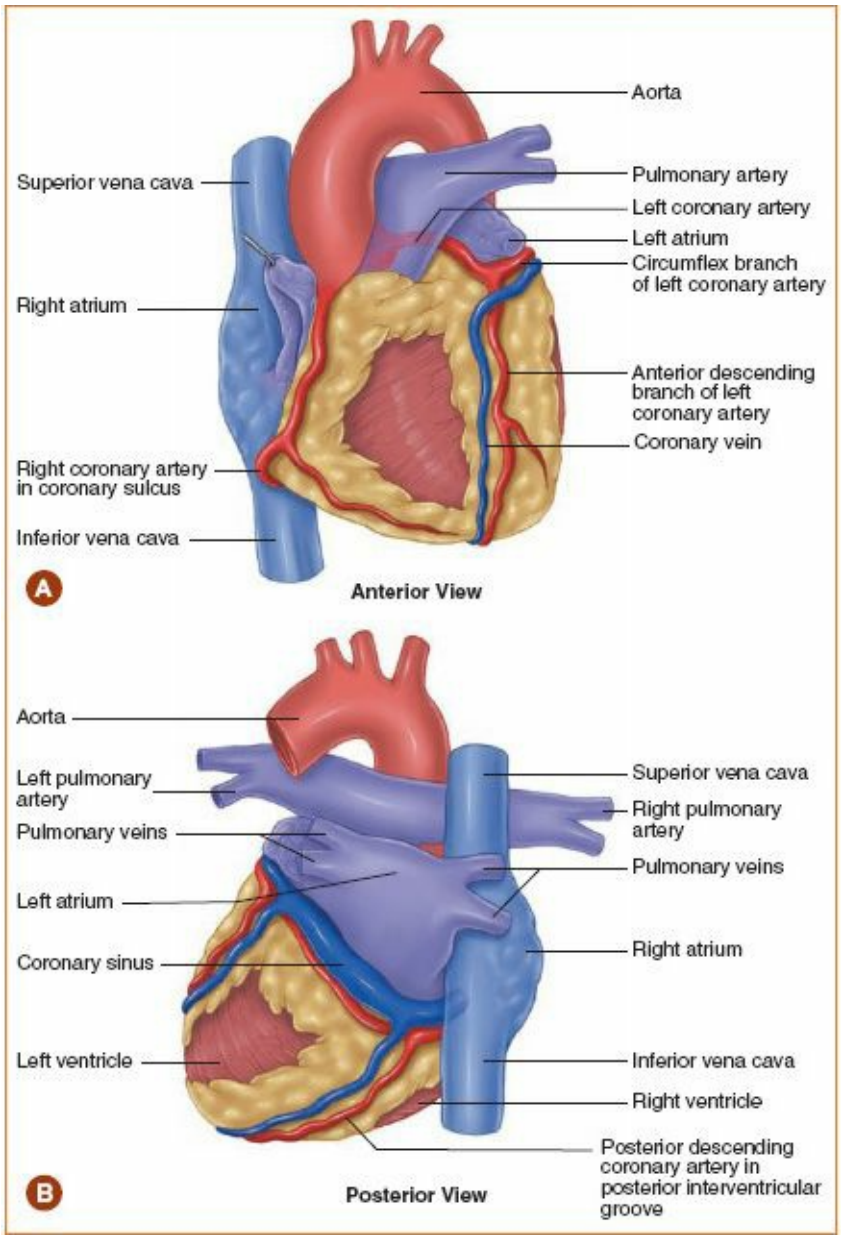


Figure 13-2 Coronary arteries. **A.** Anterior view, showing the takeoff point of the left and right main coronary arteries from the aorta. **B.** View from below and behind, showing the coronary sinus.

With atrial contraction, the contents of each atrium are squeezed into the respective ventricle to complete ventricular filling (atrial kick). Approximately 70% to 90% of the blood in the atria fills the ventricles by gravity; the remaining 10% to 30% comes from atrial contraction or atrial kick. At the beginning of ventricular contraction, the atrioventricular (AV) valves snap shut, the two ventricles contract (ventricular systole), and the semilunar valves are forced open. Blood squeezed out of the right ventricle moves forward, through the pulmonic valve, and into the pulmonary arteries. Blood from the left ventricle is pushed through the aortic valve and out into the aorta. Systole is usually accomplished in a little more than half the time it takes to fill the ventricles, about 280 milliseconds.

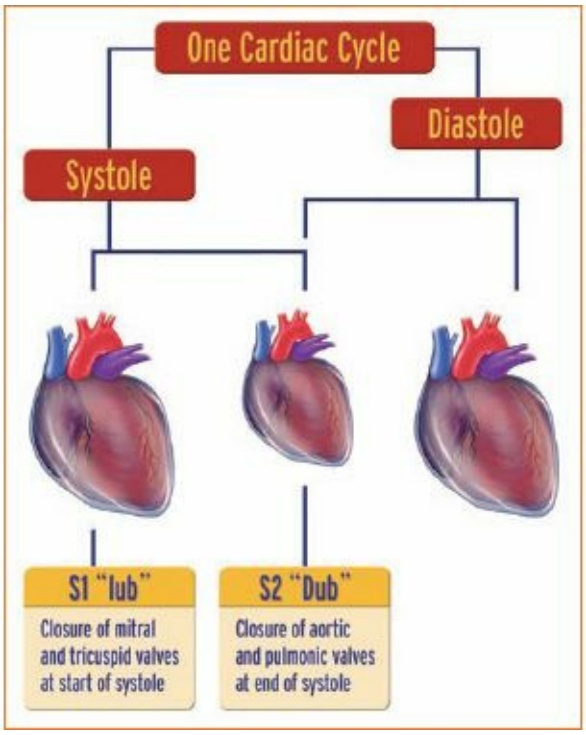


Figure 13-3 The normal S_1 and S_2 heart sounds.

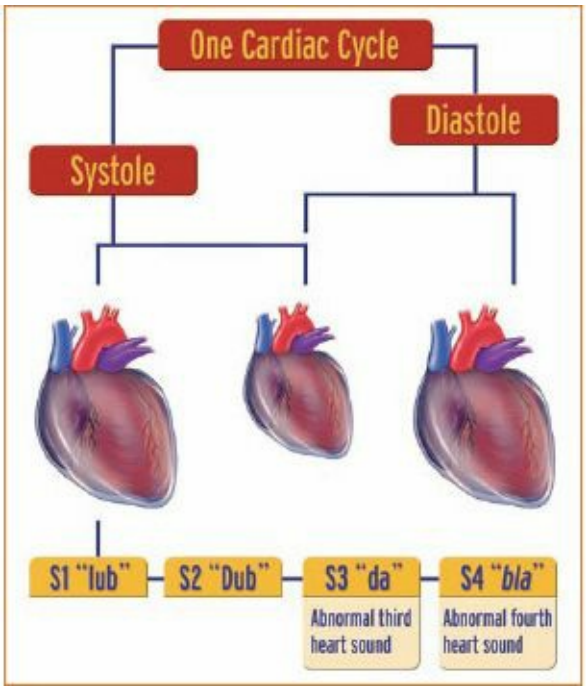


Figure 13-4 The abnormal S_3 and S_4 heart sounds.

TABLE 13-1 Heart Sounds

Heart Sound	Usual Location	Cause
S_1	Loudest in the mitral area, using the diaphragm	Normal
Split S_1	Between the apex of the heart and the sternum, usually during expiration, using the diaphragm	May be normal, nonsynchronous closing of mitral and tricuspid valves or pathologic, such as in a bundle branch block, an atrial septal defect, or

		pulmonary stenosis
S_2	Loudest in the aortic area, using the diaphragm	Normal
Split S_2	Loudest in the pulmonic area, usually on inspiration, using the diaphragm	May be normal, nonsynchronous closing of aortic and pulmonic valves or pathologic, such as in hypertension
S_3/ventricular gallop	Heard in early diastole just after the S_2; may be heard at the apex (left ventricular gallop) or at the left sternal border (right ventricular gallop); loudest on expiration with the patient in the lateral position, using the bell of the stethoscope	Always abnormal; indicates ventricular filling. May be normal in children and young adults or seen in diseases such as mitral valve regurgitation, hyperthyroidism, and congestive heart failure
S_4/atrial gallop	Heard late in diastole just before S_1, at the apex or over the suprasternal notch; heard best with the bell	May be normal (children and young adults); indicates increased resistance to ventricular filling after the atrial contraction; often seen after a myocardial infarction
Systolic murmur	Turbulent blood flow through the valves of the heart, during systole, using the diaphragm; location depends on cause	May be normal or seen with aortic or pulmonic stenosis or mitral or tricuspid regurgitation
Diastolic murmur	Turbulent blood flow through the valves of the heart, during diastole, using the diaphragm; location depends on cause	Always abnormal; seen with mitral or tricuspid stenosis, or aortic or pulmonic regurgitation
Pericardial friction rub	Scratchy, high-pitched sound heard throughout the cardiac cycle; best heard with the patient leaning forward, at the third intercostal space to the left of the sternum, using the diaphragm	Often present in pericarditis

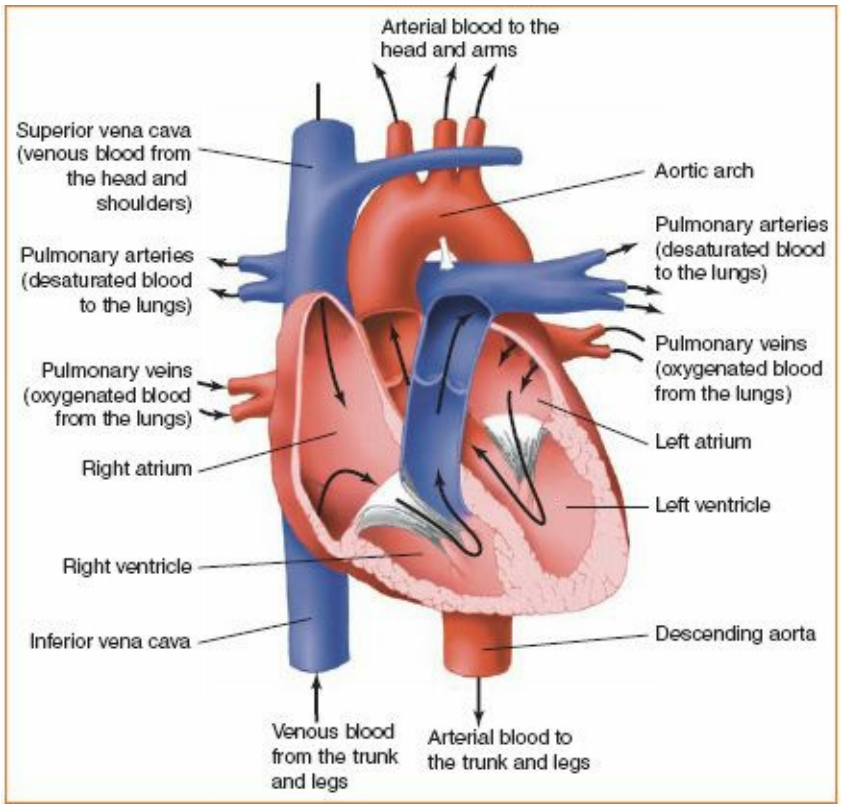


Figure 13-5 Blood flow through the heart.

Functionally, the heart is actually two pumps—a right pump and a left pump, separated by a thin wall (the interventricular septum)—that just happen, for purposes of efficiency, to be housed in one organ and to work in parallel **Figure 13-5**.

The right side of the heart is a low-pressure pump: It pumps against the relatively low resistance of the pulmonary circulation. The right atrium collects oxygen-poor venous blood from the venae cavae and the coronary sinus and pumps it into the right ventricle, which pumps the blood into the pulmonary artery for distribution to the alveoli and oxygenation.

The pulmonary veins collect the now oxygen-rich blood and return it to the left atrium, which pumps it into the left ventricle. The left side of the heart is a high-pressure pump: It drives blood out of the heart against the relatively high resistance of the systemic arteries.

At any given time, a major proportion of the body's blood flow may be shunted into one of the two circulation systems: the systemic circulation or the pulmonary circulation. If, for example, the right side of the pump fails and cannot squeeze out its contents efficiently, blood will back up behind the right atrium into the systemic veins, which then become engorged and distended. Distention of the external jugular veins signals that there is considerable back pressure from the right side of the heart throughout the systemic circulation. As pressure increases within the systemic veins, fluid starts to leak into the surrounding tissues, causing the tissues to swell. When enough fluid has leaked into the interstitial spaces, the swelling becomes visible as edema in the subcutaneous tissues (specifically, the lower extremities); it is less readily visible but equally present in the liver, walls of the intestine, and other internal tissues.

By contrast, if the left side of the pump fails, blood backs up behind the left atrium into the pulmonary circulation. As pressure builds up in the pulmonary veins, fluid is squeezed into the alveoli, producing the characteristic signs and symptoms of pulmonary edema: dyspnea, crackles, and frothy sputum.

The Blood Vessels

Figure 13-6 provides a review of the major arteries in the body.

Arterial walls are highly sensitive to stimulation from the autonomic nervous system. In response to that stimulation, the arterial diameter may change significantly as arteries contract and relax. Baroreceptors are involved in this process by sensing changes in blood pressure and, in turn, stimulating the autonomic nervous system. When baroreceptors sense increased blood pressure, they induce parasympathetic nervous system discharges that result in lowered heart rate and decreased myocardial contractility. Decreased blood pressure causes baroreceptor stimulation of the sympathetic nervous system, with a resulting increased heart rate and greater myocardial contractility. By this mechanism, the brain is able to respond to changes in blood pressure in the body. Blood pressure is influenced not only by the cardiac output and the volume present in the system, but also by the relative constriction or dilatation of arteries.

Chemoreceptors also play a role in autonomic nervous system activation in response to sensing changes resulting from respiration. When they note changes in P_{O_2} and pH or P_{CO_2}, they promote a process that not only stimulates the autonomic nervous system (thereby affecting heart rate and myocardial contractility) but also involves cell depolarization, which leads to release of dopamine and ultimately causes the respiratory centers in the brain stem to increase or decrease minute ventilation. Hypoxia or acidosis sensed by the chemoreceptors leads to sympathetic stimulation with resultant increased heart rate and contractility, whereas hyperoxia or alkalosis leads to parasympathetic stimulation with decreased heart rate and lessened contractility.

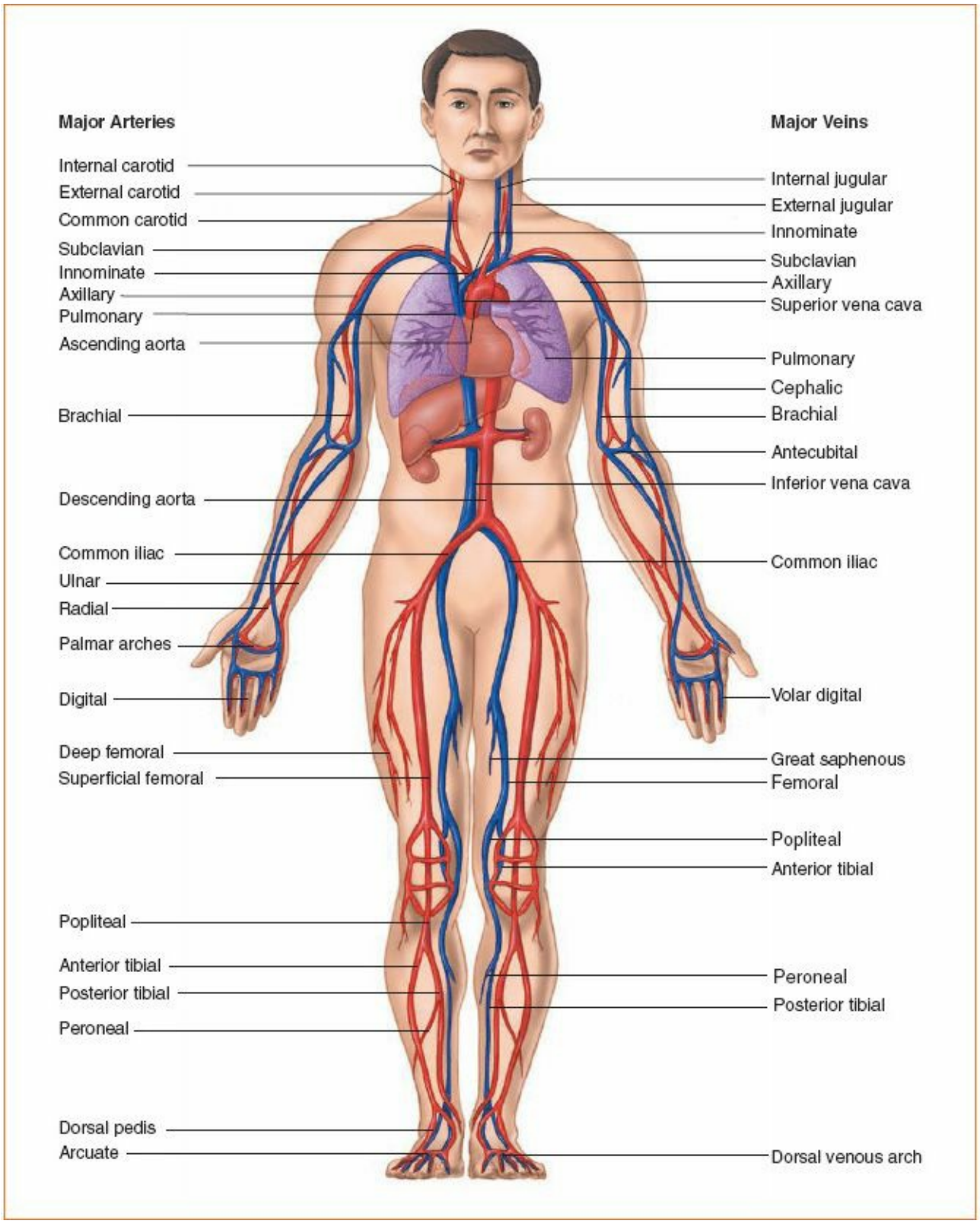


Figure 13-6 The major arteries and veins.

Functioning of the Pump

The following terms are important to understand how the heart functions as a pump:

- **Cardiac output (CO)** is the amount of blood that is pumped out by either ventricle. The left and right ventricles are approximately equal in interior size, so the two ventricles have relatively equivalent outputs. The normal CO for an average adult is 5 to 6 L/min.
- **Stroke volume (SV)** is the amount of blood pumped out by either ventricle in a single contraction (heartbeat). Normally, the SV is 60 to 100 mL, but the healthy heart has considerable spare capacity

and can easily increase the SV by at least 50%.

- **Heart rate** is the number of cardiac contractions (heartbeats) per minute—in other words, the pulse rate. The normal rate for adults is 60 to 100 beats/min.

The volume of blood that either ventricle pumps out per minute equals the volume of blood it pumps out in a single contraction times the number of contractions per minute:

$$CO = SV \times \text{Heart Rate}$$

To meet changing demands, the heart must be able to increase its output several times over in response to the body's increased demand for oxygen. The heart can increase its output by increasing the SV, increasing the heart rate, or both.

The heart has several ways of increasing SV. One characteristic of cardiac muscle is that, when it is stretched, it contracts with greater force to a limit—a property called the Frank-Starling mechanism. If an increased volume of blood is returned from the systemic veins to the right side of the heart or from the pulmonary veins to the left side of the heart, the muscle surrounding the cardiac chambers must stretch to accommodate the larger volume. The more the cardiac muscle stretches, the greater the force of its contraction, the more completely it empties, and, therefore, the greater the SV. From the CO equation, it is clear that any increase in SV, with the heart rate held constant, will cause an increase in the overall CO.

Preload (the pressure under which a ventricle fills) is influenced by the volume of blood returned by the veins to the heart. In situations of increased oxygen demand, the body returns more blood to the heart (preload increases), and CO consequently increases through the Frank-Starling mechanism. In a diseased heart, the same mechanism is used to achieve a normal resting CO (which explains why some diseased hearts become enlarged).

The heart can also vary the degree of contraction of its muscle *without* changing the stretch on the muscle; this ability is called contractility. Changes in contractility may be induced by medications that have a positive or negative **inotropic** effect. The ventricles are never completely emptied of blood with any single beat. However, if the heart squeezes into a tighter ball when it contracts, a larger percentage of the ventricular blood will be ejected, thereby increasing SV and overall CO. The nervous system regulates the contractility of the heart from beat to beat. When the body requires increased CO, nervous signals increase myocardial contractility, thereby augmenting SV.

The heart can also increase its CO, given a constant SV, by increasing the number of contractions per minute (positive **chronotropic** effect).

The Frank-Starling mechanism is an intrinsic property of heart muscle—that is, it is not under nervous system control. By contrast, contractility and changes in heart rate are regulated by the nervous system.

The Electrical Conduction System of the Heart

Heart muscle can generate its own electrical impulses without stimulation from nerves, a property called automaticity. In addition, the heart is endowed with an electrical conduction system, a specialized conduction tissue that can rapidly propagate electrical impulses to the muscular tissue of the heart. These impulses may be recorded by the provider on the ECG; therefore, all CCTPs need to understand the relationship between the activities of the body's electrical conduction system and 12-lead ECG interpretation.

Visualizing the process of impulses traveling in different directions helps in understanding what is happening in the heart. Visualizing the travel of impulses also promotes an understanding of how any changes in the structure or function of the heart can alter their travel.

The Dominant Pacemaker: The Sinoatrial Node

Theoretically, any cell within the heart's electrical conduction system can act as a pacemaker. In the normal heart, however, the dominant pacemaker is the sinoatrial (SA) node **Figure 13-7**. The SA node receives blood from the right coronary artery (RCA). If the RCA is occluded, as in a myocardial infarction (MI), the SA node will become ischemic. The subsequent death of the conduction cells will prevent the SA node from firing.

The SA node initiates the electrical discharge that passes through the **intra-atrial pathways** (or intranodal pathways); in physiology, these are sometimes called atrionodal pathways and include the anterior or Bachman bundle, middle bundle, and posterior internodal system. The SA node is the fastest pacemaker in the heart. Electrical impulses generated in this node spread across the two atria through internodal pathways in the atrial wall in about 80 milliseconds, causing the atrial tissue to depolarize as they pass. In 85% to 90% of humans, the blood supply for the SA node comes from a branch of the RCA; in 10% to 15% of humans, it comes from a branch of the left circumflex artery. The conduction of the impulse is delayed in the AV node for about 120 milliseconds so that the atria can empty into the ventricles.

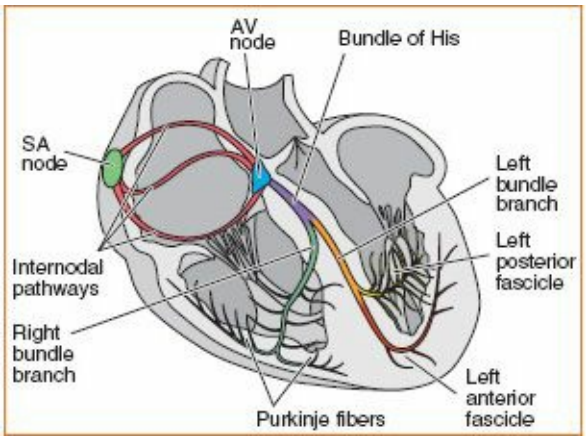


Figure 13-7 The electrical conduction system.

When the atrial rate becomes very rapid, not all atrial impulses can get through the AV junction. Normally, however, impulses pass through it into the bundle of His and then move rapidly into the right and left bundle branches located on either side of the interventricular septum. Next, they spread into the Purkinje fibers, thousands of fibrils distributed through the ventricular muscle. It takes about 80 milliseconds for an electrical impulse to spread across the ventricles, during which time the ventricles contract simultaneously. The effect on the velocity of conduction is referred to as the **dromotropic** effect.

Depolarization and Repolarization

As the electrical impulses travel along these pathways, they come into contact with each cardiac cell, which continues the process of generating electrical impulses or depolarization through the properties of automaticity and conductivity. **Depolarization**—the process by which muscle fibers are stimulated to contract—comes about through changes in the concentration of electrolytes across cell membranes. Myocardial cells, like all cells in the body, are bathed in an electrolyte solution. Chemical pumps inside the cell maintain the concentrations of ions within the cell, in the process creating an electrical gradient across the cell wall. Consequently, a resting (polarized) cell normally has a net charge of –90 mV with respect to the outside of the cell.

When the myocardial cell receives a stimulus from the conduction system, the permeability of the cell wall changes through opening of specialized channels in such a way that sodium ions (Na^+) rush into

the cell, causing the inside of the cell to become more positive. Calcium ions (Ca^{++}) also enter the cell—albeit more slowly and through a different set of specialized channels—helping maintain the depolarized state of the cell membrane and supplying calcium ions for use in contraction of the cardiac muscle tissue. This reversal of electrical charge—depolarization—starts at one spot in the cell and spreads in a wave along the cell until the cell is completely depolarized. The entire process causes a "wave of depolarization" as the electrical charges pass in all directions from one cell to another. The larger the mass of cardiac muscle, the larger this wave of depolarization will be. As each cell depolarizes, it creates a small electrical vector. In the wave of depolarization, the sum of all these small vectors creates the electrical axis, described later in more detail.

As the cell depolarizes and calcium ions enter, mechanical contraction of the cardiac cells occurs. During diastole, the coronary arteries fill and cardiac circulation occurs. As the cells contract from the base of the heart through the apex, the blood is pumped through the pulmonary and systemic circulation.

Following depolarization, each cell must recharge or repolarize by shifting intracellular electrolytes back into place to ready itself for this continual process. The cell is able to recover from depolarization through a process called **repolarization**. Repolarization starts with the closing of the sodium and calcium channels, which stops the rapid inflow of these ions. Next, special potassium channels open, allowing a rapid escape of potassium ions (K^+) from the cell. This process helps restore the inside of the cell to its negative charge; the proper electrolyte distribution is then reestablished by pumping sodium ions out of the cell and potassium ions back in. After the potassium channels close, this sodium-potassium pump helps move sodium and potassium ions back to their respective locations. For every three sodium ions this pump moves out of the cell, it moves two potassium ions into the cell, thereby maintaining the polarity of the cell membrane. To accomplish this task, the sodium-potassium pump moves ions against the natural gradient by a process called active transport, which requires the expenditure of energy.

TABLE 13-2 The Role of Electrolytes in Cardiac Function

Electrolyte	Role in Cardiac Function
Sodium (Na^+)	Flows into the cell to initiate depolarization
Potassium (K^+)	Flows out of the cell to initiate repolarization • Hypokalemia → increased myocardial irritability • Hyperkalemia → decreased automaticity/conduction
Calcium (Ca^{++})	Has a major role in the depolarization of pacemaker cells (maintains depolarization) and in myocardial contractility (involved in contraction of heart muscle tissue) • Hypocalcemia → decreased contractility and increased myocardial irritability • Hypercalcemia → increased contractility
Magnesium (Mg^{++})	Stabilizes the cell membrane; acts in concert with potassium, and opposes the actions of calcium • Hypomagnesemia → decreased conduction • Hypermagnesemia → increased myocardial irritability

Table 13-2 summarizes the roles of the various electrolytes in cardiac function.

A myocardial cell cannot respond to an electrical stimulus from the conduction system normally unless it is fully polarized. The period when the cell is depolarized or in the process of repolarizing—the so-called **refractory period**—consists of two phases. In the **absolute refractory period**, the heart muscle has been drained of energy and needs to recharge; it will not contract during this period. In the **relative refractory period**, the heart is partially charged, albeit not strongly enough to create a full contraction.

Secondary Pacemakers

The SA node normally has the most rapid intrinsic rate of firing (60 to 100 times per minute), so it will literally outpace any slower conduction tissue. If it becomes damaged or is suppressed, any component of the conduction system may act as a secondary pacemaker. The farther down the conduction system the pacemaker is, the slower its intrinsic rate of firing. Thus, the AV junction will spontaneously fire 40 to 60 times per minute, whereas the ventricular Purkinje system, which is farther removed from the SA node, will spontaneously fire only 20 to 40 times per minute.

Measuring the Electrical Conduction Activity of the Heart

The electrical conduction events in the heart can be recorded on an ECG as a series of waves and complexes **Figure 13-8**. The depolarization of the atria produces the P wave. It is followed by a brief pause as conduction is momentarily slowed through the AV node and the AV junction, which can be seen on the ECG as the **PR interval**. Collectively, the electrical impulses that pass through the right and left bundle branches and Purkinje fibers, representing depolarization of the ventricles, are depicted by the **QRS complex**. Repolarization of the atria and ventricles produces the **T wave**; however, the atrial repolarization wave is small and is buried within the QRS complex, so it is not seen. The larger ventricular T wave follows the QRS complex. The **ST segment**, or the section of the complex that is from the end of the QRS complex to the beginning of the T wave, is important in ECG interpretation and represents the period of inactivity between ventricular depolarization and repolarization. Mechanically, it represents the time that the myocardium is maintaining contraction. Finally, a U wave may be present after the T wave. There is no definitive answer as to what it represents, but it correlates with certain conditions discussed later in this chapter.

Any event that changes the structure or function of the cardiac cells typically results in change in the normal size and shape (morphologic features) of the complexes recorded on the ECG. The strategically placed ECG leads detect this electrical wave of depolarization in the selected positive lead, and the wave is recorded on the ECG as the familiar P-QRS-T waveform.

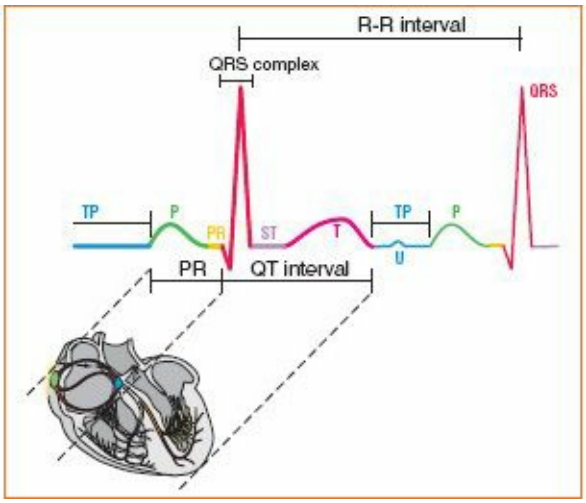


Figure 13-8 The electrocardiogram and cardiac events.

Cardiac Monitoring: 12-Lead ECG

Monitoring vs Assessment

Today's cardiac monitors and defibrillators are typically multiparameter devices that can combine continuous 3-lead ECG monitoring, 12-lead electrocardiography, semiautomatic and manual defibrillation, external pacing, capnography, pulse oximetry, blood pressure monitoring, end-tidal carbon dioxide measurements, CO measurements, and other capabilities.

Assessment of the patient's cardiac status takes place before monitoring. This assessment should include an analysis of the cardiac rhythm and 12-lead ECG to determine any underlying electrical disturbances caused by changes in the structure or function of the heart as a result of disease, ischemia, injury, or infarction.

Cardiac monitoring generally refers to the continuous observation of the patient's condition in relation to his or her cardiac rhythm. Typically, monitoring uses lead configurations for viewing from one to three leads simultaneously. If a patient's heart rhythm changes during the monitoring process, it can quickly be verified in multiple leads and the appropriate intervention can be initiated as directed by protocol.

Lead Placement

The equipment used to record the ECG may vary widely. Lead placement could also vary when using a machine that derives the 12-lead ECG from a 5- to 6-lead system. Several manufacturers currently produce this equipment, and each has its own proprietary lead set and different lead configurations. Major problems can result from using different techniques to acquire the "standard" 12-lead ECG. A "gold standard" for 12-lead ECG interpretation is the ability to perform serial evaluations for comparison; however, it is important to use consistent lead placements to ensure that all providers compare "apples to apples." Therefore, for simplicity and consistency, in this chapter the discussion on 12-lead ECG will use the "standard" lead placements and the views recorded from them.

The terms **lead**, **electrode**, and **cable** are often used interchangeably, which can be misleading; standard terminology should be used to avoid confusion. The electrode is the sensing device that connects directly to the skin and is usually a self-adhesive pad with some form of conduction media embedded in it to improve the connection of the electrode to the skin, thus improving the quality of the ECG. The lead is the designated position of the electrode (ie, limb and precordial/chest leads). The "lead" names the electrode placement. This is why the two terms are often used synonymously. The cable is the physical wire that connects the electrode to the ECG monitor.

Before connecting the electrodes to the chest, the skin should be prepared to help improve the quality of the ECG. The skin should be clean, dry, and slightly abraded by rubbing with a gauze pad. In addition to drying the skin, this step helps remove oils and improves the connection with the electrode. In a patient with excessive chest hair (or back hair with the use of additional nonstandard posterior leads), the hair should be shaved with a safety razor before connecting the electrodes. Other unusual conditions affecting the quality of the connection with the electrode to the skin should be corrected as necessary.

To get an accurate illustration and record the electrical activity of the heart, the leads must be placed so that they each provide a unique view **Figure 13-9**.

The six **limb leads** (I, II, III, aVR, aVL, and aVF [a indicates augmented; V, voltage; R, right arm; L, left arm; F, foot]) provide a view of the heart in a vertical plane, also called the frontal plane, and the six **precordial leads** (V_1 to V_6), or chest leads, show a horizontal plane. With the leads placed in standard locations, each provides a view of a particular region of the heart. For this reason, it is important to place leads in the proper location each time. The combination of limb leads and precordial leads provides a

three-dimensional view of the heart **Figure 13-10**. The six limb leads are created using only four electrodes (right arm, left arm, right leg, and left leg); the four electrodes on the body create six actual readings on the ECG.

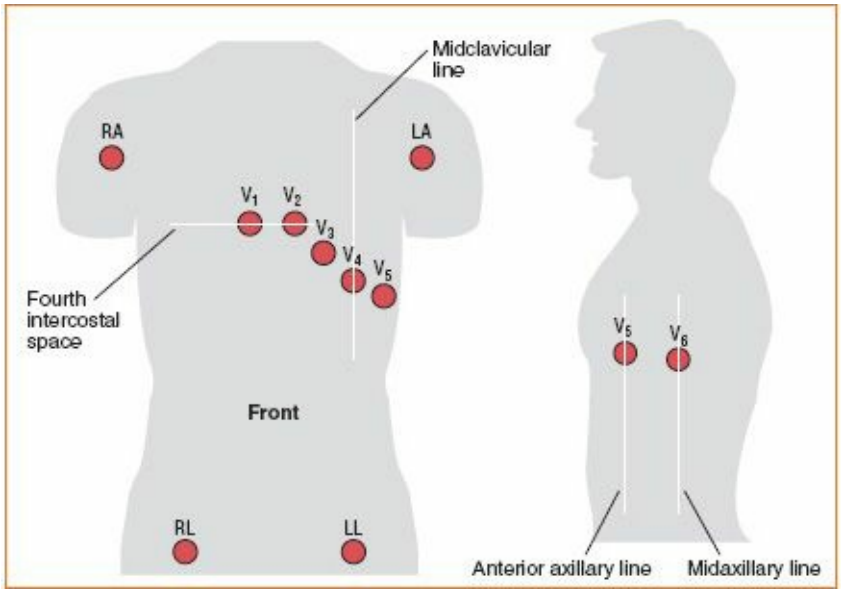


Figure 13-9 Limb lead and precordial lead placement. LA indicates left arm; LL, left leg; RA, right arm; RL, right leg.

The average of all electrical impulses generated in the heart can be detected and recorded by the ECG monitor through the combination of leads. These electrical impulses are displayed on the cardiac monitor and can be printed in a standard ECG format. To better understand the normal waveforms such as P-QRS-T and any additional electrical impulses or changes in the typical presentation, CCTPs should recall that electricity flows from negative toward positive. All ECG leads are considered positive when selected to record the electrical impulse. If the flow of electrical activity proceeds unimpeded toward a positive lead, it will be represented as a positive deflection on the ECG. If the flow of electrical activity flows away from a positive lead, it will be deflected negatively. An electrical impulse that crosses perpendicular to a positive lead is called isoelectric **Figure 13-11**.

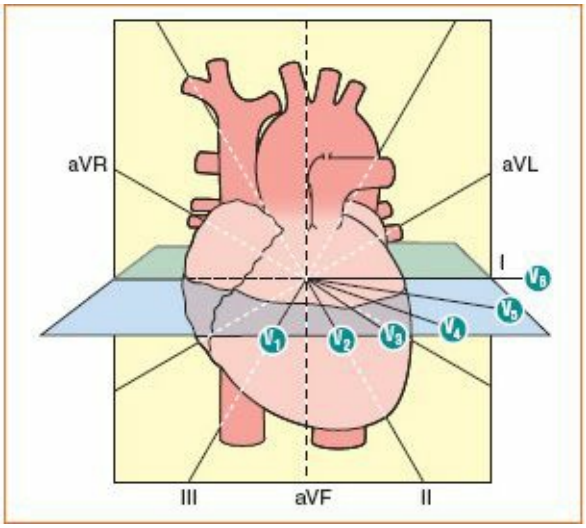


Figure 13-10 The combination of limb and precordial leads provides a three-dimensional view of the heart.

If the electrical impulse of the heart is deflected by a change in structure or function, it will be less

positive, aberrantly conducted, or deflected away from the selected electrode. Because the heart is three dimensional and the electrical impulses travel through the cardiac tissue in all directions, the leads of the cardiac monitor must be placed in the proper anatomic location to best detect these impulses. Traditionally, leads have been categorized as unipolar or bipolar, depending on how they function. The standard limb leads I, II, and III are bipolar leads because they use two leads, a positive and a negative, to measure the electrical potential as it flows from one to the other (negative to positive). The augmented leads (aVR, aVL, and aVF) and the precordial (chest) leads (V_1 through V_6) are unipolar because they use a single positive recording electrode.

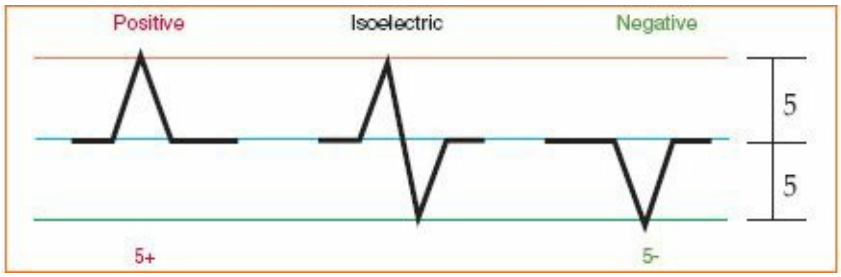


Figure 13-11 Representation of positive, isoelectric, and negative QRS deflections.

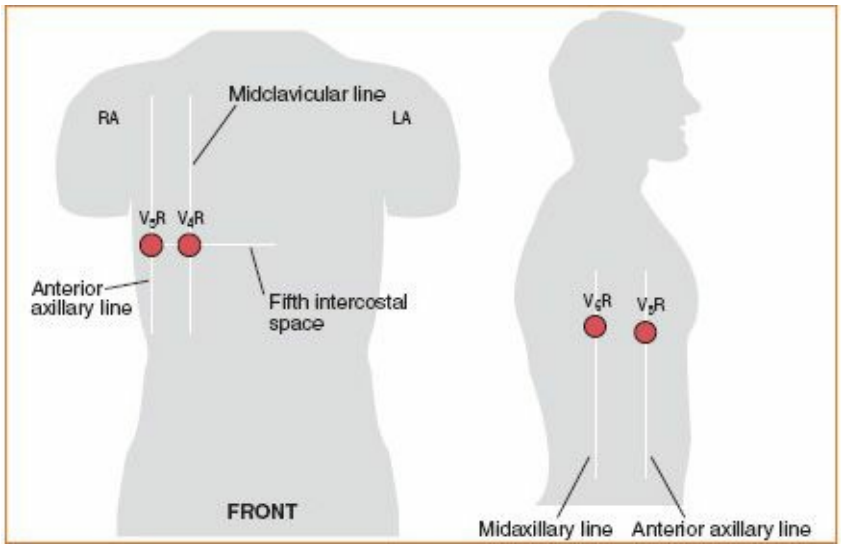


Figure 13-12 Placement of right-sided leads. LA indicates left arm; RA, right arm.

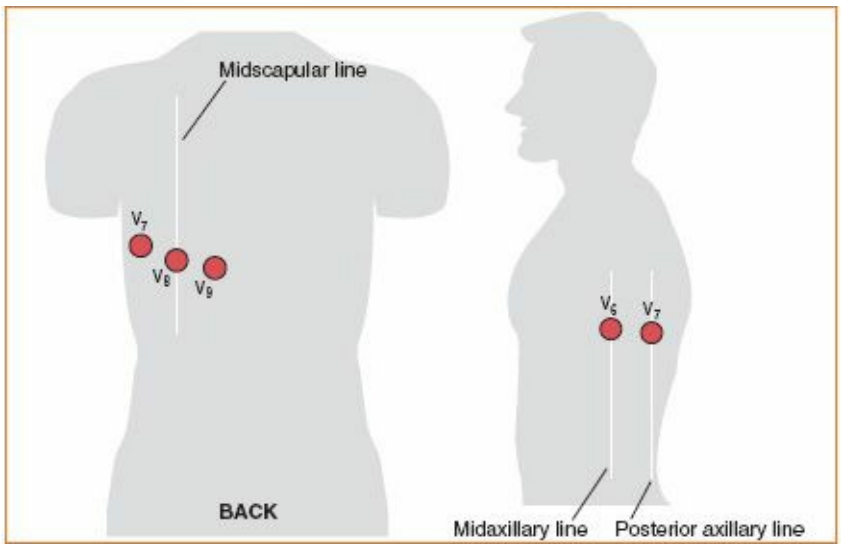


Figure 13-13 Placement of posterior leads.

As shown in Figure 13-9, the limb leads (extremity leads) include the right arm, left arm, right leg, and left leg, as marked on the lead connectors. Accurate, reproducible ECGs can be obtained by placing these limb leads on the arms or shoulders and lower abdomen as long as they are at least 10 cm from the heart, as shown in Figure 13-10. The precordial (chest) leads must be consistently placed in the proper locations:

- V_1 is placed in the fourth intercostal space to the right side (patient's right) of the sternum.
- V_2 is placed in the fourth intercostal space to the left side (patient's left) of the sternum.
- V_3 is placed between V_2 and V_4.
- V_4 is placed in the fifth intercostal space (patient's left) midclavicular line.
- V_5 is placed between V_4 and V_6.
- V_6 is placed in the fifth intercostal space (patient's left) midaxillary line.

The use of additional or nonstandard right-sided heart leads, V_4R, V_5R, and V_6R **Figure 13-12**, can provide useful information, as can the posterior heart leads V_7, V_8, and V_9 **Figure 13-13**, in suspected right ventricular and posterior MIs. The addition of these leads can increase sensitivity and specificity in the detection and location of the infarct. They are also indicated when a patient has clinical findings suggestive of acute coronary ischemia, but the results of the standard 12-lead ECG are normal or nondiagnostic.

There is specialized equipment that can be used for an ECG performed with additional right-sided leads, but this equipment is not necessary. An ECG of the additional leads can be obtained by using a standard 12-lead ECG machine. The standard principles and methods of reading the additional-lead ECG remain the same as for the 12-lead ECG, only the anatomic location that the leads are "looking" at is different. When switching standard 12-lead ECG leads to conform to the nontraditional right-sided and posterior leads, the ECG printout should be labeled to represent the leads recorded.

Before running an ECG with right-sided leads, the standard 12-lead ECG should be completed first. Then, the additional leads are attached as follows, using the precordial (chest) leads (right and left refer to the patient's right and left sides, respectively):

- V_4R is placed in the right midclavicular line, fifth intercostal space, and connected using the V_3 lead.
- V_5R is placed in the right anterior axillary line, on a straight line from V_4R, and connected using the V_2 lead.
- V_6R is placed in the right midaxillary line, on a straight line from V_5R, and connected using the V_1 lead.
- V_7 is placed in the left posterior axillary line, on a straight line from V_6, and connected using the V_4 lead.
- V_8 is placed in the left midscapular line, on a straight line from V_7, and connected using the V_5 lead.
- V_9 is placed in the left paraspinal line, on a straight line from V_8, and connected using the V_6 lead.

The bipolar lead MCL1 (modified chest lead 1) has been used as a substitute for cardiac monitoring when using a three-lead system. Modern research, however, has shown that QRS morphologic features, when recorded simultaneously with V_1 and MCL1, are different in a significant number of cases (38%), and this recording is not recommended when it is possible to obtain a true V_1 lead. Given the availability

of advanced monitoring systems in the critical care transport setting, the 12-lead ECG and multilead monitoring should be the standard of care.

Confirmation of Lead Placement

Confirming lead placement may seem like a redundant act, but in the critical care transport environment in particular, with moving a patient from one facility to another, there are innumerable opportunities for leads to become disconnected. As described later in this chapter, because each lead "looks" at a particular anatomic area of the heart, mixing up the leads will cause erroneous ECG readings, which could result in inappropriate therapeutic interventions. Placement should be confirmed before transport and several times during transport.

Recording the 12-Lead ECG

The acquisition of a 12-lead ECG is a simple procedure that takes only a few minutes. In critical care transport, it can be assumed that the patient has already had baseline ECG evaluations and that the ECG monitoring system has been connected to the patient before transport. With interfacility transports, CCTPs must ensure that the monitoring system and lead configuration at the initial facility are consistent with those that will be used during transport. A "new" 12-lead ECG should be recorded and evaluated before transport and repeated at regular intervals, when any changes in the ECG are noted, and on arrival at the receiving facility; however, obtaining additional ECGs should never delay the transport of a patient with an **ST-elevation myocardial infarction (STEMI)** to definitive care (STEMI is discussed later in this chapter). To obtain the best ECG quality, the patient should remain as still as possible while the ECG is being obtained to minimize artifact seen on the recording. The supine position is recommended; however, an adequate ECG can be obtained with the patient in a semisitting (semi-Fowler) position. (A patient's position other than supine during ECG acquisition should be noted on the ECG tracing.) The patient's condition will dictate the position of evaluation during transport. If all required electrodes for 12-lead ECG monitoring have not been applied to the skin, they should be placed before the patient is moved. The skin should be prepared as previously described to provide adequate contact with the electrodes. Following this preparatory phase, with the leads connected to the corresponding electrodes, the 12-lead ECG can be obtained.

Before obtaining a 12-lead ECG, standardization, or calibration, is necessary. With the 12-lead ECG, amplitude and duration are important factors, especially in analyzing ST segments and making other measurements. Depending on the equipment, the calibration or standardization should be set so that 1 mV is 10-mm high and 200-milliseconds wide. Some equipment can be set to half-standard calibration. This calibration is usually done when the complexes are so tall that they overlap each other on the ECG paper when recorded. Another calibration on newer machines is the paper speed, which should be set at the standard 25 mm/s, not 50 mm/s, which may have been done to help evaluate the morphologic features of ECG complexes in a tachycardic rhythm. Be sure to check your machine's calibration. Most of the newer 12-lead ECG machines can provide a printout of all 12 leads along with a rhythm strip in lead II or another designated lead.

Interpretation of the 12-Lead ECG

Interpretation of the 12-lead ECG in the critical care transport setting requires CCTPs to adjust to a unique work environment. This setting is neither fully in-patient nor prehospital but a combination of each. Patients who would typically be cared for only in the critical care unit of a hospital will be packaged, moved out of the relative safety or comfort of "the unit," and placed in a transport vehicle. Sometimes there can be multiple moves and several vehicles before the prescribed destination is reached.

Interpretation of a 12-lead ECG in this environment can pose unique challenges. Typically, time is critical, and using ECG calipers, rulers, axis wheels, and straight edges is particularly difficult in a moving vehicle.

All prospective CCTPs must have a full understanding of the standard practices and principles of 12-lead ECG interpretation and monitoring before embarking on this program. The following will provide basic ECG interpretation skills that should already be familiar to CCTPs. What will be emphasized is the understanding of the process behind the ECG changes and how they are depicted on the ECG monitor or printout. Some field-expedient methods of interpretation will be discussed to help build on the base knowledge expected of a health care provider entering this program.

Figure 13-14 provides a graphic representation of what the electrical impulses look like when transmitted to the 12-lead ECG. As shown in Figure 13-8, the configuration of the complexes varies somewhat from lead to lead; therefore, before attempting to identify any abnormalities, CCTPs must know what a typical complex looks like from each lead. In addition to knowing what each complex looks like, CCTPs must know what a normal 12-lead ECG pattern looks like. If the ECG does not fit this pattern, an abnormality is present.

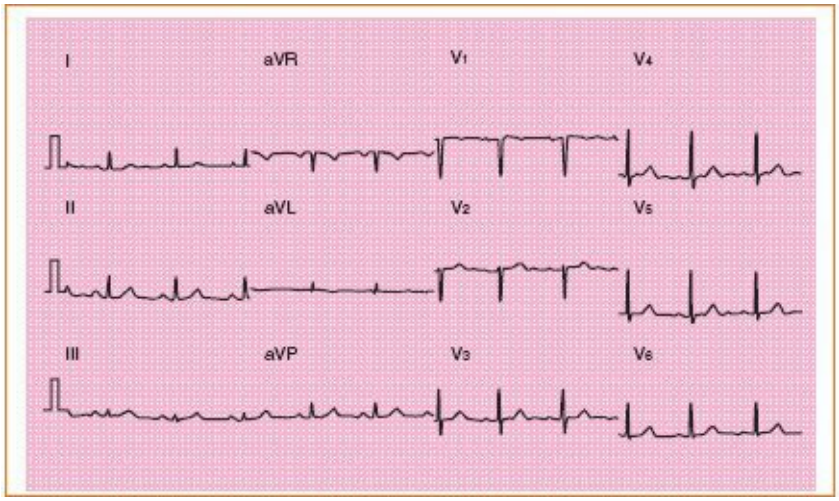


Figure 13-14 A normal 12-lead electrocardiogram.

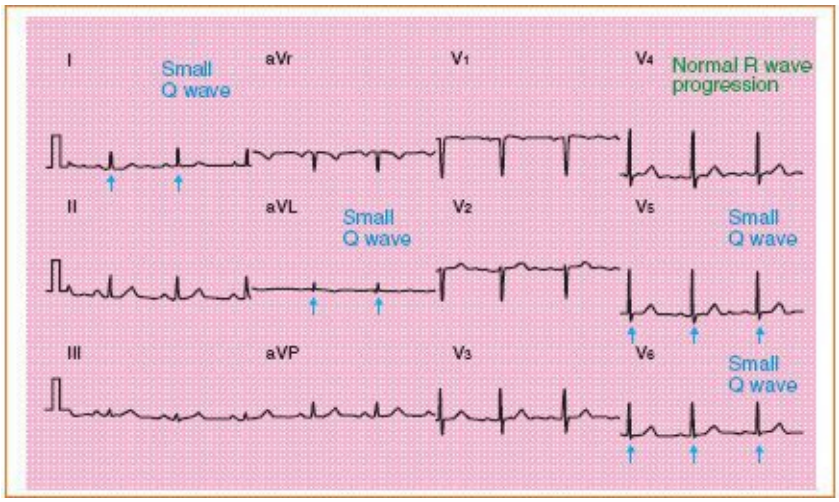


Figure 13-15 A small Q wave in leads I, aVL, V_5, and V_6 representing septal depolarization. This electrocardiogram also shows normal R-wave progression.

In the limb leads, the P wave is typically upright in leads I, II, aVL, and aVF. It is frequently biphasic in lead III and is purely negatively deflected in lead aVR. In the precordial leads, the P wave is typically

upright in leads V_5 and V_6. Lead V_1 is biphasic, and leads V_2 and V_4 are variable. These are the typical presentations for the P wave, although its appearance can vary from person to person.

The rules for measuring the PR interval remain unchanged from those for routine cardiac monitoring. The PR interval is a representation of the time from the start of atrial depolarization to the start of ventricular depolarization and also includes the delay in conduction that occurs at the AV junction. The PR interval usually lasts from 120 to 200 milliseconds (0.12 to 0.20 seconds).

The QRS complex is a representation of ventricular depolarization. The septal fascicle delivers the electrical impulse to the interventricular septum. The septum is the first part of the ventricles to depolarize. Septal depolarization is not always seen on the ECG, but when it is, a small Q wave may be seen in leads I, aVL, V_5, and V_6. **Figure 13-15** provides examples of each precordial lead and demonstrates the concept of R-wave progression.

The T wave will usually be recorded in a positive deflection in the same leads that record a positive deflection of the R wave. In other words, it is usual to find positive T waves in the same leads that have tall R waves.

As discussed in the lead placement section, each lead provides an enhanced view of a particular region of the heart. As a result, the electrical activity picked up by this lead placement is recorded as complexes from these different perspectives. Understanding that each lead represents a different enhanced view of the heart is the first step in learning to scan the 12-lead ECG by anatomic location (**Figure 13-16** and **Table 13-3**), which will become more relevant as CCTPs begin "localizing" areas of the heart to determine where ischemia, injury, and infarction have occurred. The colors in Table 13-3 will be used to represent these same leads and areas in figures later in this chapter.

It is also useful to know the intrinsic rates of cardiac cells because a problem may be located simply by the heart rate that is produced **Figure 13-17**. The higher in the conduction system in the heart, the faster the "firing rate" will be. The slower pacers lower in the conduction system will not "normally" fire as long as the higher pacers are functioning properly.

Systematic Approaches

For reading 12-lead ECGs, there are many standard methods of analysis, ranging from 5 to 12 or more steps. The method used is not as important as using one method to ensure following a consistent approach. Methods stated herein assume a basic understanding of the elements essential to the fundamentals of ECG interpretation and 12-lead ECGs.

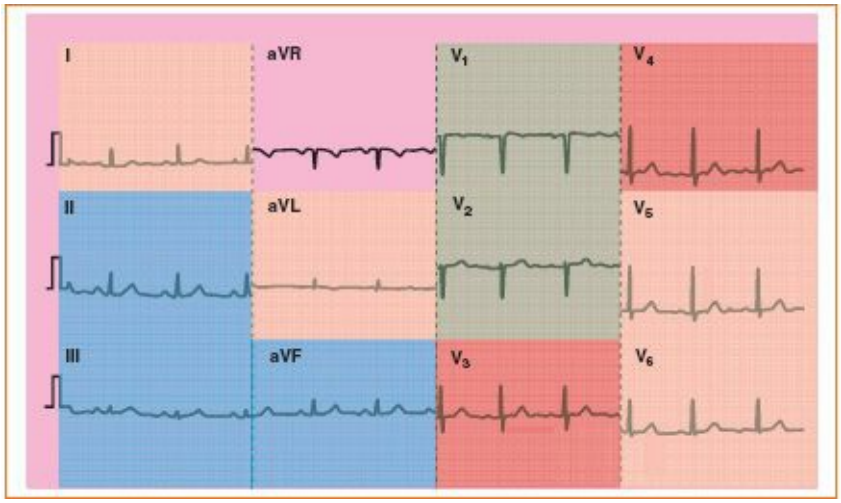


Figure 13-16 The relationship between leads and views of the heart.

TABLE 13-3 Leads Listed by the Area of the Heart That They View

Lead	Localized Area
V_1	Septum
V_2	Septum
V_3	Anterior
V_4	Anterior
II	Inferior
III	Inferior
aVF	Inferior
I	Lateral
aVL	Lateral
V_5	Lateral
V_6	Lateral
aVR	None

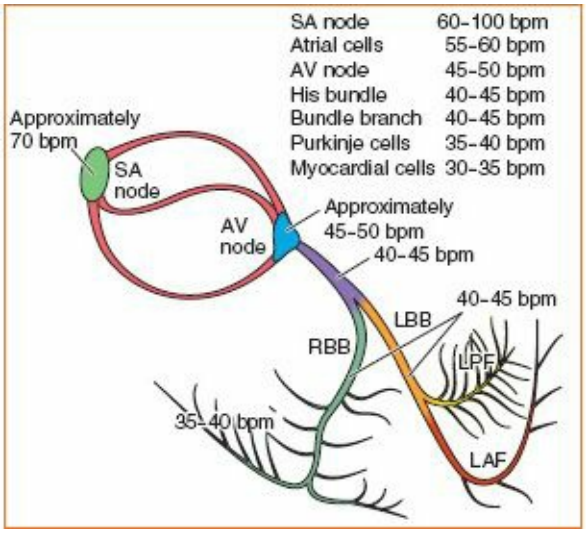


Figure 13-17 Intrinsic rates of pacing cells.

The keys to being able to quickly read an ECG are experience and, as stated, consistency. Interpretation of a 12-lead ECG is a skill that must be practiced repeatedly using a standard method of analysis. However, even among specialists examining a routine ECG tracing, there can be widely divergent conclusions. The reason this disparity is mentioned is to encourage the use of sound methods of interpretation, documentation of findings, and consideration of every piece of patient information.

During the transport of a critically ill patient, the ECG rhythm must be monitored continuously for changes that require a rapid, appropriate response as indicated by local protocol and national guidelines. In this setting, however, monitoring should not be mutually exclusive of serial evaluations of the 12-lead ECG. For systematic analysis of the 12-lead ECG, the interpretation of rhythm and arrhythmias is a fundamental building block. In rhythm and arrhythmia interpretation, there are, in general, 10 key elements

that must be assessed.

- General
  1. Is the rhythm fast or slow?
  2. Is the rhythm regular or irregular? If irregular, is it regularly irregular or irregularly irregular?
- P waves
  3. Are there any P waves?
  4. Are all the P waves the same?
  5. Does each QRS complex have a P wave?
  6. Is the PR interval constant? QRS complexes
  7. Are the P waves and QRS complexes associated with one another?
  8. Are the QRS complexes narrow or wide?
  9. Are the QRS complexes grouped or not?
  10. Are there any dropped beats?

Considering the ever-moving environment of patient transport, for CCTPs, a rapid assessment technique should be adapted to consistently review and identify changes seen on the 12-lead ECG. The approach shown in **Table 13-4** takes into consideration a variety of situations that must be identified and monitored and/or treated right away.

When examining an ECG tracing, CCTPs should make notes of findings as they are discovered to keep an accurate and a complete record of changes for serial evaluations. The ECG is only one simple test that can give information about the anatomy, pathology, and pathophysiology of the heart and also about how medications are affecting it.

The 12-lead ECG shows groupings of complexes that are recorded simultaneously by each lead. The rhythm strip, which typically appears at the bottom of the ECG, records for the entire time that the 12-lead ECG is being obtained. For comparison of cardiac electrical events, the series of complexes viewed in lead I is being recorded at the same time as the complexes in the other leads. Their appearance is different because of the angle at which the electrical vectors are viewed.

TABLE 13-4 Rapid 12-Lead ECG Assessment

Verify that aVR is negative (helps to ensure proper lead placement).
Assess rate and rhythm.
Determine the axis (leads I and aVF; also determined by the cardiac monitor's internal diagnostic hardware and printed on the 12-lead ECG).
Identify conduction abnormalities • Left bundle branch block • Hypertrophy • Aneurysm • Pericarditis • Drug or electrolyte imbalance effects • Early repolarization

Find signs of ischemia, injury, and infarction

- T-wave inversions
- ST-segment elevation
- Significant Q waves

Identify acute MI patterns

- Anterior
  - ST-segment elevation in V_1, V_2, V_3, V_4
  - ST-segment depression in leads II, III, aVF
- Inferior
  - ST-segment elevation in II, III, aVF
  - ST-segment depression in V_1, V_2, V_3, or I, aVL
- Lateral
  - ST-segment elevation in I, aVL, V_5, V_6
  - ST-segment depression in II, III, aVF
- Septal
  - ST-segment elevation in I, aVL, V_1, V_2
- Posterior
  - Tall, wide R waves and reciprocal ST-segment depression in V_1, V_2
  - T-wave inversion and ST-segment elevation in alternative posterior leads V_7, V_8, V_9
- Right ventricular
  - ST-segment elevations in V_4R, V_5R, V_6R

Consider the patient's clinical picture.

Axis Determination

As previously mentioned, each cell in the heart can produce its own electrical impulse. Each impulse varies in intensity and direction. The term vector is used to describe these electrical impulses, and the axis is the direction of the wave of depolarization as it passes through the heart. The sum of all of the electrical impulses is called the **mean electrical axis**.

As the electrical impulses or vectors travel through the heart toward the larger muscle mass of the left ventricle, the mean electrical axis should progress in a downward and leftward direction. Because the mean electrical axis is the sum of all vectors traveling in different directions, it makes sense that if there is a change in the patient's normal or preexisting axis, a change has occurred in the structure or function of the heart. For example, if there is hypertrophy in one of the ventricles, the sum of the electrical vectors would shift toward the enlarged ventricle. If there is an infarction, the area of myocardium affected will not transmit electrical impulses through it, causing a shift away from the infarction. If a section of the electrical conduction system is diseased or blocked, the impulses or vectors will shift away from the damaged area.

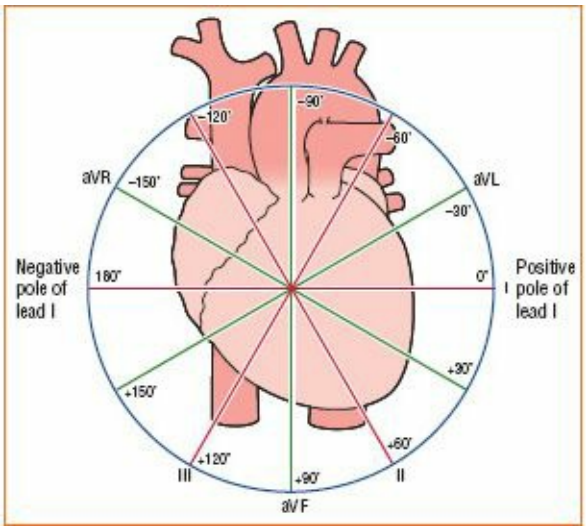


Figure 13-18 The hexaxial system.

Although the electrical axis is measured and evaluated by the internal diagnostic software of the cardiac monitor and printed on most standard 12-lead ECGs, any health care provider who "reads" ECGs must know how these determinations are made. This section shows how to determine the direction of the mean electrical axis.

The **hexaxial system** uses a circular diagram divided into 12 equal segments to describe the frontal plane that is created using the limb leads (I, II, III, aVR, aVL, and aVF) **Figure 13-18**. As mentioned, the mean electrical axis is the sum of all vectors generated in the wave of depolarization. To isolate the direction of the axis, two leads are required. To illustrate how this combination of leads will be used, the hexaxial circle is divided into four quadrants moving clockwise from 0° to +90°, +90° to 180°, 180° to –90° and –90° to 0°. A wave of depolarization directed into one of these quadrants will give the general direction of the mean electrical axis.

The QRS complex is a representation of the depolarization of both ventricles. The sum of all the vectors generated creates the mean QRS vector in a direction downward and to the patient's left. The mean QRS vector is used to determine the mean electrical axis.

The first lead examined to determine the direction of the mean QRS vector is lead I. Electrical impulses that flow toward the positive electrode create an upright deflection. An electrical impulse that flows away from the positive electrode has a negative deflection. Lead I is the positive lead located at 0°. As the wave of depolarization travels toward lead I, it is represented by a positive deflection on the ECG. If the QRS complex is negatively deflected in lead I, the wave of depolarization is shifted away from the left, which means that it is "deviated" toward the right, a right axis deviation (RAD).

The second lead examined to determine the axis is aVF. In aVF, the positive electrode is located on the left leg or lower left abdomen at least 10 cm from the heart. Electrically, this is at + 90°. If the wave of depolarization is downward, it is moving toward the positive electrode in lead aVF and will be represented by a positive deflection of the QRS on the ECG. If the QRS is positive in leads I and aVF, the mean electrical axis is downward (aVF) and toward the patient's left (lead I). This is the normal axis range **Figure 13-19**.

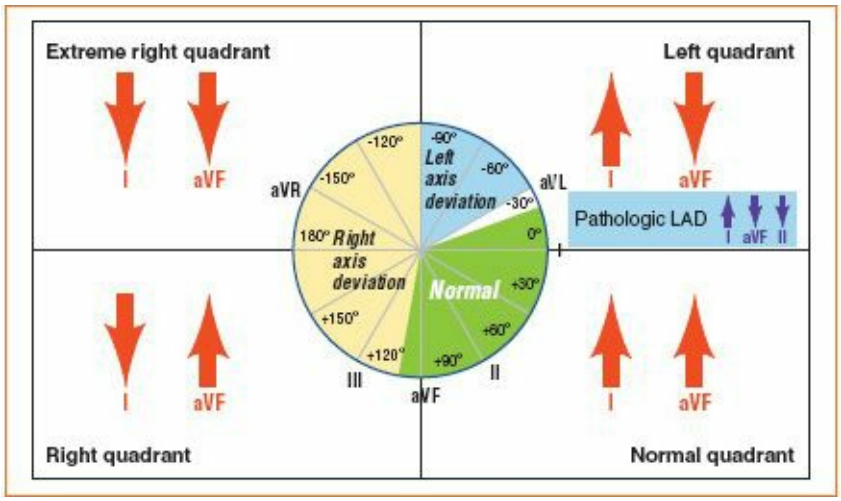


Figure 13-19 Electrocardiographic axis presentation of left axis deviation (LAD) and right axis deviation (RAD).

If the QRS complex is deflected positively in lead I, the axis is directed toward the left. A QRS complex that is negatively deflected in aVF at the same time that it is positive in lead I indicates that the axis is deflected upward and to the left, a left axis deviation (LAD).

If lead I is deflected negatively, the axis is deviated to the right. If aVF is deflected negatively at the same time, the axis is shifted upward and to the right, causing an extreme RAD. **Figure 13-20** shows sample ECG waves for practice in determining the quadrant in which the mean electrical axis is located.

Extreme RAD, –90° to 180°, is rare. It occurs when the heart is depolarizing inferiorly to superiorly. Extreme RAD can be seen in ventricular tachycardia (VT), atypical MIs, hyperkalemia, and, occasionally, right ventricular hypertrophy (RVH).

The mean QRS axis is in a "quadrant" of normal, RAD, extreme RAD, or LAD. Noting a change in electrical axis during the transport of a critically ill patient alerts CCTPs to search for the cause of the deviation. Once an abnormal axis is determined, other associated abnormalities can be found. Further evaluation can lead to an intervention. The reality in this setting is that noting the change of axis is more important than calculating the degree of axis. There are, however, some rapid assessment techniques that will help narrow down the degree of axis.

Before determining the degree of the mean electrical axis, it is important to know that there are some overlaps in the quadrants. The methods already discussed provide good approximations of the axis by isolating the quadrant in which it lies. The true pathologic measurements on the hexaxial system are shown in **Figure 13-21**.

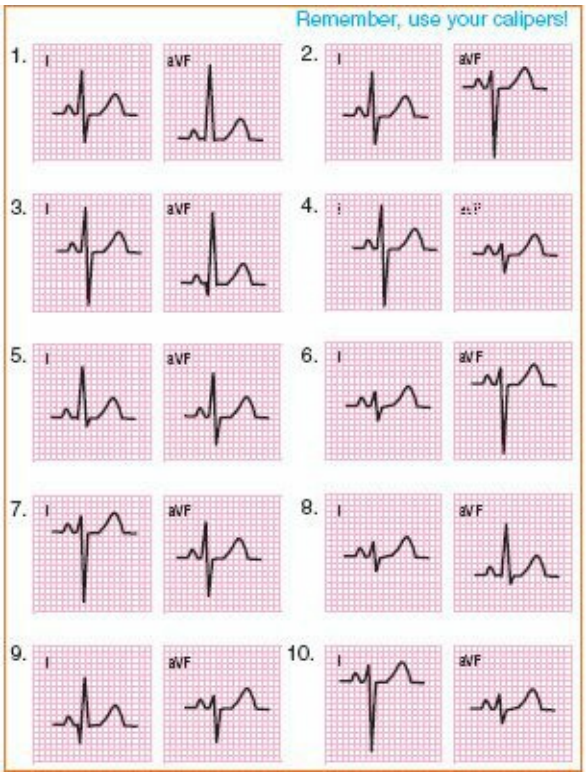


Figure 13-20 Sample electrocardiographic waves from which to determine the mean electrical axis.

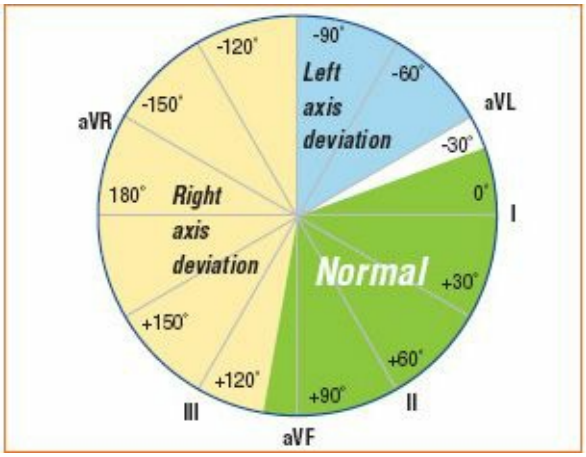


Figure 13-21 Pathologic measurements of the hexaxial system.

The normal ranges for the electrical axis extend from –20° to +100°. The 10° overlap from +90° to +100° in the right quadrant is not clinically significant. If the axis lies in either of the two right quadrants, the patient has an RAD.

In determining the left axis, it should be noted that "normal" extends to –20°. The area from –21° to –29° is considered the "physiologic" left axis and is neither pathologic nor normal. The area from –30° to –90° is the true pathologic area of the left quadrant, or LAD. The techniques already discussed should be used to isolate the axis to the left quadrant by determining if the QRS is positive in lead I and negative in aVF. If lead II is also negatively deflected, the left axis is between –30° and –90° and verifies a true pathologic LAD.

As shown in Figure 13-18, there are a number of ways to further calculate the direction and intensity of the electrical axis. An easy way to make this determination is to use the hexaxial system mentioned earlier. This system uses a circular diagram oriented in the frontal (coronal) plane. The diagram is divided into 12 equal segments around the heart, and each division of the diagram is labeled in degrees.

The divisions of the lower half of the diagram are given values ranging from 0° to 180°, and the divisions in the upper half of the diagram are each given a value of 0° to –180°. The limb leads (I, II, III, aVR, aVL, and aVF) are overlaid onto this diagram, each oriented to its electrical position on the ECG. Lead I is located at 0°, lead II at +60°, lead aVF at +90°, lead III at + 120°, aVR at –150°, and aVL at –30°. On the hexaxial diagram, each lead has a corresponding lead that is 90° away or perpendicular to the other **Figure 13-22**.

To use this system to determine the mean electrical axis, the QRS complex that has the lowest amplitude and is the most **isoelectric** in one of the limb leads is located first. Then, the corresponding perpendicular lead is found. For example, if the most isoelectric QRS complex is in lead II, located at + 60°, the corresponding perpendicular lead would be aVL, which uns between + 150° and –30°. Next, a determination is made about whether the QRS is deflected positively or negatively in the perpendicular lead. If it is positive, the mean electrical axis is + 150°. If the QRS is negatively deflected, the mean electrical axis is –30°. **Figure 13-23** shows both ends of each limb lead and their corresponding values. (The arrows show which is positive and which is negative.)

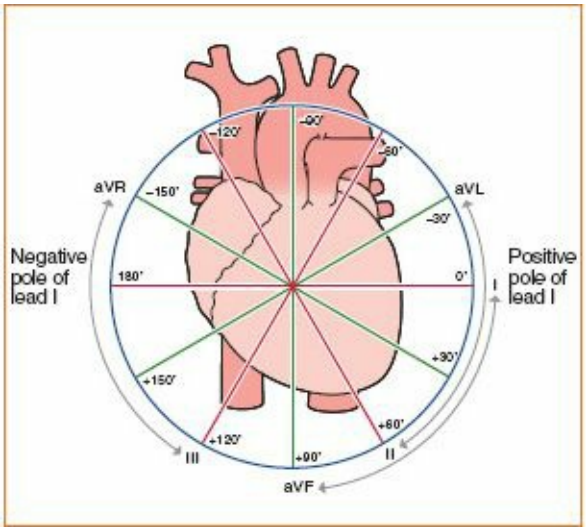


Figure 13-22 Perpendicular leads of the hexaxial system.

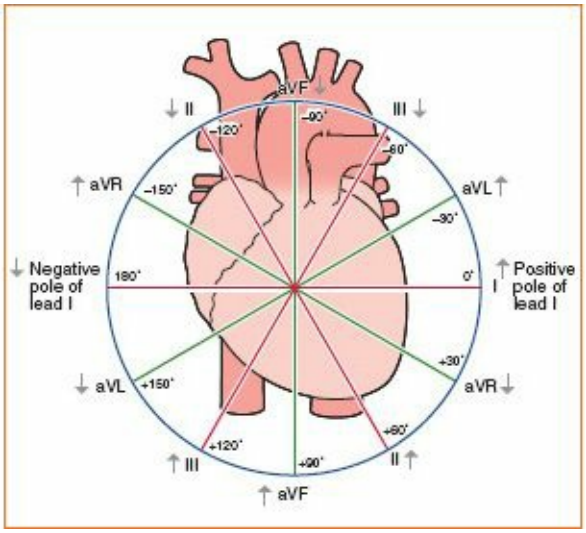


Figure 13-23 The hexaxial system with labeled limb leads, degrees, and arrows indicating the QRS deflection.

A baseline ECG on the cardiac monitor to be used during transport is required before transporting a critically ill patient. A complete assessment of the ECG should be made, noting any abnormalities,

including the mean electrical axis. An axis that deviates outside the normal range or is different from the patient's pre-existing axis usually has clinical implications. When an axis deviation is detected, CCTPs should always check for corresponding ECG changes that can provide clues to the underlying cause of the deviation.

Some common cardiac causes for axis deviation are ischemia and infarction. If the electrical conduction system is affected, the axis will be directed away from the damaged area. One of the most common areas affected is the left anterior-superior fascicle of the left bundle branch. The result is a left anterior hemiblock (LAH). If the wave of depolarization is stopped by an inferior wall MI (IWMI), the electrical impulse can be redirected upward and to the left, causing an LAD. With left ventricular hypertrophy (LVH), the enlarged ventricle can draw the electrical axis toward this larger muscle mass, resulting in LAD. Ectopic beats and rhythms can originate anywhere in the heart, directing the wave of depolarization in a right or left direction, causing an axis deviation in the affected area. An RAD is caused by events that redirect the electrical axis toward the right. A congenital cause is **dextrocardia**, which occurs when the heart develops in a right-facing position, creating a mirror image of the normal left-facing heart. Left posterior hemiblock (LPH) occurs with a conduction defect to the left posterior-inferior fascicle of the left bundle branch. Just as an enlarged left ventricle can cause the axis to shift left, an enlarged right ventricle, or RVH, can cause an RAD.

Other conditions that are not cardiac in origin can also contribute to a shift in axis. Any condition that causes the heart to be displaced from the downward leftward position can contribute to this shift. For example, a tall thin person can have a heart that is displaced (vertically) slightly more downward and away from the left, which can result in a right shift in axis. Children have larger right ventricles compared with adults, so their "normal" axis points toward the right. Chronic obstructive pulmonary disease can lead to RVH, causing an RAD. An LAD can also be caused by mechanical shifts. In obese or pregnant patients, the increased intra-abdominal pressure can push up on the diaphragm, displacing the heart into a more horizontal or left position, resulting in an LAD. **Table 13-5** summarizes possible causes of various axis deviations.

TABLE 13-5 Possible Causes of Axis Deviation

Axis Deviation	Possible Cause
Right	RVH LPH Chronic obstructive pulmonary disease Dextrocardia Ectopic beats and rhythms Normal in children
Left	LAH LVH IWMI Ectopic beats and rhythms Obesity Pregnancy

Abbreviations: IWMI, inferior wall myocardial infarction; LAH, left anterior hemiblock; LPH, left

posterior hemiblock; LVH, left ventricular hemiblock; RVH, right ventricular hypertrophy.

Bundle Branch Blocks and Hemiblocks

When a disruption of the electrical or mechanical response to the wave of depolarization occurs in the heart, bundle branch blocks (BBBs) and **hemiblocks** may occur. These events may be identified through characteristic changes on the ECG.

Right Bundle Branch Block

If there is a block in electrical conduction of the right bundle branch, CCTPs should think of how the wave of depolarization progresses. The electrical impulses that originate in the atria proceed as normal up to the block, with no change in waveforms or intervals. The impulses will continue down the left bundle branch and its fascicles as usual but are stopped in the right bundle branch **Figure 13-24**.

When a block occurs, the wave of depolarization must continue through the right ventricle by cell-to-cell conduction. Because cell-to-cell conduction progresses more slowly than conduction through the normal electrical conduction pathway, the representative complex displayed on the ECG will occur late as well, causing a prolonged QRS interval of 120 milliseconds or more. The ECG initially shows the upstroke of the R wave conducted by the left ventricle and its fascicles. Immediately behind this is the delayed right ventricle depolarization resulting in a second R wave called R′ (R prime). The combination of the normally conducted electrical impulse through the left bundle branch and the delayed conduction through the right bundle branch results in the up and down (“rabbit ear”) appearance of the QRS complex **Figure 13-25**.

The best lead to view the changes associated with a right BBB is the precordial lead, V_1, which shows the RSR′ complex. Lead V_2 can also show the RSR′. The RSR′ complex is one in which a second R wave appears in the second half of the QRS complex. The R′ representing the additional vector created by the delayed electrical impulse through the right ventricle will be positively deflected in lead V_1. In addition to the RSR′ seen in lead V_1, the delayed conduction through the right ventricle causes a “slurring” of the S wave in the lateral limb lead, lead I, and the left-sided precordial lead, V_6. Because other conditions can cause an RSR′ (eg, incomplete right BBB, some pulmonary conditions, RVH, and preexcitation syndromes such as Wolff-Parkinson-White syndrome), for an RSR′ to be diagnostic of a right BBB, the slurred S wave is required to make the diagnosis. **Figure 13-26** represents the various leads on an ECG and shows relevant areas for a right BBB diagnosis. Note: This style of illustration is used repeatedly in this chapter.

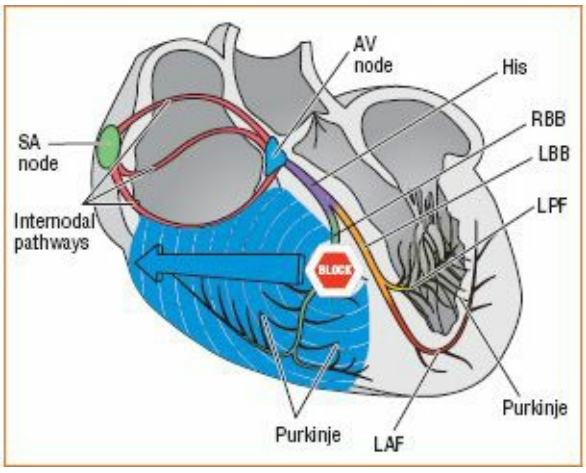


Figure 13-24 Right bundle branch (RBB) block. AV indicates atrioventricular; LAF, left anterior fascicle; LBB, left bundle branch; LPF, left posterior fascicle; SA, sinoatrial.

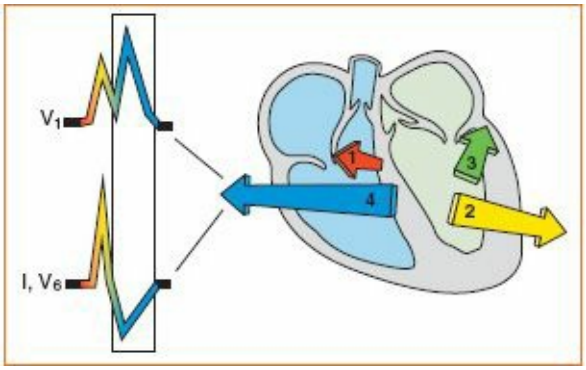


Figure 13-25 The effect of a right bundle branch block on the electrocardiogram.

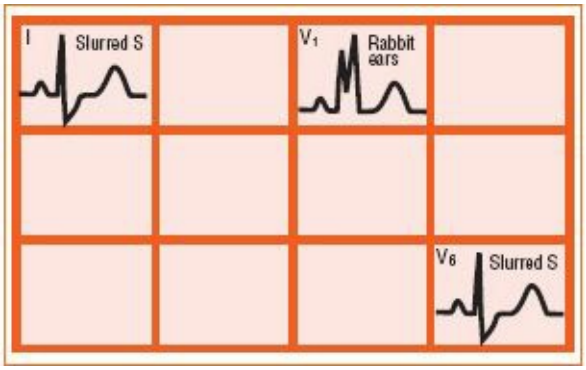


Figure 13-26 Electrocardiographic changes seen with a right bundle branch block.

Therefore, the main criteria for the diagnosis of right BBB are as follows:

1. QRS prolongation of 120 milliseconds or more
2. Slurred S wave in leads I and V_6
3. RSR′ pattern in V_1 with the R′ wave taller than the R wave

A right BBB can occur with disruption of the electrical conduction system, and it can be present in a normal healthy heart.

The QR′ Wave

Of the many potential causes of an R′ in lead V_1, the one that demands the most attention is the Qr′ or qR′ wave (when written, the larger of the two waves is represented by the uppercase letter) **Figure 13-27**.

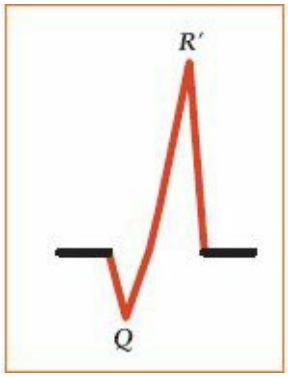


Figure 13-27 QR′ wave.

V_1 is one of the precordial leads that represents the depolarization of the anterior septum (V_2 is the other). A Q wave that occurs in lead V_1 in the presence of a right BBB is evidence of anteroseptal ischemia or infarction. If this waveform appears during transport of a patient in critical condition, early intervention may prevent ischemia from progressing to infarction.

Figure 13-28 provides an example of a right BBB. With practice, using pattern scanning of the ECG, CCTPs will see the RSR′ in the precordial leads that represent septal depolarization. Because the RSR′ alone is not sufficient to make a diagnosis of right BBB, the lateral leads, I and V_6, should be scanned.

There is no specific treatment required for isolated right BBB. The patient should, however, be seen for periodic follow-up evaluations.

Left Bundle Branch Block

A left BBB is caused by a disruption of the electrical conduction of the left bundle or both fascicles (the **left anterior fascicle** and the **left posterior fascicle**) of the left bundle. The electrical impulses that originate in the atria proceed as normal, with no change in waveforms or intervals up to the block. The electrical impulses continue to flow unimpeded down the right bundle. With the electrical conduction of the left bundle branch blocked, the wave of depolarization must then proceed by cell-to-cell transmission beginning right to left **Figure 13-29**.

The delay of electrical conduction in the left bundle branch or its fascicles results in a complex that is 120 milliseconds or longer in duration. Because normal ventricular depolarization proceeds down and to the left, the inferior and lateral leads (I, V_5, and V_6) will show the most direct results of the block. With left BBB, the complexes in these leads will not have Q waves and will typically have tall monomorphic R waves. When the conduction of electrical impulses is delayed, the normal sharp rise of the R wave is replaced with a more gradual or bowed, upward appearance. The peak of the R wave is notched, wide, or both. Reciprocal findings are present in the right-sided chest leads with wide, deep, monomorphic S waves. Repolarization is also affected in BBBs. In a left BBB, the T waves should be **discordant** or deflected opposite of the terminal deflection of the QRS complexes **Figure 13-30**.

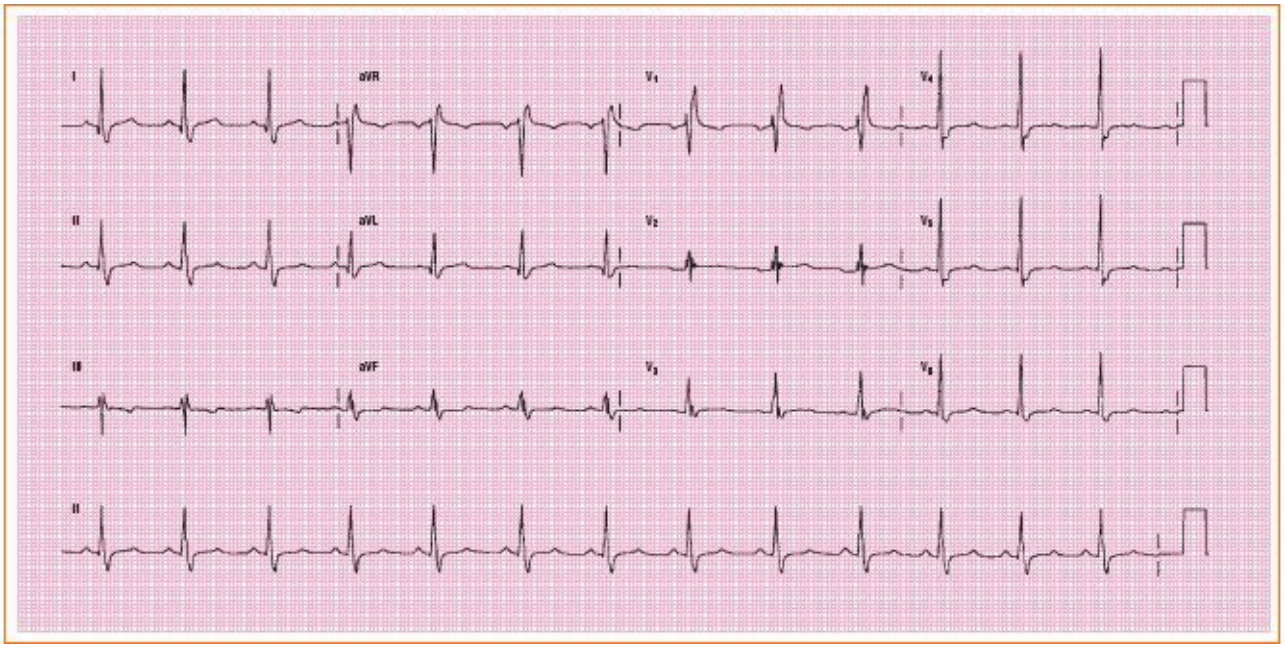


Figure 13-28 A 12-lead electrocardiogram showing a right bundle branch block.

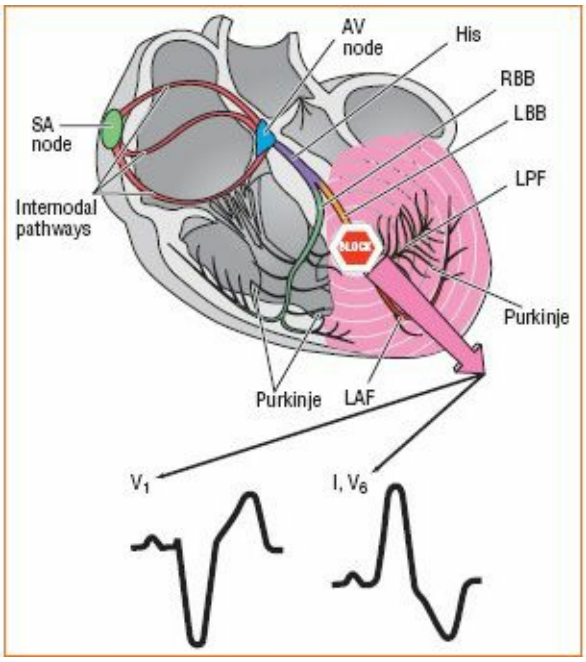


Figure 13-29 Left bundle branch (LBB) block. AV indicates atrioventricular; LAF, left anterior fascicle; LPF, left posterior fascicle; RBB, right bundle branch; SA, sinoatrial.

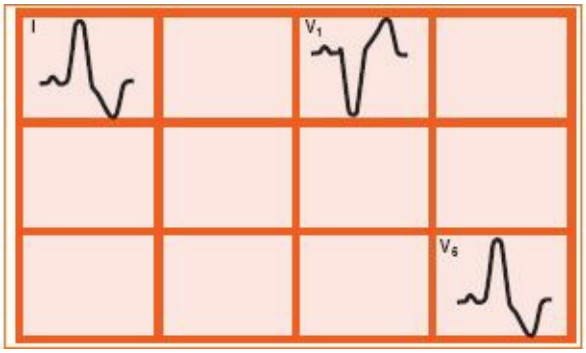


Figure 13-30 Electrocardiographic changes seen with a left bundle branch block.

The main criteria for the diagnosis of a left BBB are as follows:

1. Complex duration of 120 milliseconds or longer
2. Wide R waves in leads I and V_6, with no Q wave
3. Wide, deep S waves in lead V_1; may also have a small r wave

Lowercase letters are used to represent small waves, where as capital letters are used to represent large waves. Therefore, “r wave” represents a small r wave, whereas “R wave” represents a large R wave.

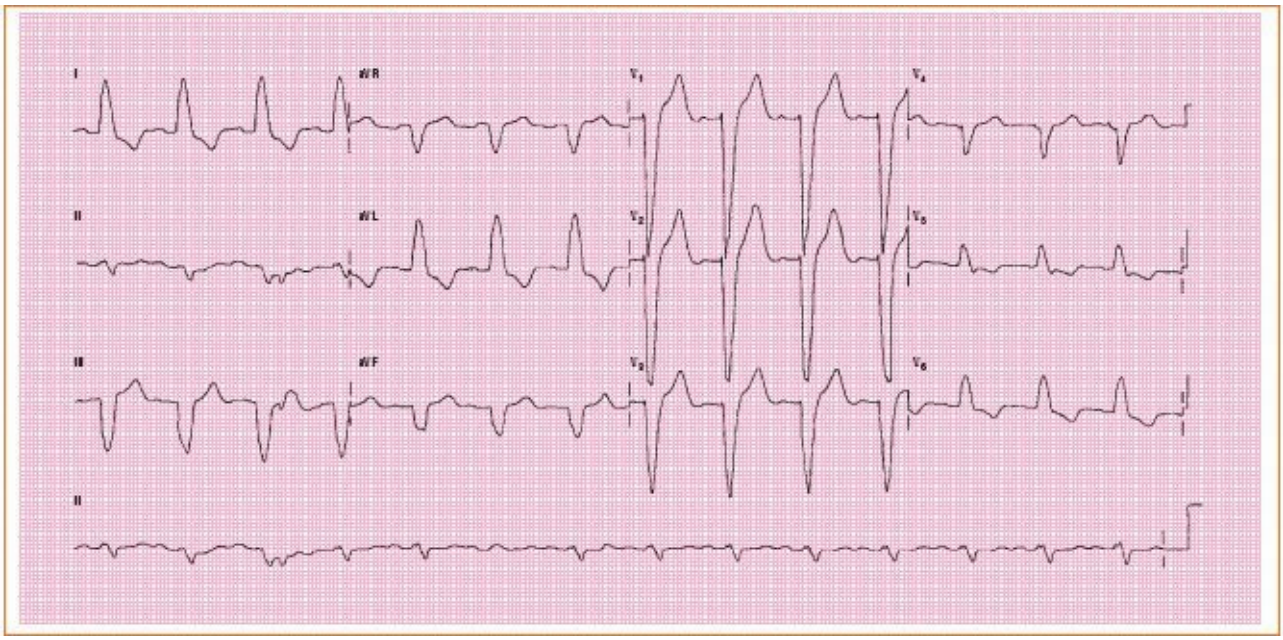


Figure 13-31 A 12-lead electrocardiogram showing a left bundle branch block.

Although a right BBB can be present in a healthy heart, a left BBB seldom occurs in a healthy heart and usually means that there is a serious problem within the conduction system or ischemia from coronary artery disease. A pacemaker rhythm can also give wide complexes, so CCTPs should be watchful for pacemaker spikes with a rhythm that has this appearance.

Figure 13-31 is an example of a left BBB. The pattern of wide complexes of 120 milliseconds or longer and discordant T waves in every lead indicates that this is a disturbance that affects the ventricular conduction system. The presence of P waves, however, provides assurance that the rhythm originates in the atria and is not in the ventricles. The QRS complexes are deflected positively in the lateral leads (I, V_5, and V_6) with corresponding wide, deep S waves in the reciprocal lead, V_1.

Patients with a left BBB require complete cardiac evaluation, and patients with a left BBB and near-syncope or syncopal episodes may require a pacemaker. Guidelines for device-based therapy of cardiac rhythm abnormalities have been established and revised by the American College of Cardiology and the American Heart Association.

Hemiblocks

A hemiblock is a block in the electrical conduction of one of the two fascicles of the left bundle branch. An LAH is the same as a left anterior fascicular block, and an LPH is the same as a left posterior fascicular block. Unlike complete BBBs, hemiblocks cause little to no widening of the QRS complexes because the resultant cell-to-cell transmission of the electrical impulses is much less.

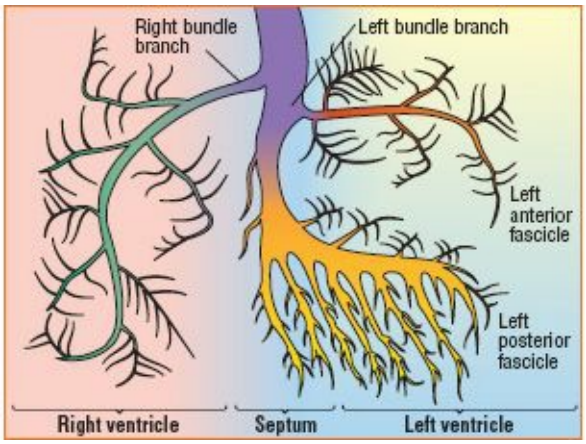


Figure 13-32 Ventricular electrical conduction system.

Each fascicle branches off of the left bundle branch, and the structures of the two are quite different **Figure 13-32**.

The left anterior fascicle forms a thin bundle of fibers that conducts electrical impulses to the anterior and lateral walls of the left ventricle. The left posterior fascicle forms a broad network of fibers that conducts electrical impulses to the inferior and posterior walls of the left ventricle. Observing the structural differences of these two fascicles makes it easy to understand why a hemiblock occurs more frequently in the left anterior fascicle. It would take a smaller area of ischemia or infarction to impede the electrical conduction to a single strand of fibers than to block the conduction to a broad network of fibers. Even though the blood supply to all areas of the heart varies somewhat from person to person, the left posterior fascicle typically receives blood from branches of the left and right coronary arteries, which means that coronary occlusions would have to occur in both sources of blood supply to have a major effect on the left posterior fascicle.

Left Anterior Hemiblock

An LAH occurs when electrical conduction to the left anterior fascicle is blocked **Figure 13-33**.

With conduction to the anterior-superior part of the left ventricle blocked, depolarization of this area is from the interventricular septum and retrograde conduction from the inferior and posterior walls. The direction of the wave of depolarization for this blocked area is now spreading upward and to the left, causing a shift in the electrical axis to the pathologic left, which makes an LAD between –30°and –90° the main criterion for the diagnosis of LAH. Additional findings on the ECG include a qR complex or a large R wave in lead I. An rS complex also may be found in lead III. These small q and r waves are the result of the interventricular septum depolarizing unopposed.

The criteria for diagnosing LAH are as follows:

1. Left axis deviation within –30° to –90°
2. qR complex or large R wave in lead I
3. rS complex in lead III (likely to also appear in lead II and aVF)

Figure 13-34 is an example of an LAH. The most obvious feature is an LAD. The QRS complex is positively deflected in lead I and negatively deflected in lead aVF. To further isolate this vector to the pathologic range or –30° to –90°, lead II should be examined. Negative deflection, as in this example, confirms the diagnosis of LAD and is presumptive of LAH. Other criteria for LAH are also met in this example. There is a qR complex in lead I, and there are rS complexes in leads II, III, and aVF. The Q waves that appear in leads I and aVL are insignificant. They are normal and represent the first vector of ventricular depolarization. For a Q wave to be significant and represent an MI, it must be more than one third the total height of the QRS complex that it appears with and must also be more than 40 milliseconds in duration.

Left Posterior Hemiblock

An LPH is exceedingly rare because of the dual blood supply to the area and the broad network of fibers that have to be blocked **Figure 13-35**. In addition to being rare, LPH is more difficult to diagnose. When the depolarization of the inferior and posterior portions of the left ventricle is delayed, the resultant wave of depolarization is directed further inferior and to the right. This rightward deflection results in an RAD. The electrical impulses conducted up to the block are unopposed and depolarize the interventricular

septum and the superior-anterior walls of the left ventricle. This is shown on the ECG as a small r wave in lead I and a small q wave in lead III.

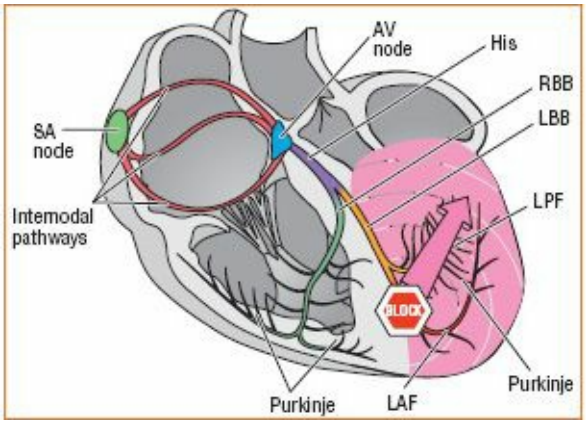


Figure 13-33 Left anterior hemiblock. AV indicates atrioventricular; LAF, left anterior fascicle; LBB, left bundle branch; LPF, left posterior fascicle; RBB, right bundle branch; SA, sinoatrial.

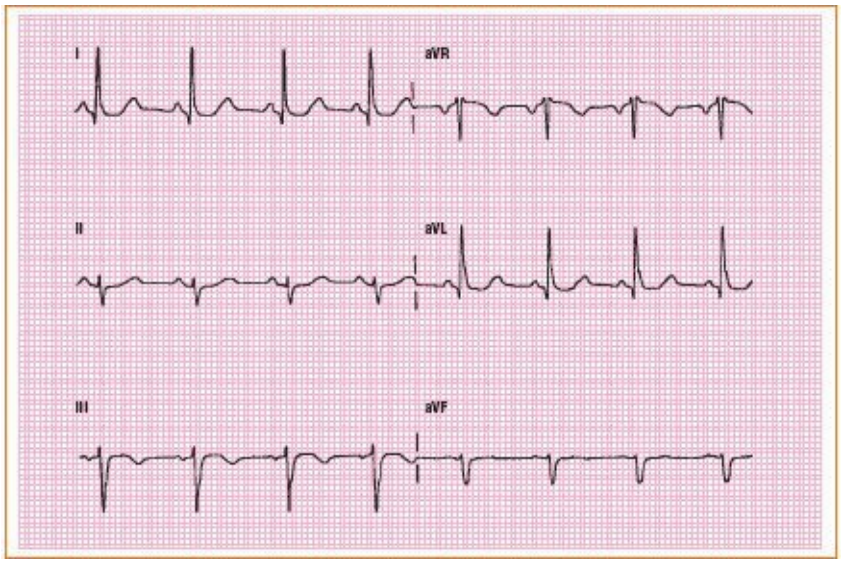


Figure 13-34 A 12-lead electrocardiogram showing a left anterior hemiblock.

The criteria for diagnosing LPH are as follows:

1. RAD of +90° to 180°
2. r wave in lead I and q wave in lead III
3. Exclusion of right atrial enlargement and/or RVH

An LPH is a diagnosis of exclusion—one reason it is more difficult to diagnose. Other causes of RAD, such as right atrial enlargement and RVH, must be ruled out. Certain chronic lung diseases that cause an overload of the right atrium can be the cause of an RAD. With these additional factors to consider in diagnosing LPH, when an RAD is found, an r wave in lead I and a q wave in lead III should be sought. With no other evidence of right atrial enlargement or RVH, LPH needs to be considered.

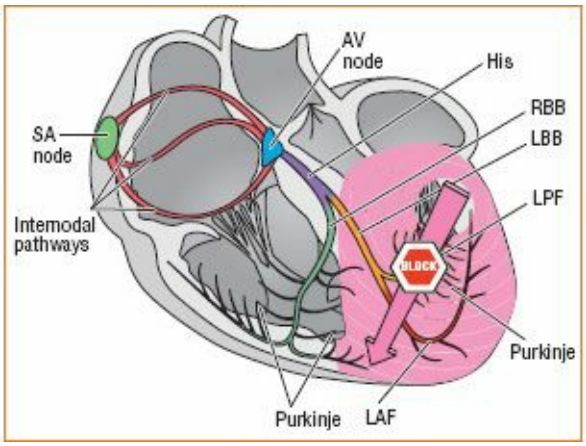


Figure 13-35 Left posterior hemiblock. AV indicates atrioventricular; LAF, left anterior fascicle; LBB, left bundle branch; LPF, left posterior fascicle; RBB, right bundle branch; SA, sinoatrial.

Figure 13-36 is an example of an LPH. The figure shows an RAD. There is also an s wave in lead I and an insignificant q wave in lead III. There is no evidence of right atrial enlargement. A pulmonary condition that makes the diagnosis of LPH impossible in many cases is pulmonary embolism. In 15% to 30% of patients with a pulmonary embolism, there is an ECG pattern of $S_1Q_3T_3$ caused by acute right ventricular strain (S_1Q_3 is also a criterion for LPH). This pattern has an s wave in lead I and a q wave and an inverted T wave in lead III. In this situation, a clinical history suggestive of pulmonary embolism is important. A thorough history and a reason for transport should be ascertained.

Bifascicular Blocks

As the name of this condition implies, two conduction pathways are blocked at the same time. A **bifascicular block** includes concurrent findings of right BBB with LAH or LPH.

When right BBB with LAH occurs as a chronic condition, it is considered stable. A new onset of bifascicular block with ischemia, however, is not stable. This presentation will show a right BBB pattern with a slurred s wave in leads I and V_6 and an up-and-down (rabbit ear) appearance of the RSR′ in V_1. The QRS duration will be 120 milliseconds or longer. In addition, with LAH, the typical pattern will include an LAD and rS waves in lead III.

A bifascicular block with a right BBB and an LPH is always considered an unstable rhythm. Owing to the amount of myocardium that has to be damaged to cause a true LPH, it commonly involves other conduction pathways of the ventricles (tight bundle branch). This block often deteriorates into a complete heart block, especially in the presence of an acute MI. Because of the extensive damage it takes to cause an LPH, it does not take much more to progress to a higher degree of block. When a bifascicular block includes a right BBB and an LPH, the typical right BBB pattern with RAD and a small q wave in lead III will be found. **Figure 13-37** illustrates two bifascicular block patterns. The right BBB is in black, the LAH is in blue, and the LPH is in green.

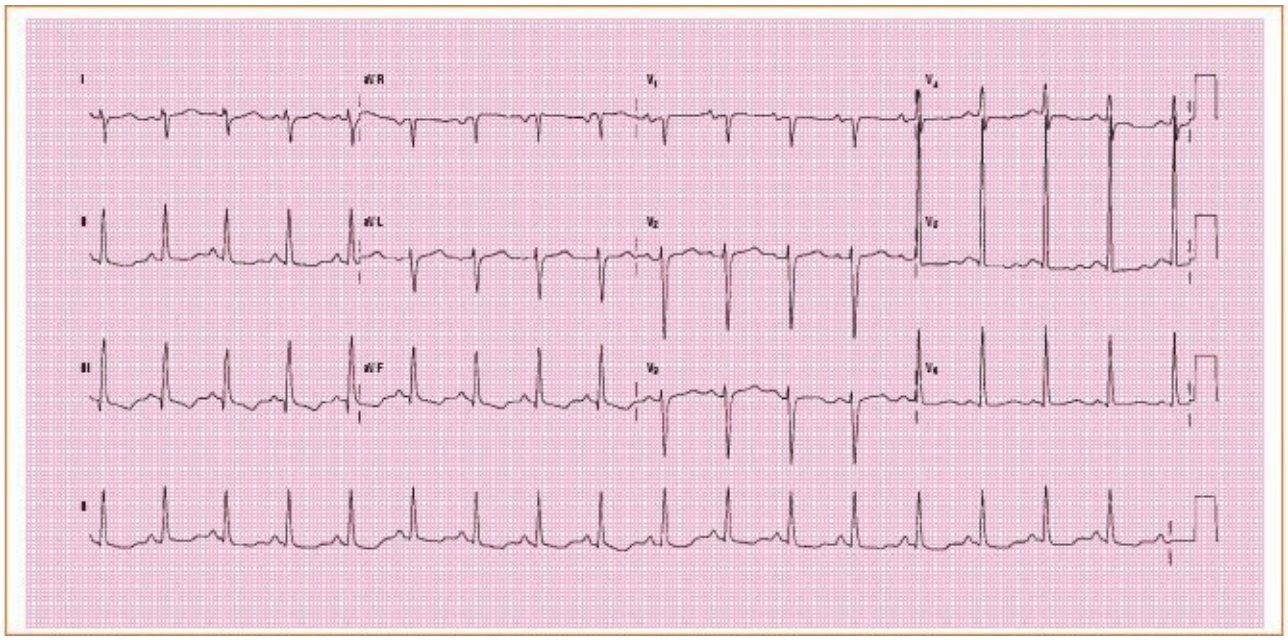


Figure 13-36 A 12-lead electrocardiogram showing a left posterior hemiblock.

Figure 13-38 provides an example of a bifascicular block that includes a right BBB with an LAH. First, the ECG should be scanned for abnormal patterns. In the example, the QRS intervals are 120 milliseconds or more. There is a slurred S wave in leads I and V_6. An RSR′ is present in lead V_2. (RSR′ can be found in the septal precordial leads V_1 and/or V_2.) These criteria so far indicate a right BBB. The evaluation is continued, and the mean electrical axis is determined. The QRS complex is positively deflected in lead I and negatively deflected in aVF, indicating an LAD. To further isolate the axis, lead II is examined. It is negatively deflected, placing the axis between –30° and –90°, the pathologic left axis. This finding provides the additional criterion to determine that an LAH is also present. Bifascicular blocks with a right BBB and an LAH are generally stable unless they occur in the presence of an acute MI. The additional ischemia and tissue death could take out the remaining left posterior fascicle, causing a complete heart block. In a critically ill cardiac patient with an existing bifascicular block consisting of a right BBB and an LAH, signs of ischemia or infarction indicate the need for an acute pacemaker placement, especially in the presence of a second- or third-degree AV block. This procedure will not likely be available in the transport setting, so medical control should be contacted and the patient should be prepared for external cardiac pacing. The receiving facility may need to be urgently prepared for pacemaker placement and should be notified of developments so that an implantable pacemaker will be available without delay on arrival.

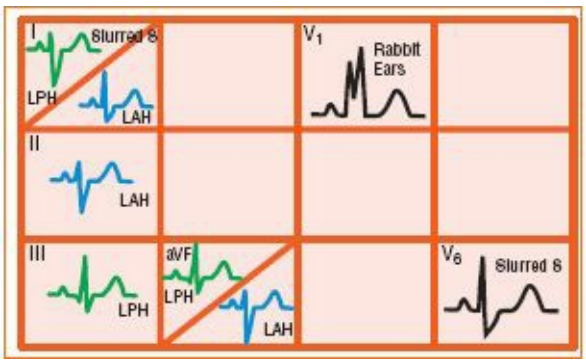


Figure 13-37 Electrocardiographic changes seen with a bifascicular block.

Figure 13-39 provides an example of a bifascicular block with a right BBB and an LPH. First, the

ECG is scanned for abnormal patterns. Slurred S waves are found in leads I and V_6. There is an RSR′ in V_1 consistent with a right BBB. The axis is determined to be in the right quadrant because the RSR is negatively deflected in lead I and positively deflected in aVF. To further isolate the axis, the most isoelectric limb lead with the smallest amplitude should be found. In this example, it is lead aVR. In the hexaxial system, the perpendicular lead (90° away) to aVR is lead III. Lead II should be assessed to determine if it is positively or negatively deflected. In this case, lead II is positive, placing the mean electrical axis at + 120° (RAD). There is also an S wave in lead I and a qS complex in lead III, consistent with an LPH. There are no exclusionary findings to contradict a diagnosis of LPH. Finally, in this example, there is also a first-degree heart block. This pattern of a right BBB with an LPH is inherently an unstable rhythm owing to the extensive myocardial damage necessary to cause the LPH in the first place. Additional ischemia or even a small infarction could extend the conduction defect to include the anterior fascicle as well.

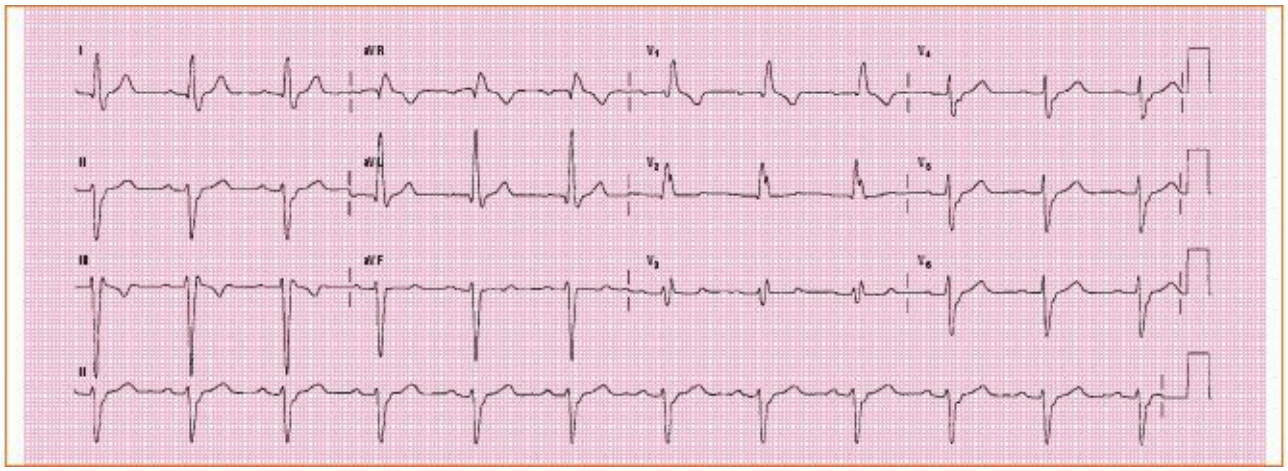


Figure 13-38 A 12-lead electrocardiogram showing a bifascicular block with a right bundle branch block and a left anterior hemiblock.

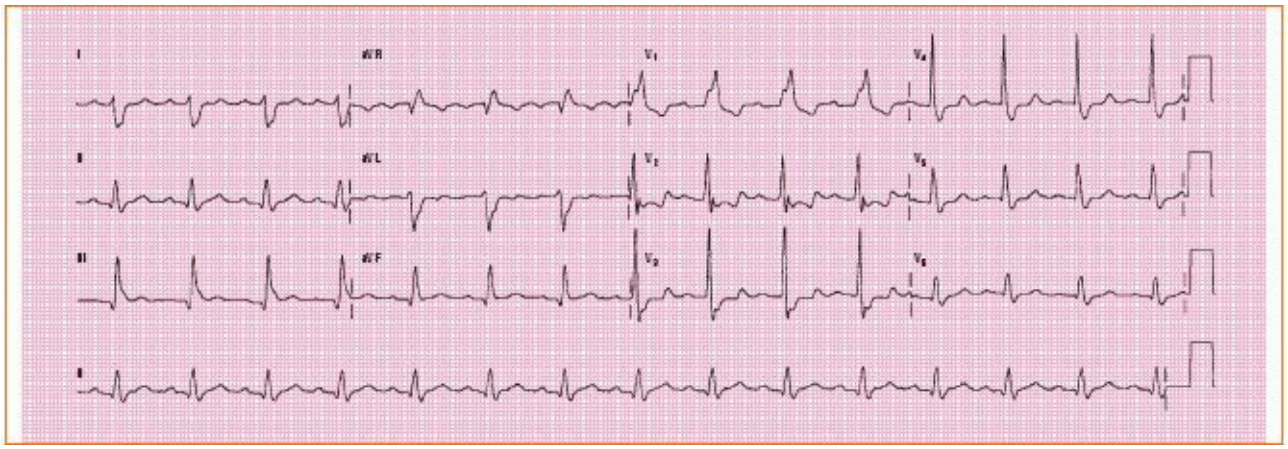


Figure 13-39 A 12-lead electrocardiogram showing a bifascicular block with a right bundle branch block and a left posterior hemiblock.

Trifascicular Block

The term **trifascicular block** is used to describe the combination of bifascicular block (right BBB with a block in the left anterior fascicle or left posterior fascicle) that occurs with a first-degree heart block (prolonged PR interval). Trifascicular block is important to diagnose because it is difficult to tell if the prolonged PR interval seen with the first-degree heart block component is the result of disease in the AV

node or to diffuse distal conduction system damage. If the block at the AV node level becomes a complete heart block, the escape rhythm that will originate is from the bundle of His. This escape rhythm will produce heart rates in the 40s and be symptomatic for fatigue, near-syncope, or complete syncopal episodes. If there is diffuse conduction system damage, the escape rhythm may be fascicular or ventricular, which may result in heart rates that are life-threateningly low.

The presence of a trifascicular block after an MI implies extensive cardiac damage. True trifascicular blocks require immediate temporary pacing followed by the placement of a permanent pacemaker.

Selected ECG Findings

The following ECG findings have been selected for review because they can appear alone or coexist with the conditions already discussed. They may help CCTPs identify the many changes in structure and function that occur with chronic conditions or during an acute cardiac event and, hopefully, help put the pieces of the clinical puzzle together and show how they reflect onto the ECG.

Hyperacute T Waves

Within the first few minutes of coronary occlusion, the T wave can become tall and narrow because of the ischemia that is present. This is called peaking and is sometimes referred to as a hyperacute T wave. This presentation is transient, beginning within minutes to a few hours before the T wave inverts (flips). The first change that might appear with hyperacute T waves is an upward slanting of the ST segment and a subtle enlargement of the T wave that is disproportionate to the QRS complex. The hyperacute T waves are localized to the area of ischemia and infarction and may be associated with depression of the J point and a prolonged QT interval. The hyperacute T waves are different from those seen in other conditions. In hyperkalemia, for example, the T wave peaking will be tall and narrow and have a "tenting" appearance. These T-wave changes will appear through all leads of the ECG. Hyperacute T waves that appear as a result of ischemia and infarction appear only in leads that view the area of ischemia and infarction.

ST-Segment Elevation

ST-segment elevation is caused by changes that affect ventricular depolarization and repolarization. Non-MI changes can also cause this condition and include left BBB, ventricular rhythms, LVH, pericarditis, and early repolarization.

As the complete ECG picture of infarction evolves, within a few hours the ST segment usually returns to baseline. A persistent ST-segment elevation may indicate the formation of a ventricular aneurysm.

In addition to myocardial injury, ST-segment elevation can be seen in a number of other conditions, such as early repolarization, and is made evident by elevation of the J point. The J point is where the ST segment "takes off" from the QRS complex, and elevation in the absence of other findings has no pathologic implications. The way to determine the difference between benign elevation of the J point and elevation caused by myocardial injury is by the distinctive configuration seen with myocardial disease. In benign J-point elevation, the T wave is clearly distinguished as a separate wave. With myocardial disease, the elevated J point bows upward and merges with the T wave **Figure 13-40**.

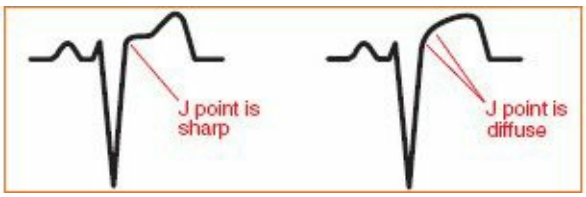


Figure 13-40 Sharp and diffuse J points.

Differential Diagnosis

ST-Segment Elevation

- Myocardial disease
- Left BBB
- Ventricular rhythms
- LVH
- Pericarditis
- Early repolarization
- Ventricular aneurysm

Hypertrophy

Hypertrophy is thickening or excessive growth; in this chapter, it refers to any chamber of the heart. Hypertrophy is an increase in muscle mass caused by an increased workload on the heart when it has to pump against resistance. Dilation is an enlargement of the chamber cavity, and both conditions can occur together. The end result is that the heart has a larger mass in the affected area. As discussed in the section on axis determination, the larger the muscle mass, the higher the concentration of electrical impulses or vectors.

Atrial Enlargement

Left atrial enlargement results in a prolonged electrical conduction time through the left atrium. With normal electrical conduction being initiated by the SA node in the right atrium, the initial impulse is transmitted without delay through this smaller chamber and is recorded on the ECG as the beginning or leading side of the P wave. As the electrical impulse is delayed through the enlarged left atrium, it gives rise to the end or trailing side of the P wave. Overall, the resulting P wave has a notched double-humped appearance **Figure 13-41**.

A P wave that is greater in duration than 120 milliseconds in limb leads I and II and has a notched appearance is known as **P mitrale**.

Right Atrial Enlargement

Right atrial enlargement is caused by cardiac and pulmonary conditions affecting the pressures in the right atrium. Causes include mitral stenosis or regurgitation, chronic obstructive pulmonary disease, and pulmonary emboli. It occurs commonly in the pulmonary condition **P pulmonale**, characterized by tall, peaked P waves found in leads II and III that have an amplitude of 2.5 mm (2.5 mV) or more. P waves can be peaked, but must be 2.5 mm or higher; if they are less than 2.5 mm, they are not typically associated with right atrial enlargement **Figure 13-42**.

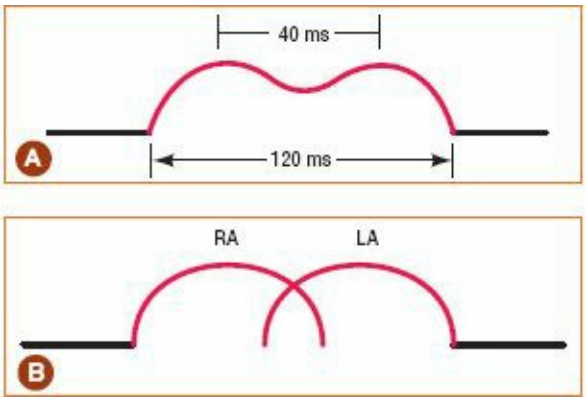


Figure 13-41 **A.** A notched P wave greater than 120 milliseconds in the limb leads shows P mitrale. **B.** The cause of a notched P wave. LA indicates left atrium; RA, right atrium.

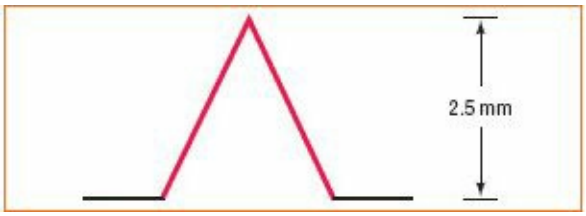


Figure 13-42 P pulmonale.

When the evidence suggests atrial enlargement, but the measurements are not enough to make the diagnosis, some different P-wave patterns might emerge. **Biphasic** P waves in lead V_1 are common. The biphasic P wave indicates nonspecific **intra-atrial conduction delay**. Often, biphasic P waves seen in IACD are related to atrial enlargement, but the wave is not significant enough to be classified as P mitrale or P pulmonale.

Comparing the P waves in leads V_1 and V_6 can be helpful. When the leading half of the P wave is taller and wider in V_1 than it is in V_6, right atrial enlargement is probable. If the trailing half of the P wave is wider and deeper than the leading upright half is in V_1, left atrial enlargement is probable. If the trailing end of the P wave is wider and deeper than one small block on the ECG (1 mm in height by 40 milliseconds in duration), left atrial enlargement is likely.

Both atria can be enlarged at the same time, referred to as biatrial enlargement. To make the determination of biatrial enlargement, any combination of the aforementioned criteria can be applied. For example, in the inferior leads II, III, and aVF, with lead II being the best view for left atrial enlargement, a notched P wave greater than 120 milliseconds in duration or a tall, peaked P wave of 2.5 mm or more in amplitude is found. Then in lead V_1, there is a biphasic P wave meeting the criteria for right or left atrial enlargement. Any combination of right atrial enlargement and left atrial enlargement equals biatrial enlargement **Figure 13-43**.

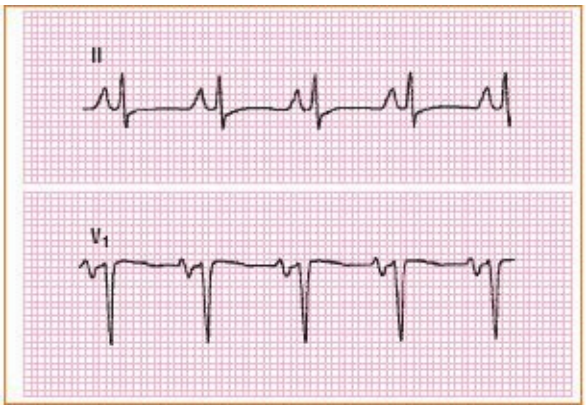


Figure 13-43 Biatrial enlargement.

Left Ventricular Hypertrophy

An enlargement or an increase in muscle mass of the left ventricle is known as LVH. LVH is the end-organ response to increased pressure or overload seen in hypertension. With increased detection and aggressive treatment of hypertension, there is a relative decrease in the ECG incidence of LVH. Patients who have evidence of LVH on the ECG have a higher risk of developing coronary artery disease, making them more susceptible to the sequelae of that disease. People who have LVH are actually more susceptible to increased mortality than people who experience an MI without an LVH.

Although a normal axis is the most common, a physiologic LAD is often present. A pathologic left or right axis can occur with LVH, but the hypertrophy is usually not the cause. In general, the increased R wave amplitude in the leads that look at the left ventricle forms the basis of an ECG diagnosis of LVH. The increased amplitude of waves seen on the ECG, in particular the precordial leads, is caused by a two-fold process. First, as the left ventricle enlarges, there is a greater concentration of vectors, increasing the wave of depolarization toward the precordial leads. Second, the increase in muscle mass typically extends anteriorly, bringing the heart physically closer to the electrodes on the chest. This positioning may be the reason for a normal axis instead of a pathologic axis deviation.

There are several ECG criteria used to determine LVH; however, for simplification, increased R wave amplitude in leads overlying the left ventricle and increased S wave amplitude in leads overlying the right ventricle are discussed. To assess for LVH, the deepest S wave in V_1 or V_2 is found, followed by the tallest amplitude of R wave in V_5 or V_6. If the sum of the deepest S wave and the tallest R wave is 35 mm or more, LVH is likely. The following criteria are very useful in making a determination of LVH. The more criteria that are met, the greater the likelihood that the patient has LVH.

1. Any precordial lead of 45 mm or more
2. An R wave in aVL of 11 mm or more
3. An R wave in lead I of 12 mm or more
4. An R wave in lead aVF of 20 mm or more

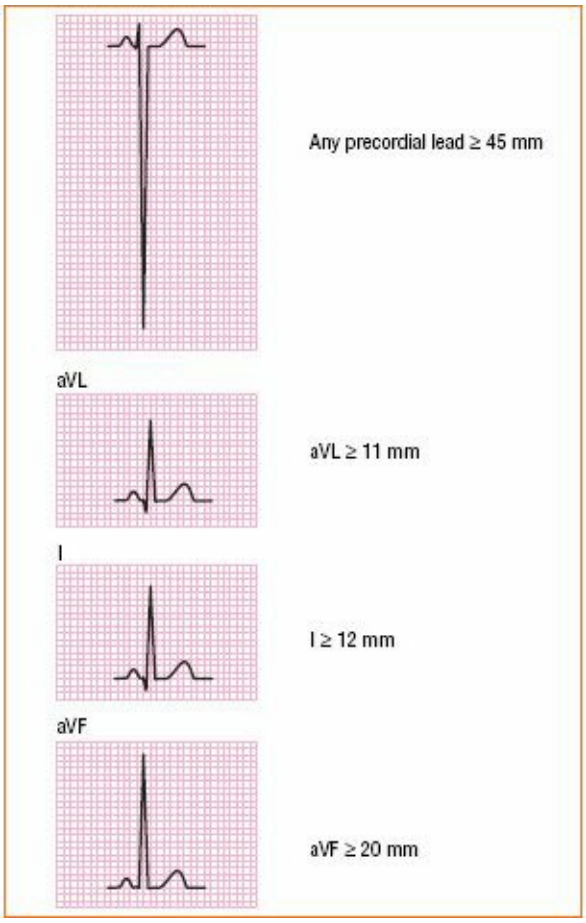


Figure 13-44 Examples of criteria for left ventricular hypertrophy.

Figure 13-44 demonstrates several criteria for the diagnosis of left ventricular hypertrophy.

The ECG in **Figure 13-45** also demonstrates several criteria for the diagnosis of LVH. The S wave is largest in V_1, which is added to the largest R wave found in V_6; the sum is 45 mm or more. Second, the R wave in aVL is well beyond the criterion of being 11 mm or more. In lead I, the R wave is clearly 12 mm or more. More than one of the criteria typically exist on the same ECG, and as many as possible should be found.

Right Ventricular Hypertrophy

The right ventricle can also become hypertrophied. This is usually caused by right ventricular overload because of a pulmonary disease such as pulmonary hypertension. The ECG is quite different in RVH than in LVH because of the electrical vectors being drawn in the anterior and rightward direction. Therefore, a right shift in axis in leads I and aVF and in precordial leads V_1 and V_2 for increased R wave deflection would be expected **Figure 13-46**.

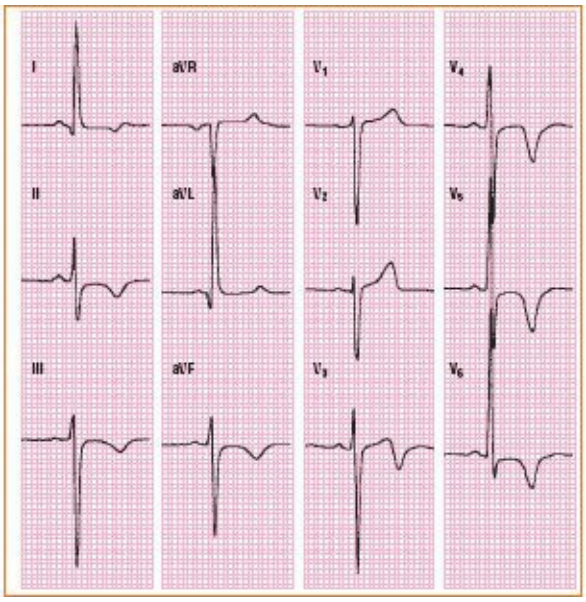


Figure 13-45 A 12-lead electrocardiogram showing several criteria for left ventricular hypertrophy.

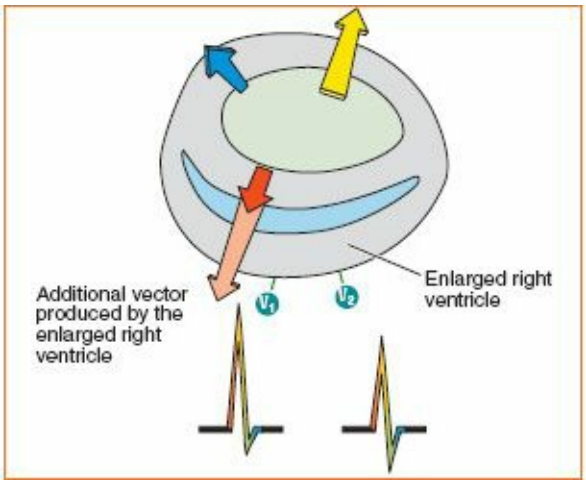


Figure 13-46 Right ventricular hypertrophy.

In RVH, the first finding is often a large R wave in lead V_1 or V_2. This should stand out because during normal conduction, there would be a larger S wave. Owing to the large R waves seen in V_1 or V_2, the normal R-wave progression pattern is altered. The shift of electrical vectors may also cause an S wave in V_6 that is larger than the R wave. The other criterion for RVH is the RAD that lies between 100° and 180°.

Strain Pattern

When severe ventricular hypertrophy occurs, the myocardium can become so thick that the coronary blood supply may be reduced. As a result, the subendocardium, or innermost layer of the heart, is most likely to become ischemic. When hypertrophy is uncomplicated by ischemia, the changes seen in QRS complexes are used to diagnose ischemia. When subendocardial ischemia is present, changes to the ST segment and T wave also appear as a **strain pattern**.

In LVH, the strain pattern in the right precordial leads, V_1 to V_3, is seen as ST-segment elevation with a concave appearance of the upward slope. The T wave is upright and asymmetric in these leads. The precordial leads V_4 to V_6 show a downward-sloping ST-segment depression with an inverted asymmetric T wave. The key to identifying the strain pattern is that it is greatest in the lead with the tallest

and deepest QRS pattern.

The ECG in **Figure 13-47** shows LVH with strain. First, the criteria are met to make the diagnosis of LVH by adding the S wave in V_2 to the R wave in V_5, and the sum is 35 mm or more. The strain pattern is demonstrated by the concave up-sloping of the ST segments in the right precordial leads V_1 and V_2 with asymmetric upright T waves. The right precordial leads V_5 and V_6 show the strain pattern as down-sloping ST segments with asymmetric inverted T waves.

Strain patterns also occur in RVH. The mean electrical axis is anterior and to the right, causing an RAD. This situation causes an increased R wave in the right precordial leads V_1 and V_2. The associated strain pattern shows a downward-sloping depressed ST segment and inverted asymmetric T waves. If the T wave is biphasic instead of inverted, the leading half will usually be negatively deflected and the trailing half will be positive in RVH.

It is important to distinguish between the strain pattern and ST-segment and T-wave changes seen in ischemia and infarction. The strain pattern is problematic, but the ST and T-wave changes can be immediately life threatening. The key is to look closely at the shapes of the patterns. The ST-segment elevations or depressions in ischemia are usually flat and not sloping. The T waves in ischemia are usually symmetric and not asymmetric. Another key used to define the two entities is the J point. In ischemia or infarction, the J point is more sharp or clearly defined. The point at which the T wave takes off from the ST segment is seen. In LVH with strain, the J point is usually more diffuse and slopes up or down depending on the lead being viewed. The clinical correlation between ECG findings and other patient information is most important.

Preexcitation Syndromes

The preexcitation syndrome that is of most interest in the critical care setting is **Wolff-Parkinson-White (WPW) syndrome**. The many situations that occur as a complication to this syndrome should be studied in depth in a cardiology or 12-lead ECG textbook. This section briefly reviews WPW syndrome.

WPW syndrome occurs when there is an accessory pathway for electrical current to bypass the AV node to enter the ventricles. It occurs in less than 3% of the population. This pathway is known as the bundle of Kent. In people with WPW syndrome, the accessory pathway does not have the same ability to slowly conduct electrical impulses as it does through the AV node, which puts the person at risk for extremely fast heart rates that cause hemodynamic instability. The accessory pathway can occur on the right side, connecting the right atrium to the right ventricle, or on the left side, connecting the left atrium to the left ventricle.

There are two basic events when premature ventricular depolarization occurs through the accessory pathway. First, the PR interval is shortened to less than 120 milliseconds. Second, the QRS complex is widened to more than 100 milliseconds. The widening is not the result of a delay in ventricular activation but rather the result of premature activation. Typically in WPW syndrome, most of the ventricular depolarization is activated through the normal conduction pathways. The electrical impulse that passes unobstructed through the bundle of Kent causes early depolarization of a section of the ventricle, resulting in a QRS complex that has a characteristic slurred upstroke called a **delta wave** **Figure 13-48**. The delta wave is not seen in all leads, so all leads should be checked.

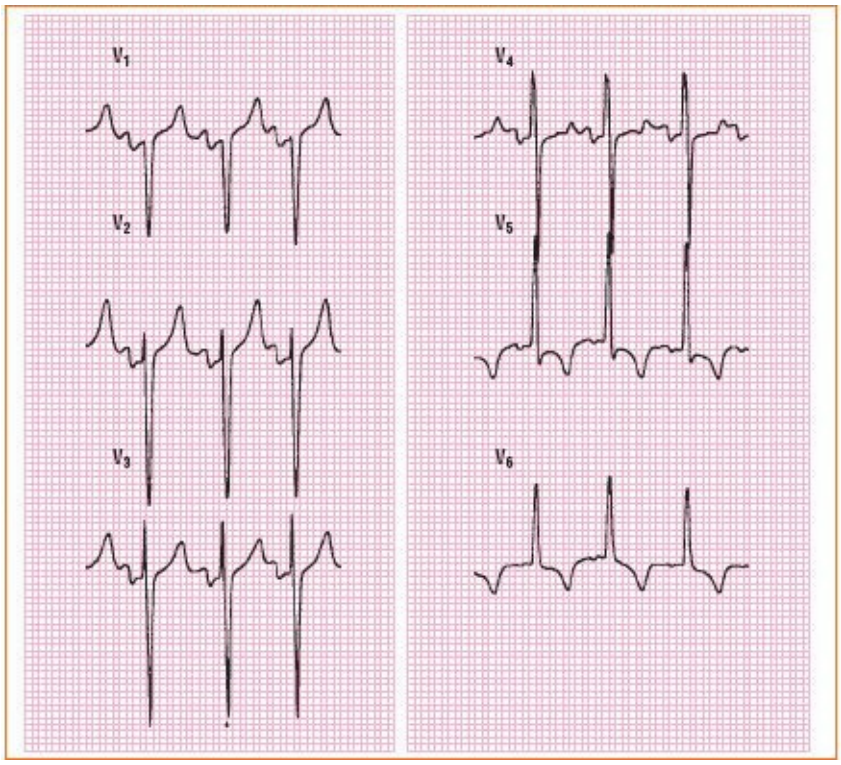


Figure 13-47 Precordial leads showing left ventricular hypertrophy with strain.

In many people with WPW syndrome, the preexcitation causes few clinical problems. It is well documented, however, that this syndrome can predispose a person to dangerous tachyarrhythmias. The two tachyarrhythmias that occur most often in WPW syndrome are **paroxysmal supraventricular tachycardia** (a supraventricular tachycardia [SVT] that starts and ends abruptly) and atrial fibrillation.

In the critical care transport setting, CCTPs should be aware of the potential for wide complex tachycardia developing that is difficult to distinguish from VT.

In WPW syndrome, when tachycardia is present, the electrical impulses can travel down the normal conduction pathway, through the AV node, and back up the accessory pathway. This condition is called orthodromic and usually results in a narrow complex tachycardia. The abnormal conduction can progress the other way as well, in which the electrical impulse travels through the accessory pathway and back up through the AV node. This condition is called antidromic and causes a wide complex tachycardia. The antidromic tachycardias can be very fast if caused by atrial fibrillation or flutter in which transmission is one to one.

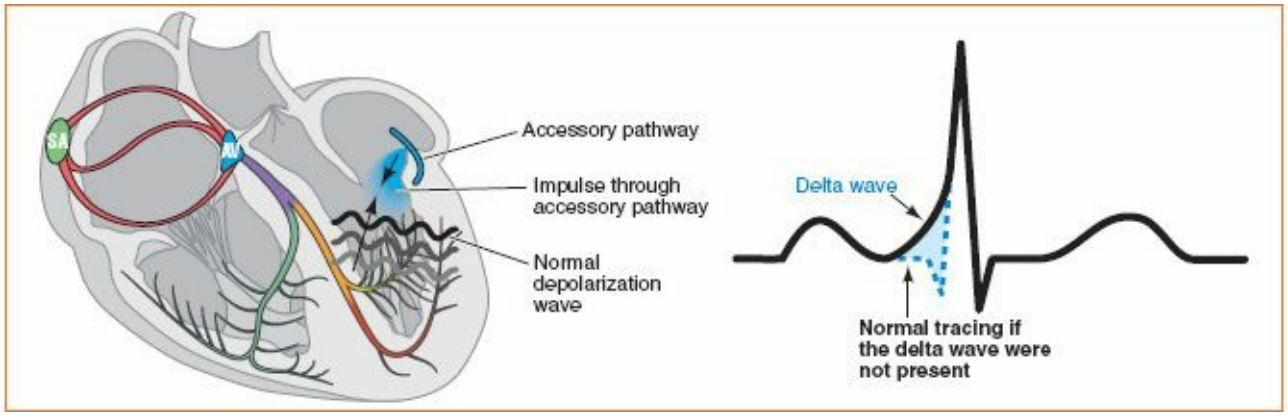


Figure 13-48 Impulse propagation through an accessory pathway and formation of the delta wave.

Transport Management

Wolff-Parkinson-White Syndrome

- Provide electrical cardioversion if the patient's condition is unstable.
- Administer procainamide, but only under the direction of a cardiologist and if the patient is in stable condition.

Treatment of WPW tachyarrhythmias may require electrical cardioversion if they are unstable. Electrical cardioversion is considered the safest treatment. Management with drug therapy produces unpredictable results. Previously, patients with WPW syndrome and atrial fibrillation with rapid ventricular response may have been treated with amiodarone (Cordarone) to stabilize the heart rate, but this treatment has fallen in disfavor because of the possibility of degrading to ventricular fibrillation.

Under the direction of a cardiologist and if the patient is in stable condition, procainamide (Procanbid, Pronestyl, Pronestyl-SR, Procan SR) may also be administered while watching for hypotension to develop and watching for widening of the QRS. Procainamide blocks the accessory pathway but increases conduction through the AV node. Because of the risk of hypotension with rapid IV administration, slow IV infusion is required. Procainamide given slowly has a prolonged onset of action and may not reach therapeutic levels for 40 to 60 minutes. Procainamide may control the atrial fibrillation rate through the accessory pathways, but because of the increased conduction through the AV node, it can also create a dangerous conventional atrial fibrillation. Careful consideration of all variables is necessary to make the right therapeutic decision. In any patient with WPW syndrome and atrial fibrillation, particularly if hemodynamic instability is occurring, cardioversion is the treatment of choice.

The use of adenosine (Adenocard) and other AV node blockers (diltiazem [Cardizem, Dilacor, Tiazac], verapamil [Calan, Covera, Isoptin, Verelan], other calcium channel blockers, and beta-blockers) in atrial fibrillation and atrial flutter should be avoided. Patients with a rapid heart rate and narrow complex tachycardias may also undergo cardioversion.

The definitive treatment for WPW syndrome is ablation of the accessory pathway.

Controversies

Although amiodarone is often thought to be safe in atrial fibrillation with WPW syndrome, it is not. In a case series of twelve patients who received amiodarone, seven experienced ventricular fibrillation. The safest treatment of WPW tachyarrhythmias is cardioversion.

Pericarditis

Pericarditis is an inflammation of the pericardium, the membrane that surrounds the heart. It can be caused by a number of disorders and conditions, such as infection, MI, autoimmune disorders, chest trauma, cancer, drugs, and other causes.

Pericarditis may cause ST-segment elevation and T-wave flattening or even inversion. These ECG findings are consistent with evolving MI, and the signs and symptoms can also mimic an MI. There are features on the ECG that can be helpful to distinguish the two. First, the ST-segment and T-wave changes in pericarditis tend to be throughout all leads of the ECG. The same changes seen with ischemia, injury, and infarction tend to occur only in the leads that are viewing the affected area of the heart. In

pericarditis, the T wave usually does not invert until the ST segment has returned to baseline. In infarction, inversion of the T wave occurs after ST-segment elevation. Finally, Q waves do not form in pericarditis.

Another finding on an ECG that is consistent with pericarditis is the appearance of low voltage in all leads because of pericardial effusion that often accompanies pericarditis. ST and T-wave changes can usually still be seen in this situation. Fluid in the pericardial sac can cause a condition called **electrical alternans** because the heart is able to move or rotate. The electrical axis can vary from beat to beat and is most easily recognized on the ECG by the varying amplitude of each complex.

Long QT Syndrome

Long QT syndrome is a congenital disorder that can be worsened by a variety of factors, including physical exertion, female sex, electrolyte disturbances (hypokalemia and hypomagnesemia), hypothermia, abnormal thyroid function, structural heart disease, bradycardia, medications, and drug overdose **Table 13-6**.

Long QT syndrome is characterized by the prolonged QT interval as seen on the ECG; under certain conditions, it has a tendency to deteriorate into ventricular tachyarrhythmias, including **torsade de pointes** (a very rapid rhythm related to VT), which can lead to syncope or sudden cardiac death. The rhythm is too fast to maintain effective CO, so the patient loses consciousness, often without warning.

TABLE 13-6 Drug Classes That May Worsen Long QT Syndrome

Antianginal
Antiarrhythmic
Antibiotics
Anticancer
Antihistamine
Anti-infectives
Antimalarial
Antinausea
Antipsychotic
Gastrointestinal stimulants
Opiate agonists
Sedatives

It is the deterioration of long QT syndrome that poses a management challenge for CCTPs. Carefully evaluating the ECG, reviewing the patient's medications, and obtaining a medical history will help provide clues to the diagnosis and will alert about the possibility of a tachyarrhythmia developing during transport. For example, CCTPs who will be transporting a cardiac patient who has recently started taking a n **antiarrhythmic** known to increase the QT interval (such as amiodarone [Cordarone]) should be prepared to treat any adverse effects.

As mentioned, long QT syndrome occurs primarily as a congenital defect but can be caused by a

variety of conditions, medications, and drug overdoses. The measurement of the QT interval depends on the heart rate (the faster the heart rate, the shorter the QT interval; the slower the heart rate, the longer the QT interval) and, therefore, must be corrected to make this adjustment. The corrected QT interval, or QTc, as printed on the 12-lead ECG, has already been corrected for heart rate by the cardiac monitor's diagnostic software.

The QT interval is measured from the start of the Q wave to the end of the T wave. Normal values for the QTc are between 300 and 440 milliseconds for men and between 300 and 450 milliseconds for women. During transport, if any patient's QTc reaches 500 milliseconds and the patient's rhythm has not deteriorated into a ventricular tachyarrhythmia such as torsade de pointes, preventive medical intervention may be indicated. This intervention includes correcting electrolyte imbalance, the use of beta-blockers (such as propranolol [Inderal]), and being prepared to treat a developing ventricular tachyarrhythmia.

Typically, long QT syndrome is asymptomatic until the patient becomes physically active or experiences emotional stress (eg, during critical care transport). Symptoms usually begin in the preteen to teenage years but can be present from right after birth to middle age. The initial clinical sign of sudden loss of consciousness is often misdiagnosed as a vasovagal event or seizure. To make the correct diagnosis, a health care provider should give close attention to a history of loss of consciousness during physical activity.

Long-term treatment for this condition is with beta-blockers. They should be started for women who have QTc-interval prolongation of greater than 460 milliseconds and for men who have a QTc-interval prolongation of greater than 440 milliseconds. Patients who cannot tolerate or do not respond well to the medication may need a pacemaker, an ICD (discussed later in this chapter), or a surgical procedure called a cervicothoracic stellectomy.

In the critical care transport setting, CCTPs should watch for the prolonged QTc and the tachyarrhythmias that may develop as a result. Torsade de pointes can occur in short bursts of 15 seconds or less or as a longer hemodynamically unstable rhythm. The treatment for torsade de pointes includes correcting the underlying cause, overdrive pacing, and the IV administration of magnesium sulfate in a dose of 1 to 2 g. Other therapies for patients in unstable condition may include electrical cardioversion and defibrillation. Basic and advanced life support measures should be used as directed by local protocol and national standards. This example is for illustration only, and all treatment should be under direct supervision of a physician or within standing protocols approved by the agency or jurisdiction.

Transport Management

Prolonged QT Syndrome

Preventive Measures

- Correct the electrolyte imbalance.
- Administer a beta-blocker (such as propranolol [Inderal]).
- Be prepared to treat a developing ventricular tachyarrhythmia.

Torsade de Pointes

- Consult with a physician or standing protocols.
- Apply BLS and ALS measures.
- Correct the underlying causes (such as hypomagnesemia and drug overdose).
- Consider initiating overdrive pacing.
- Consider administering magnesium sulfate.

- Consider initiating electrical cardioversion or defibrillation for patients in unstable condition.

Ventricular and Wide Complex Tachycardia

Wide complex tachycardia in general refers to a cardiac rhythm of more than 100 beats/min with a QRS duration of 120 milliseconds or more. This rhythm can present a diagnostic dilemma for health care providers because it can be of ventricular or supraventricular origin. Identifying the origin of the arrhythmia can lead to the correct diagnosis and proper therapeutic interventions.

The differential diagnosis for wide complex tachycardia includes the following major categories:

- VT (most common)
- SVT with aberrance
- Preexcited tachycardias
- Electrographic artifact
- Ventricular paced rhythms

Evaluation of the electrocardiogram is the cornerstone for making the correct diagnosis of wide complex tachycardia. A variety of different diagnostic criteria have been suggested to distinguish the various origins of wide complex tachycardia, and each has its own sensitivity and specificity. This section discusses some of the more common methods of making the diagnosis.

Differential Diagnosis

Wide Complex Tachycardia

- VT
- SVT with aberrance
- Preexcited tachycardias
- Electrographic artifact
- Ventricular paced rhythms

Transport Management

Wide Complex Tachycardia

Patients in a Hemodynamically Unstable Condition

- Perform synchronized cardioversion.
- Give an antiarrhythmic drug to maintain a stable rhythm.

Patients in a Hemodynamically Stable Condition

- Review the history, physical examination, and ECG findings.
- Give amiodarone (Cordarone; first choice) or procainamide (Procanbid, Pronestyl, Pronestyl-SR, Procan SR).
- Give lidocaine (Xylocaine), in case of VT of ischemic origin.
- Give adenosine (Adenocard), if SVT is suspected.
- Give an antiarrhythmic drug to maintain a stable rhythm.

New-Onset Wide Complex Tachycardia (During Transport)

- Provide basic life support (the ABCs).
- Initiate ACLS measures.
- Initiate immediate defibrillation.
- Secure the airway.
- Provide CPR.
- Perform endotracheal intubation.
- Support ventilations with a bag-mask device with supplemental oxygenation.
- Continue CPR as indicated.
- Administer epinephrine or a one-time dose of vasopressin.
- Circulate the medication by performing CPR and repeated defibrillations.
- Give amiodarone if ventricular fibrillation or pulseless VT persists.
- Alternatively, give lidocaine, magnesium sulfate, or procainamide.

The most useful ECG criterion for establishing the diagnosis of VT is the presence of AV dissociation with more ventricular than atrial events. This finding practically rules out an SVT origin. An AV dissociation can be implied during wide complex tachycardia with the presence of fusion beats (simultaneous activation of the ventricular myocardium through normal conduction and from an ectopic ventricular focus). A capture beat or Dressler beat that is seen in VT is a narrow QRS complex that results from an atrial electrical impulse (p wave) that captures the ventricular myocardium through the normal conduction system. A narrow complex beat can also be seen during wide complex tachycardia in a patient with SVT and BBB when a premature ventricular beat originates from or close to the nonconducting bundle branch and fuses with the impulse traveling down the contralateral bundle. This results in a simultaneous ventricular depolarization on both sides of the septum and is demonstrated by a narrow QRS complex.

The QRS duration can also be used to help distinguish between VT and SVT. Studies have shown that nearly 70% of VTs have a QRS duration greater than 140 milliseconds; SVTs typically never have a QRS duration greater than 140 milliseconds. To further expand this criterion, in a patient with a right BBB with a QRS duration of greater than 140 milliseconds, VT is the likely diagnosis. In a left BBB, a QRS duration of greater than 160 milliseconds indicates VT.

The electrical axis seen during wide complex tachycardia is also useful to determine the origin of the arrhythmia. If the axis is 180° to –90° (extreme RAD), the wide complex tachycardia is not likely to be an SVT because this pattern is inconsistent with any type of typical bundle branch or fascicular block. If there is electrical concordance across the precordium (all QRS complexes pointing in the same direction in ECG leads V_1 through V_6), the tachycardia is more likely a VT and is rarely consistent with an SVT.

The morphologic features of the QRS complex provide another way to help identify the different origins of wide complex tachycardia. The usefulness in distinguishing VT from SVT in a wide complex tachycardia is limited, however, because some measurements are required, making the method less accurate and more time consuming in a moving transport vehicle. The following discussion of QRS morphologic features in wide complex tachycardia is provided to help CCTPs better understand the various ECG findings with wide complex tachycardia from different origins. In a patient with a right BBB, the precordial lead V_1 should be examined. If the left R wave is taller than the R′ or if the QRS is biphasic with an Rs or qR pattern, the origin of wide complex tachycardia is likely VT. In a patient with a left BBB, again, precordial lead V_1 should be examined. If the duration of the initial r wave is greater than 30 milliseconds, the time from the beginning of the QRS to the nadir of the S wave is greater than 70

milliseconds, and the down stroke of the S wave is notched, the origin is likely to be VT. If the QRS complex in precordial lead V_6 has a monophasic QS or biphasic rS with an r to S ratio of less than 1 during a right BBB wide complex tachycardia, VT is likely. If the intrinsicoid deflection (beginning of the QRS complex to the beginning of the down slope of the R wave) in the precordial lead V_6 is greater than 80 milliseconds, VT is the likely origin of the wide complex tachycardia.

Management

The initial management of patients in wide complex tachycardia primarily depends on their hemodynamic status. Patients who have low blood pressure, pulmonary edema, severe chest pain, or other evidence of poor perfusion associated with the wide complex tachycardia should be promptly treated using synchronized cardioversion.

When a patient with wide complex tachycardia remains in hemodynamically stable condition, more time can be spent reviewing information from the history, physical examination, and ECG findings. As mentioned, the majority of cases of wide complex tachycardia are VT. Making the correct diagnosis, however, is the safest approach to management.

With a patient with wide complex tachycardia in whom the cause is uncertain and the patient's condition is hemodynamically stable, amiodarone (class III [Cordarone]) is the drug of choice because it can terminate SVT and VT. It is also useful in patients with a poor ejection fraction (the percentage of blood ejected out of the left ventricle during each contraction) because of its favorable hemodynamic profile.

Procainamide (class IA [Procanbid, Pronestyl, Pronestyl-SR, Procan SR]) can also be used with caution in patients with wide complex tachycardia in hemodynamically stable condition. There are several disadvantages to using procainamide. It can cause a sudden drop in blood pressure with rapid IV administration and is contraindicated in patients with complete heart block during wide complex tachycardia because it suppresses the nodal or ventricular pacemakers and can result in asystole. As with all drugs, its indications and contraindications should be fully understood before administering it to any patient.

Lidocaine (Xylocaine; class IB) is useful for VT of ischemic origin, but it does not terminate SVT. In a patient who is conscious and in hemodynamically stable condition, in higher doses, it can cause confusion.

If SVT is suspected, adenosine (Adenocard) is a useful and diagnostic medication. It works by blocking conduction through the AV node, which is an essential part of the tachycardia circuit in most SVTs. It can sometimes terminate atrial tachycardias and, rarely, VT. Because adenosine has a very short half-life, it rarely causes complications in patients with wide complex tachycardia. An exception is a patient with an irregular wide complex tachycardia as a result of atrial fibrillation and preexcitation over an accessory pathway. In this case, adenosine can block conduction through the AV node, allowing only conduction through the accessory pathway, and may lead to ventricular fibrillation.

Following termination of wide complex tachycardia, preventive treatment for maintaining a stable rhythm should be initiated. This treatment will help prevent recurrent episodes, minimize the patient's symptoms, and help protect the patient from sudden cardiac death. For CCTPs, intervention is generally limited to pharmacologic treatment with antiarrhythmic drugs, usually a continuation of the drug that terminated the wide complex tachycardia in the first place. Additional in-hospital treatments may include the use of an ICD and catheter ablation procedures.

Patients with or whose rhythm converts to wide complex tachycardia rhythm can pose a diagnostic dilemma, especially for a transport team in transit. At times, only minimal information about the patient's history and clinical and laboratory findings is known. If wide complex tachycardia develops during transport, a rapid evaluation must be performed and the appropriate treatment must be initiated in a

confined environment with relatively limited resources. BLS is always the first step in patient management, closely followed by ACLS practices. If the patient is in a hemodynamically unstable condition, wide complex tachycardia should be managed as if it were VT until proven otherwise, and synchronized cardioversion would be the initial therapy of choice.

Polymorphic or pulseless VT should be treated as ventricular fibrillation. If this is a witnessed condition that occurs during transport, the patient should already be connected to a cardiac monitor and have a patent IV line. The patient may also already have a secured airway. Immediate defibrillation is indicated because it is a witnessed arrest. The treatment should be in accordance with current standard American Heart Association/BLS/ACLS guidelines and include the following steps: Defibrillate at 200, 200 to 300, and then 360 J; if using a biphasic defibrillator, use three consecutive shocks at 150, 150, and then 200 J. Ensure that the airway is secured, and provide CPR as indicated. Perform endotracheal intubation if the tube is not already in place, and support ventilations with a bag-mask device with supplemental oxygenation. Continue CPR as indicated. Because the patient should already have an IV line in place, epinephrine may be initially administered, 1.0 mg IV, and repeated at 3- to 5-minute intervals as long as the arrhythmia persists. (Peripheral IV lines in a transport setting can be particularly vulnerable to becoming dislodged. If this occurs, until another line can be established, the following medications can be administered through a patent endotracheal airway: epinephrine, atropine, and lidocaine [Xylocaine].) An alternative one-time dose of vasopressin, 40 U IV, can be given in place of the initial epinephrine. After each drug is administered, circulate the medication by performing CPR and repeating defibrillations at 360 J (200 J biphasic). If ventricular fibrillation or pulseless VT persists, amiodarone (Cordarone), 300 mg IV, is indicated. This may be repeated with a second dose of 150 mg in 5 to 10 minutes. Alternatively, lidocaine (Xylocaine) in a 1.0 to 1.5 mg/kg bolus may be given and repeated every 5 minutes up to a maximum dose of 3.0 mg/kg. Magnesium sulfate, 1.0 to 2.0 mg, can be considered for its antiarrhythmic effects. Procainamide (Procanbid, Pronestyl, Pronestyl-SR, Procan SR) is also used to treat this rhythm and is administered at 20 to 50 mg/min to a total dose of 17 mg/kg. When administering an antiarrhythmic medication, it is recommended that the maximum dose be reached before moving to the next drug of choice. Continued resuscitative efforts should be directed by a physician on the transport team or in direct radio communication and consultation with a physician with telemetry capabilities.

Electrolyte and Drug Effects

Electrolytes have an essential role in the proper functioning of cells and are found in the intracellular and extracellular fluids. Important electrolytes are sodium, potassium, calcium, and magnesium. It is the exchange of electrolytes into and out of cells that generates electrical energy for cellular depolarization and repolarization. As the cells depolarize electrically, they have a mechanical response in the form of contraction, and when they repolarize, they relax and expand back to their original state. In the cardiac muscle, this process is repeated continuously for a lifetime. Proper levels of these electrolytes are essential to keep the cells functioning correctly. When changes in electrolytes occur, their effects are often seen on the ECG.

Certain drugs have an effect on the electrolyte channels of the cell membranes, changing how the electrolytes flow into and out of the cells. These drugs can alter conduction patterns that have an effect of how the ECG is represented.

This section discusses ECG effects of two of the most clinically important electrolytes, potassium and calcium, because they produce the most recognizable changes on the ECG.

Hyperkalemia

Potassium is the primary intracellular ion (the other is phosphate). An elevation of the potassium level is called **hyperkalemia**. Hyperkalemia is the most dangerous of all electrolyte abnormalities. This condition

can cause death and prevents some drugs used in resuscitation efforts from being effective. On the ECG, hyperkalemia can cause changes in the appearance of all waveforms, representing a change that has occurred within the cell, and it can cause virtually any arrhythmia. Rapid recognition and correction of this electrolyte disturbance is essential to reversing any harmful effects. The main ECG changes found in hyperkalemia are the following:

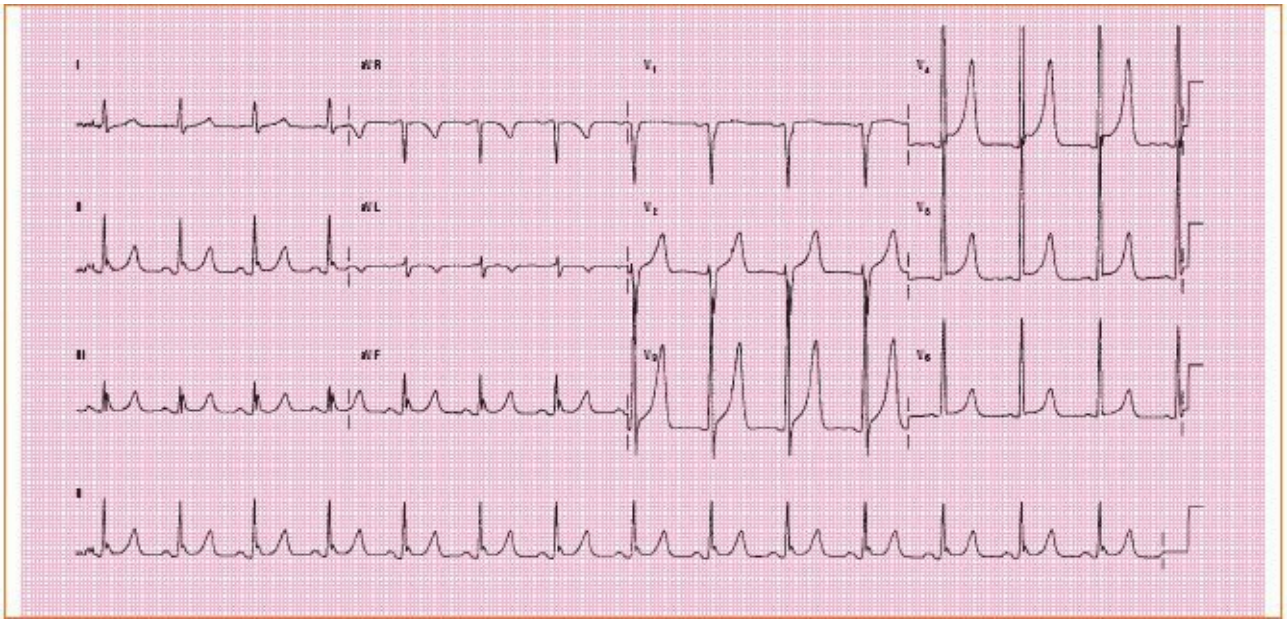


Figure 13-49 A 12-lead electrocardiogram showing T-wave changes with hyperkalemia.

1. T-wave abnormalities (tall and peaked)
2. Intraventricular conduction delays
3. P-wave abnormalities (missing or decreased amplitude)
4. ST-segment changes simulating an injury pattern
5. Cardiac arrhythmias (predominantly bradycardias)
6. Sinusoidal ECG pattern

On the ECG, T-wave abnormalities are the first to appear with hyperkalemia. These changes start to appear when the potassium level exceeds 5.5 mEq/L. The most common changes are tall, narrow, peaked T waves, but these changes are not seen in every patient with hyperkalemia. A prolonged QT interval may be present. As the potassium level increases, the height of the T wave decreases and widens; the PR, QRS, and QT intervals also widen; and the amplitude decreases.

The ECG in **Figure 13-49** represents hyperkalemia. There are tall, narrow, peaked T waves in V_2 through V_4. Tall T waves also appear in the inferior leads II, III, and aVF. When the T wave is two thirds the height of the R wave, it is considered pathologic.

A patient with known hyperkalemia or renal failure with suspected hyperkalemia should have supportive treatment with IV access and cardiac monitoring. In the presence of hypotension or marked QRS widening, three choices for medication administration include IV bicarbonate, calcium, or insulin given with 50% dextrose; any one of these or a combination of these may be given. Calcium should not be given if digoxin toxicity is suspected. Magnesium sulfate (2 g over 5 minutes) may be used alternatively with digoxin toxicity–related cardiac arrhythmias. Additional medical treatments include the use of calcium carbonate or gluconate, sodium bicarbonate, albuterol (Proventil, Ventolin, Volmax), furosemide (Lasix), and binding resins such as sodium polystyrene sulfonate (Kayexalate).

Transport Management

Hyperkalemia

- Provide supportive treatment with IV access and cardiac monitoring.
- In the presence of hypotension or marked QRS widening, give IV bicarbonate, calcium, and insulin together with 50% dextrose.
- Give magnesium sulfate for digoxin toxicity–related cardiac arrhythmias.
- Give additional medical treatments—for example, calcium carbonate or gluconate, sodium bicarbonate, albuterol (Proventil, Ventolin, Volmax), furosemide (Lasix), and binding resins such as sodium polystyrene sulfonate (Kayexalate).

Hypokalemia

A decrease in the level of potassium is called **hypokalemia**. Hypokalemia does not cause the dramatic changes on the ECG as at the other end of the spectrum with hyperkalemia. There are some mild, nonspecific changes, such as ST-segment depression, slightly decreased amplitude of the T waves, and minimal prolongation of the QRS interval. Probably the most common abnormality in hypokalemia is the presence of a prominent U wave. The U wave (shown in Figure 13-8) is usually small and follows the T wave. Hypokalemia does not typically cause arrhythmias by itself.

Differential diagnoses for U waves include the following:

1. Hypokalemia
2. Bradycardia
3. LVH
4. Central nervous system events
5. Drug use: digoxin, class I antiarrhythmics, phenothiazines

Treatment for hypokalemia is aimed at decreasing potassium losses, replenishing potassium stores, evaluating for potential toxic effects, and determining the cause to prevent additional losses.

In the transport setting, a few things can be done to decrease potassium losses. First, if possible, the use of diuretics and laxatives should be discontinued. If diuretics are required for severe heart failure, potassium-sparing diuretics should be used, diarrhea and vomiting should be treated, and H_2 blockers should be used to decrease loss from nasogastric suction.

Replacing lost potassium is done by measuring the loss and estimating the replacement. Intravenous potassium is not well tolerated because it can be highly irritating to veins and can be given only in relatively small doses (ie, 10 mEq/h). With close medical supervision, in an emergency situation, as much as 40 mEq/h can be administered through a central line.

In evaluating for the potential toxic effects of hypokalemia, monitoring for cardiac arrhythmias and prompt treatment are essential. This is one condition in which a more aggressive approach would be taken to IV potassium replacement.

Finally, intervention to prevent future losses of potassium is not a major component of interfacility care. By preventing further losses and treating significant hypokalemia, a first step in prevention has been accomplished.

Differential Diagnosis

Hypokalemia

- Bradycardia
- LVH
- Central nervous system events
- Drug use: digoxin, class I antiarrhythmics, phenothiazines

Transport Management

Hypokalemia

- Discontinue diuretics and laxatives.
- Use potassium-sparing diuretics if required for severe heart failure.
- Treat diarrhea and vomiting.
- Use H_2 blockers to decrease nasogastric suction losses.
- Measure the potassium loss, estimate the replacement, and administer through a central line.
- Monitor for cardiac arrhythmias, and treat them promptly if they occur.

Hypercalcemia

Hypercalcemia is a disorder that is most commonly caused by malignancy or primary hyperparathyroidism. Other causes of an elevated calcium level are less common and not typically considered until the most common causes are ruled out. ECG changes are minimal, with the most significant change being a shortening of the ST segment, which, in turn, shortens the QT interval. The PR interval may be prolonged; at higher levels, the QRS may lengthen and T waves may become flat or even invert. Because hypercalcemia affects conduction times, a variable degree of heart block may develop. Arrhythmias seldom occur as a result of hypercalcemia.

Treatment of hypercalcemia includes supportive management of the ABCs and increased hydration, often with the inclusion of a loop diuretic (such as furosemide [Lasix]) to increase calcium excretion and prevent overload from hydration therapy alone.

Transport Management

Hypercalcemia

- Manage the ABCs.
- Provide hydration.
- Administer a loop diuretic.

Hypocalcemia

Hypocalcemia that occurs acutely can be the result of medication or surgical effects. It causes the opposite effects of hypercalcemia. ECG changes seen include a prolongation of the ST segment that produces an apparent lengthening of the QTc interval. The cardiopulmonary effects of hypocalcemia may include wheezing, stridor, bradycardia, and pulmonary crackles (rales), and an S_3 may be heard. The

prolonged QT interval can lead to ventricular arrhythmias (such as torsade de pointes).

Signs and Symptoms

Hypocalcemia

- ECG findings: prolonged ST segment and apparent lengthening of the QTc interval
- Wheezing
- Stridor
- Bradycardia
- Pulmonary crackles (rales)
- S_3 heart sound
- Ventricular arrhythmias (such as torsade de pointes)

Transport Management

Hypocalcemia

- Initiate IV access.
- Administer oxygen.
- Monitor vital signs and the ECG.
- Provide an infusion of calcium gluconate.
- Consider administration of magnesium to help prevent torsade de pointes.

Treatment during interfacility transfer mainly includes supportive measures, including IV fluids, oxygen, and monitoring of vital signs and the ECG. An infusion with calcium gluconate can be given over a period of 5 to 10 minutes. Magnesium may be considered when there is a prolonged QTc interval to help prevent torsade de pointes.

Conditions That Mimic Myocardial infarction

Multiple conditions may mimic the clinical and ECG findings that occur with ischemia, injury, and infarction. The following is a synopsis of these conditions:

- Pericarditis: ST-segment elevation, T-wave inversion (possible)
- Left BBB: ST-segment elevation, QS complexes
- LVH: ST-segment elevation
- Ventricular rhythms: ST-segment elevation, QS complexes
- Early repolarization: ST-segment elevation, tall T waves

Drug Effects on the ECG

There are countless medications on the market that are specifically designed to alter the function of the heart. The most notorious of these is digoxin (Lanoxicaps, Lanoxin), a digitalis preparation. According to the American Association of Poison Control Centers, there is a higher incidence of adverse effects with calcium channel blockers and beta-blockers, but the mortality rate is higher with digoxin toxicity.

Understanding the actions of digoxin and treating its adverse effects are essential to decreasing the mortality when toxicity occurs. A more in-depth discussion of digoxin toxicity is provided later in this section.

Cardiac medications can increase the rate and force of contraction or slow and protect the heart from increased demands. Virtually any function of the heart can be altered by design. There are other classes of pharmaceuticals that are not cardiac medications but have expected or unexpected cardiac side effects. It is beyond the scope of any text to address the effects of the countless illicit drugs that are available on the street or even online. The point to this discussion is that there are endless possibilities of drug effects that will alter what is ultimately seen on the ECG. **Table 13-7** provides a brief overview of the cardiac effects from a few of these classes of drugs.

Digoxin Toxicity

Digoxin (Lanoxicaps, Lanoxin) is a cardiac glycoside that produces positive inotropic and negative chronotropic activity in the heart. It is primarily indicated to treat chronic heart failure and to control the ventricular rate in atrial tachyarrhythmias (such as atrial fibrillation). The inotropic effects are a result of inhibiting the sodium-potassium adenosine triphosphatase pump. This action causes a rise in intracellular calcium and sodium and a decrease in intracellular potassium. The movement of these intracellular electrolytes results in an increased force of myocardial muscle contraction, thus causing the positive inotropic effect.

TABLE 13-7 Common Toxic Drug Effects

Drug	Possible Toxic Effects
Class I antiarrhythmics	Lengthened QRS and QTc intervals Possible AV blocks Slowed or completely blocked SA node Arrhythmias
Calcium channel blockers	Blocked AV node primarily, but extent of block varies significantly among different drugs in this class
Beta-blockers	Slowed automaticity of the SA node and the Purkinje system Blocked AV node
Amiodarone (Cordarone)	Slowed conduction everywhere: the SA node, atrium, AV node, Purkinje system, and ventricles
Phenothiazines and tricyclic antidepressants	Widened QRS and QTc interval T-wave abnormalities Arrhythmias common in overdoses

Abbreviations: AV, atrioventricular; SA, sinoatrial.
Source: Garcia T, Holtz N. Figure 16-3. In: *12-Lead ECG: The Art of Interpretation.* Sudbury, MA: Jones and Bartlett Publishers; 2001:507.

Therapeutic concentrations of digoxin (1.0-2.0 ng/mL) cause the desired positive inotropic effect and decreased electrical conduction between the SA and AV nodes. Therapeutic levels of digoxin also decrease automaticity and increase the diastolic resting membrane potential.

The margin between toxic and therapeutic doses is small, and many factors can affect this level. One of the most common causes of digoxin toxicity is drug-to-drug interaction(s). In the critical care transport setting, there may be a patient who has been taking digoxin and experiencing the desired effects, but who then experiences a cardiac, medical, or other disease state and is given another medication (or more likely, multiple medications) that can affect the digoxin level. Any drug interaction or disease state (eg, hypothyroidism or renal impairment) that can interfere with the absorption or elimination of digoxin can cause an increased serum concentration.

The electrolytes potassium and calcium must be evaluated in conjunction with suspected digoxin toxicity. If the patient is given diuretic therapy, hypokalemia can develop and increased automaticity results. Hyperkalemia exacerbates the digoxin-induced conduction delays. Also, hypercalcemia increases ventricular automaticity and can increase the effects of digoxin; therefore, the administration of calcium in a patient with digoxin toxicity can lead to more harmful effects.

At toxic levels, the excessive increase of intracellular calcium elevates the resting potential and predisposes the heart to arrhythmias. Virtually any arrhythmia can occur in digoxin toxicity, and none are specific to this condition. CCTPs need to be alert to any manifestation of increased automaticity in the face of impaired electrical conduction.

The clinical manifestations of acute digoxin intoxication may not appear for several hours. When they do, they typically include gastrointestinal symptoms of nausea, vomiting, or abdominal pain. Neurologic symptoms may include lethargy, confusion, and weakness. Symptoms associated with chronic digoxin intoxication may be difficult to discern from many other medical conditions.

Signs and Symptoms

Digoxin Intoxication

- Nausea
- Vomiting
- Anorexia
- Abdominal pain
- Weight loss
- Delirium
- Confusion
- Dizziness
- Disorientation
- Drowsiness
- Headache
- Hallucinations
- Amblyopia (partial or complete loss of vision in one eye)
- Photophobia (painful sensitivity to light)
- Scotoma (area of diminished vision within the visual field)
- Chromatopsia (visual disturbance in which objects appear abnormally colored)
- Xanthopsia (yellowish visual discoloration)

Transport Management

Digoxin Intoxication

- Manage the ABCs.
- Stabilize the patient's hemodynamics.
- Administer activated charcoal to clear the gastrointestinal tract of those who recently ingested digoxin.
- Administer atropine to treat AV, junctional, or ventricular ectopy; produce first-degree AV block; slow ventricular response to atrial fibrillation; or experience accelerated AV or junctional rhythms.
- Administer digoxin-specific antibodies (Fab fragments).

The most common ECG changes in digoxin toxicity include AV, junctional, or ventricular ectopic beats; first-degree AV block; slow ventricular response in atrial fibrillation; and an accelerated AV or junctional rhythm. More severe arrhythmias may be seen and include severe bradycardia, high-degree heart blocks, and malignant ventricular rhythms. In reviewing this list of potential rhythms, it is clear that there is no signal arrhythmia that points directly to digoxin toxicity. As mentioned, CCTPs should review the patient's medications before transport. In a patient known to be taking digoxin, when an arrhythmia occurs, especially in the presence of other clinical findings, digoxin toxicity should be considered as the underlying cause. In general, CCTPs should be aware that any accelerated rhythm with conduction delays should raise the level of suspicion of digoxin toxicity.

Any patient taking digoxin in whom an altered level of consciousness or gastrointestinal symptoms develop with or without ECG changes needs further evaluation for toxicity. Because CCTPs typically are working with data collected before transport, new electrolyte values and drug assays are usually not available en route to the receiving facility. The one value that can be obtained before transport that has some prognostic value and can provide clues to the diagnosis of digoxin toxicity (in the presence of other clinical findings that develop during transport) is the potassium level. In a patient with digoxin toxicity, a potassium level greater than 5.5 mEq/L (normal serum potassium level, 3.5-5.0 mEq/L) with normal renal function is associated with higher mortality. Toxicity can occur in a patient with normal therapeutic levels if a drug–drug interaction interferes with absorption or elimination of digoxin or in the presence of increased automaticity and impaired conduction.

If a CCTP suspects digoxin toxicity during transport, based on ECG changes along with associated signs and symptoms, the initial concern is to manage the ABCs and stabilize the patient's hemodynamics. Clearing the gastrointestinal tract of recently ingested digoxin can be beneficial; in the field, this can be accomplished with the administration of activated charcoal. This measure not only helps prevent further absorption of the drug, but also may increase its excretion from the body. Atrioventricular, junctional, or ventricular ectopy; first-degree AV block; slow ventricular response to atrial fibrillation; and accelerated AV or junctional rhythms will typically respond to administration of atropine.

In the treatment of severe digoxin toxicity, the standard of care is the administration of digoxin-specific antibodies (Fab fragments or digoxin-immune Fab [Digibind]). Fab fragments neutralize free digoxin, decrease potassium levels, and increase renal excretion.

Laboratory tests for electrolyte levels, glomerular filtration rate, creatinine level, and urea level should be obtained as soon as possible. Further treatment includes the administration of insulin and glucose to treat the associated hyperkalemia and to provide the proposed cardioprotective effects. (Insulin may have a cardioprotective effect in digoxin toxicity.) The use of calcium chloride to stabilize the myocardium is controversial. If Fab fragments are used in the treatment of digoxin toxicity, the use of sodium polystyrene sulfonate (Kayexalate) to treat hyperkalemia is *not* recommended because it will overcorrect the serum potassium level. Fab fragments have also virtually eliminated the need for a

pacemaker or cardioversion in digoxin toxicity. Early recognition and aggressive treatment of digoxin toxicity is lifesaving.

Coronary Artery Disease and Angina

Coronary artery disease (CAD) is the most common form of heart disease and is a leading cause of death in US adults. The coronary arteries supply oxygen and nutrients to the myocardium. If one of these blood vessels becomes blocked, the muscle it supplies will be deprived of oxygen (ischemia). If this oxygen supply is not quickly restored, the ischemic area of heart muscle will eventually die (undergo infarction).

Atherosclerosis is of particular concern because it affects the inner lining of the aorta and the cerebral and coronary blood vessels, leading to the narrowing of these vessels and the reduction of blood flow through them. The atherosclerotic process begins, probably in childhood, when small amounts of fatty material are deposited along the inner wall (intima) of arteries, usually at points of turbulent blood flow (such as where the arteries bifurcate or where the arterial wall has been damaged). As the streak of fat enlarges, it becomes a mass of fatty tissue, an atheroma, which gradually calcifies and hardens into a plaque. The atheromatous plaque infiltrates the arterial wall and decreases its elasticity. At the same time, it narrows the arterial lumen and interferes with blood flow through the lumen. The narrowed, roughened area of the arterial intima provides a locus for the formation of a fixed blood clot, or thrombus, which may then obstruct the artery altogether (when in a coronary artery it is known as coronary thrombosis). In addition, calcium may precipitate from the bloodstream into the arterial walls, causing arteriosclerosis, which greatly reduces the elasticity of the arteries.

Risk factors for atherosclerosis and CAD include hypertension, cigarette smoking, diabetes, high serum cholesterol levels, lack of exercise, obesity, family history of heart disease or stroke, and male sex.

Peripheral Vascular Disorders

Although atherosclerosis is rarely the primary cause of medical emergencies, it is a major contributor to other conditions that may become medical emergencies. For example, arterial bruits (turbulence or "swishing" sounds heard with a stethoscope placed over the carotid arteries) signal the presence of atherosclerosis and contraindicate the use of carotid sinus massage. Atherosclerosis can also contribute to claudication, a severe pain in the calf muscle caused by narrowing of the arteries in this muscle and leading to a painful limp. Finally, atherosclerosis may be associated with phlebitis—inflammation, swelling, and pain along the veins that can lead to the formation of blood clots and thrombophlebitis, which is venous inflammation associated with a thrombus (blood clot). If dislodged, these thrombi become emboli that could travel to the heart and through its right side, lodging in the pulmonary arterial tree and causing a pulmonary embolism.

An estimated 5 to 20 million Americans are affected by significant peripheral vascular disorders annually. The most dangerous complication of these disorders is pulmonary embolism, which causes approximately 200,000 deaths each year. Risk factors for peripheral vascular disorders include age, oral contraceptive use, smoking, recent surgery, recreational IV drug use, trauma, and extended immobilization. The identification of these risk factors has a significant role in diagnosing peripheral vascular occlusions. Signs of peripheral vascular occlusion may include pain, redness, swelling, warmth, and tenderness in the extremity; however, these signs are present in only about half of all cases. The presence of claudication indicates a significant narrowing of the peripheral arteries associated with peripheral vascular disorders. Arterial bruits are another sign of vascular narrowing that can contribute to ischemia and stroke.

Unfortunately, there is not much a critical care transport team can do for peripheral vascular disease. If a blockage or potential embolus is suspected, IV heparin should be administrated initially as a bolus and followed by a drip. If ultrasound or Doppler is available on the transport vehicle, frequent assessment of the affected limb should be done. In addition to anticoagulant therapy, efforts should be

made to maximize blood flow to the extremity by using warm compresses. Keeping the leg in the position of comfort and minimizing movement are also helpful.

Signs and Symptoms

Peripheral Vascular Disorders

- Pain, redness, swelling, warmth, and tenderness in the extremity
- Claudication
- Arterial bruits

Transport Management

Peripheral Vascular Disorders (Suspected Blockage or Embolus)

- Give IV heparin, initially as a bolus and followed by a drip.
- Monitor the affected limb with ultrasound or Doppler, if available.
- Maximize blood flow to the extremity by using warm compresses.
- Keep the affected limb in the position of comfort.
- Minimize movement of the affected limb.
- Apply a cardiac monitor, record a baseline ECG before transport, and perform frequent 12-lead ECG monitoring.

All critical care transport patients should undergo cardiac monitoring, but any patient being transported a long distance with a blood clot that has the potential to become a pulmonary embolus should be given a baseline ECG before transport and must undergo frequent 12-lead ECG monitoring. ST-segment depression in the limb leads and the precordial leads accompanied by an increase in RAD may indicate a pulmonary embolus.

Acute Coronary Syndrome

The American Heart Association and the American College of Cardiology have published guidelines for the management of patients with STEMI. In the guidelines, the term **acute coronary syndrome (ACS)** is used to describe conditions that cause an episode of ischemic discomfort (chest pain) as the result of disruption of plaque within a coronary artery.

Plaques occur as the natural evolution of atherosclerosis. The plaques that are prone to disruption are usually nonobstructive but have a large amount of macrophages and other inflammatory cells associated with them. After the plaques are disrupted from within a coronary artery, these substances promote a chain reaction of platelet activation, adhesion, aggregation, thrombin formation, and, ultimately, thrombus formation. The end product is a complete occlusion of the coronary artery or one of its branches. If there is not enough collateral circulation from branches of adjacent coronary arteries, myocardial necrosis begins within 15 minutes and spreads from the endocardium toward the epicardium.

The term ACS is used to describe any group of clinical symptoms consistent with acute myocardial ischemia. ACS represents a sudden deterioration of the condition of a coronary blood vessel. Acute myocardial ischemia typically presents as chest pain as a result of insufficient blood supply to the heart muscle, which itself is a result of CAD. The life-threatening ACS disorders are responsible for much of

the emergency medical care and hospitalization in the United States.

The broad term acute coronary syndrome includes unstable angina, MI without ST-segment elevation (NSTEMI), and MI with ST-segment elevation (STEMI). Patients with STEMI have a high probability (greater than 90%) of coronary thrombus occlusion. In comparison, patients with stable angina have only about a 1% probability of having a coronary thrombus. Of patients with unstable angina or NSTEMI, 35% to 75% may have a coronary thrombus. These numbers are not good predictors to identify the patients in whom MIs with Q waves eventually develop: Not every STEMI develops Q waves, and Q waves may develop in NSTEMI.

All patients experiencing a possible cardiac event should have a 12-lead ECG performed. Use of the 12-lead ECG enables the responder to categorize two groups of patients who have experienced ACS: patients with ST-segment elevation and patients without ST-segment elevation. Most patients whose ECG displays ST-segment elevation ultimately will have a STEMI. Patients who have ischemic discomfort (chest pain) without an ST-segment elevation are having unstable angina or an NSTEMI that usually leads to a non–Q-wave MI; these conditions are collectively known as UA/NSTEMI (unstable angina/NSTEMI). Patients with non–ST-segment elevation are ultimately diagnosed with unstable angina or not (depending on whether cardiac enzymes or biomarkers indicated evidence of cardiac necrosis). Patients who experience angina may also have ST-segment depression. Finally, some patients experiencing angina or MI may have no changes indicated by the ECG.

Early recognition of the signs and symptoms of a STEMI are essential to initiate treatment or to ensure transporting the patient to a facility where the appropriate care can be provided. Signs and symptoms are discussed later in this chapter in the section on MI and include chest and arm pain, lower jaw pain, shortness of breath, and diaphoresis. The majority of deaths as a result of STEMI occur within the first 1 to 2 hours after the onset of symptoms and are usually caused by ventricular fibrillation. This is why rapid intervention with the most appropriate reperfusion therapy must be started as soon as a diagnosis of STEMI has been confirmed.

A 12-lead ECG should be performed within 10 minutes of contacting a patient with chest discomfort or other signs and symptoms suggestive of STEMI. If this initial ECG is not diagnostic of STEMI but the patient remains symptomatic and there is a high degree of suspicion about STEMI, serial 12-lead ECGs should be performed at 5- to 10-minute intervals while maintaining continuous cardiac (ST-segment) monitoring. Newer 12-lead ECG machines are equipped with multiparameter monitoring capabilities that can assess ST-segment deviation every 30 seconds. With IWMI, right-sided chest leads should be evaluated for ST-segment elevation suggestive of right ventricular infarction (RVI). The 12-lead ECG in the critical care transport setting is central to the therapeutic decision pathway because it provides evidence of ST-segment elevation in patients who will benefit from reperfusion therapy.

In a patient with STEMI, the more leads that show ST-segment elevation on a 12-lead ECG, the higher the mortality rate. In other words, if the occlusion occurred high enough in a coronary artery that has affected a large area of the heart, multiple patterns of ischemia will appear. Other important predictors of mortality are STEMI occurring in conjunction with left BBB and in a predominantly anterior location. The current guidelines for the ECG diagnosis of acute MI require at least 1 mm (0.1 mV) of ST-segment elevation in the limb leads and at least 2 mm of elevation in the precordial leads. These elevations must be present in leads that are physically next to each other or anatomically contiguous. (Each small block measured vertically on the ECG paper equals 1 mm.)

When no ST-segment elevation is present or the ECG is normal or shows nonspecific changes, there is no evidence to suggest any benefit of fibrinolytic therapy; in fact, in such cases, fibrinolytic therapy has been shown to be harmful. If clinical signs and symptoms continue, the 12-lead ECG should be repeated every 5 to 10 minutes and monitored continuously for any ST-segment changes until serologic testing for cardiac enzymes or biomarkers can be done.

All patients with ACS should receive antithrombin and antiplatelet therapy regardless of the presence or absence of ST-segment elevation. Patients with persistent ST-segment elevation are candidates for prompt reperfusion therapy, pharmacologic or catheter based, to restore blood flow in the occluded artery. Patients without ST-segment elevation should receive anti-ischemic therapy and be considered for catheter-based therapy when indicated. Medications such as heparin or glycoprotein IIb/IIIa inhibitors can be continued in the field, but revascularization will occur in the hospital.

Another situation that warrants fibrinolytic therapy is marked ST-segment depression in leads V_1 through V_4 with tall R waves in the right precordial leads with upright T waves suggestive of a true posterior wall MI (PWMI). In this case, placement of additional electrodes in the posterior position to form leads V_7, V_8, and V_9 is recommended. Primary percutaneous coronary intervention may be appropriate in patients with true PWMIs.

Lethal ventricular arrhythmias may develop in patients with STEMI, so continuous ECG monitoring is required. Because many patients with STEMI are transferred to a higher level of care for therapeutic interventions, CCTPs are in a unique position to be monitoring patients during transport and to be able to respond quickly and appropriately as these events occur.

Most patients diagnosed with ACS are experiencing an MI in progress or severe ischemia, which can quickly become an infarction. The patients are best treated at a facility where the location of the blockage can be identified by cardiac catheterization and subsequently alleviated by stenting. Unfortunately, most hospitals do not have this capability.

The role of the critical care transport team is to transport the patient from a basic hospital to a cardiac hospital with the least possible decline in the patient's condition and to prevent further loss of heart muscle. Priorities are as follows:

1. Prevent further damage to the heart muscle
2. Reduce afterload
3. Prevent the clot from getting larger
4. Reduce myocardial oxygen demand
5. Maximize oxygen delivery
6. Reduce or eliminate pain and anxiety
7. Consider tissue plasminogen activators (TPAs) to dissolve the clot or clots

If the blood pressure is elevated, the patient should receive a nitroglycerin (Nitro-Bid IV, Tridil) drip to control blood pressure. Chest pain is managed with morphine, hydromorphone (Dilaudid), or fentanyl citrate. If there is any indication of pulmonary edema, continuous positive airway pressure (CPAP) should be started or the patient should be intubated and a ventilator with positive end-expiratory pressure (PEEP) should be used if pulmonary edema is severe. Lasix should be administered per protocol.

The patient will usually be receiving an anticoagulant heparin drip and, in some cases, an IV drip of an antiplatelet medication, such as eptifibatide (Integrilin) may also be added. These drips must be administered via an infusion pump. The patient should be monitored en route for signs of serious bleeding. Minor bleeding, especially around the mouth, is common and is not a reason to discontinue or alter the drip rates.

The patient should be given sufficient oxygen to maintain an oxygen saturation of greater than 95%. Patients who need a ventilator should be monitored with waveform capnography, and a carbon dioxide level between 35 and 45 mm Hg should be maintained.

When transport times will be long or the degree of blockage is severe to the point of being life

threatening, a TPA may be required. TPAs actually dissolve clots; however, they dissolve clots throughout the body. Before administering TPA, all of the following exclusion criteria must be met. The patient cannot have or have received the following:

1. Recent major surgery
2. Recent intracranial or spinal surgery
3. Active internal bleeding
4. A history of stroke within the last 6 months
5. Intracranial neoplasm or arteriovenous malformation
6. Severe uncontrolled hypertension
7. Pregnancy
8. Anticoagulants (such as warfarin [Coumadin])

The most common TPA for an active MI or ACS is reteplase (Retavase). Reteplase is administered in two separate doses. The first dose is 10 U administered in a 2-minute period. The second dose is also 10 U administered in 2 minutes. Reteplase is not to be administered as a rapid bolus or through a heparin line. It is not uncommon to administer heparin simultaneously with reteplase. Heparin is started after the first dose of reteplase as an initial IV bolus at 60 U/kg (maximum dose, 4,000 U) followed by a drip of 12 U/kg/h (maximum dose, 1,000 U).

Transport Management

Acute Coronary Syndrome (Interfacility Transport)

- Start a nitroglycerin (Nitro-Bid IV, Tridil) drip if the patient's blood pressure is elevated.
- Give morphine, hydromorphone (Dilaudid), or fentanyl citrate to manage chest pain.
- If there is any indication of pulmonary edema, begin CPAP or intubate the patient.
- If pulmonary edema is severe, the patient needs a ventilator with PEEP.
- Give furosemide (Lasix) per protocol.
- Monitor the heparin (anticoagulant) and eptifibatide (Integrilin; antiplatelet) drips.
- Monitor the patient for signs of bleeding and headache with altered level of consciousness.
- Give sufficient oxygen to maintain an oxygen saturation of greater than 95%.
- If the patient needs a ventilator, monitor with waveform capnography, and maintain the carbon dioxide level between 35 and 45 mm Hg.
- If the transport time will be long or the degree of blockage is severe to the point of being life threatening, administer TPAs.
- Monitor the cardiac rhythm.
- If severe bleeding occurs after administration of a TPA, turn off the heparin, manage hypotension with fluids or dopamine (Intropin), and divert to the closest hospital for further treatment.

The patient's cardiac rhythm must be monitored following the administration of reteplase because reperfusion arrhythmias are likely to follow. When cardiac tissue becomes severely ischemic, potassium leaves the intracellular space. With reperfusion, the potassium rapidly returns to the intracellular spaces, creating rhythm disturbances. Usually these disturbances are short-lived, but serious rhythm disturbances,

including cardiac arrest, have occurred. Patients must be monitored for signs of bleeding (eg, rigid abdomen with hypotension) and a headache with an altered level of consciousness. If severe bleeding occurs, the heparin drip must be turned off, hypotension must be managed with fluids or dopamine (Intropin), and the transport must be diverted to the closest hospital for treatment, possibly including a blood transfusion.

Angina Pectoris

The principal symptom of CAD is angina pectoris. Angina occurs when the supply of oxygen to the myocardium is insufficient to meet the demand. As a result, the cardiac muscle becomes ischemic, and a switch to anaerobic metabolism leads to the accumulation of lactic acid and carbon dioxide. A person who experiences angina at rest, when oxygen needs are minimal, has more severe CAD than a person who experiences angina only with vigorous exercise.

When obtaining the medical history from a patient with chest pain, it is important to distinguish between stable angina and unstable angina. Stable angina follows a recurrent pattern: A person with stable angina experiences pain after a certain, predictable amount of exertion, such as climbing one flight of stairs or walking for three blocks. The pain also has a predictable location, intensity, and duration.

Chronic, stable angina is the result of an atherosclerotic lesion causing an inadequate myocardial oxygen supply during exertion. Patients with chronic, stable angina often take nitroglycerin or some other form of nitrate for relief of anginal pain. In the critical care transport environment, nitroglycerin should be given intravenously, especially for long transports. IV nitroglycerin (Nitro-Bid IV, Tridil) is better able to control blood vessel diameter than other forms of nitroglycerin. When IV nitroglycerin is given, all other forms of nitroglycerin should be discontinued. If a patient who is being transported is not taking nitroglycerin and chest pain suddenly develops, a sublingual nitroglycerin tablet or nitroglycerin paste may be used initially to reduce afterload while IV nitroglycerin is being set up.

Unstable angina is much more serious than stable angina and indicates a greater degree of obstruction of the coronary arteries. It is caused by a thrombus or a rupture of a plaque. It is characterized by noticeable changes in the frequency, severity, and duration of pain and often occurs without predictable stress. The patient may report that the anginal attacks have grown more frequent and severe during the past several days or weeks, that they awaken him or her from sleep, or that they occur when otherwise at rest. Nitroglycerin is often not effective with this type of angina. Such attacks are often warning signs of an impending MI.

Finally, silent ischemia is the presence of angina without clinical manifestations and is only detected when ST-segment elevation is noted.

Myocardial Infarction

An MI can occur as a result of the cardiac muscle being deprived of coronary blood flow long enough for myocardial tissue death to ensue. Narrowed vessels (eg, from atherosclerotic disease), coronary artery occlusion (eg, by a thrombus), spasm of a coronary artery, or reduction of overall blood flow (eg, from shock, arrhythmias, or pulmonary embolism) are causes of an MI.

The location and size of an MI depend on which coronary artery is blocked and where along its course the blockage occurred. The majority of infarcts involve the left ventricle. When the anterior, lateral, or septal walls of the left ventricle are infarcted, the source is usually occlusion of the left coronary artery or one of its branches. Usually, IWMIs are the result of an RCA occlusion. When the ischemic process affects only the inner layer of muscle, the infarct is referred to as subendocardial. An infarct that extends through the entire wall of the ventricle is a transmural MI. The infarcted tissue is invariably surrounded by a ring of ischemic tissue—an area that is relatively deprived of oxygen but is still viable. That ischemic tissue tends to be electrically unstable and is often the source of cardiac

arrhythmias.

Acute MI is the leading cause of death in the United States, accounting for more than 500,000 deaths per year; of those deaths, 60% to 70% occur outside the hospital, during the first 2 to 3 hours after the onset of symptoms. Patients at risk for sudden cardiac death include patients with the following:

- Prior sudden cardiac arrest
- Prior MI
- Heart failure, class II to IV
- Ejection fraction of less than 40%
- Family history of cardiac arrest
- Prolonged QT interval

Of all deaths of acute MI, 90% are due to arrhythmias, usually ventricular fibrillation, which typically occur during the early hours of the infarct. Arrhythmias can be prevented or treated, so most deaths from acute MI are preventable.

Infarct Recognition and Localization

Many cardiac patients are transported from one facility to another to receive specialized procedures that are not available from the sending hospital. The physical and emotional stressors of the transport can place an additional burden on a patient in already critical condition. CCTPs must always remain acutely aware of the patient's cardiac status with rapid identification of new findings—including MI—and evolving and changing events.

There are three initial components to the diagnosis of an MI: history and physical examination, testing for cardiac enzymes, and interpretation of ECG changes associated with an MI. An MI evolves from a normal state to ischemia, injury, and, ultimately, infarction because of an event that occludes vessels and limits the oxygen-enriched blood reaching the cells. Ischemia and injury are reversible conditions that must be identified and treated to help prevent death of cardiac muscle.

As soon as a chief complaint of a cardiac nature has been noted, treatment of the patient needs to start; obtaining a focused history and performing a physical examination can wait. However, for purposes of discussion, this section proceeds through the history and physical examination.

Cardiac Enzymes

Serum tests for cardiac enzymes are not typically done during transport but are discussed here for review. Test results are frequently available in the patient's chart and may help put into perspective the patient's cardiac status. Although the ECG does not directly represent levels of cardiac enzymes that are released as cells are damaged, ECG changes are seen as an aftermath of cell damage because of the change in cell function. As myocardial cells die during an infarction, they leak their internal contents into the bloodstream. Troponins T and I are contractile proteins of the myofibril that are specific for detecting myocardial cell injury. Troponins T and I are the forms most commonly tested. The levels rise during the first 2 to 6 hours after injury. They peak from 12 to 16 hours. The cardiac troponin I level can remain elevated for 5 to 10 days, and the cardiac troponin T level can stay elevated for 5 to 14 days.

Creatine kinase (creatine phosphokinase) is an enzyme found in heart (MB fraction) and skeletal (MM fraction) muscle and in the brain (BB fraction). Creatine kinase increases in more than 90% of MIs. Because it is also found in skeletal muscle and the brain, it can be elevated during trauma and physical exertion, in postoperative patients, in patients with seizures, and in those with other conditions in which cells can be damaged. In an MI, the creatine kinase level begins to rise in 4 to 6 hours. It peaks in 24 hours and returns to normal in 3 to 4 days. The creatine kinase isoenzyme MB fraction is specific to cardiac muscle. The MB fraction rises and returns to normal sooner than the total creatine kinase level. It

rises in 3 to 4 hours and returns to normal in 2 days.

Myoglobin is an oxygen-transport protein. Damage to skeletal or cardiac muscle causes it to be released into the circulation. After an MI, the myoglobin level rises in the first 2 hours. It peaks in 6 to 8 hours and returns to normal in 20 to 36 hours. An increased myoglobin level can also be found in skeletal muscle injury and renal failure.

Because several of these markers can be found with other medical conditions, they must be used in conjunction with other clinical and laboratory findings when a cardiac event is suspected.

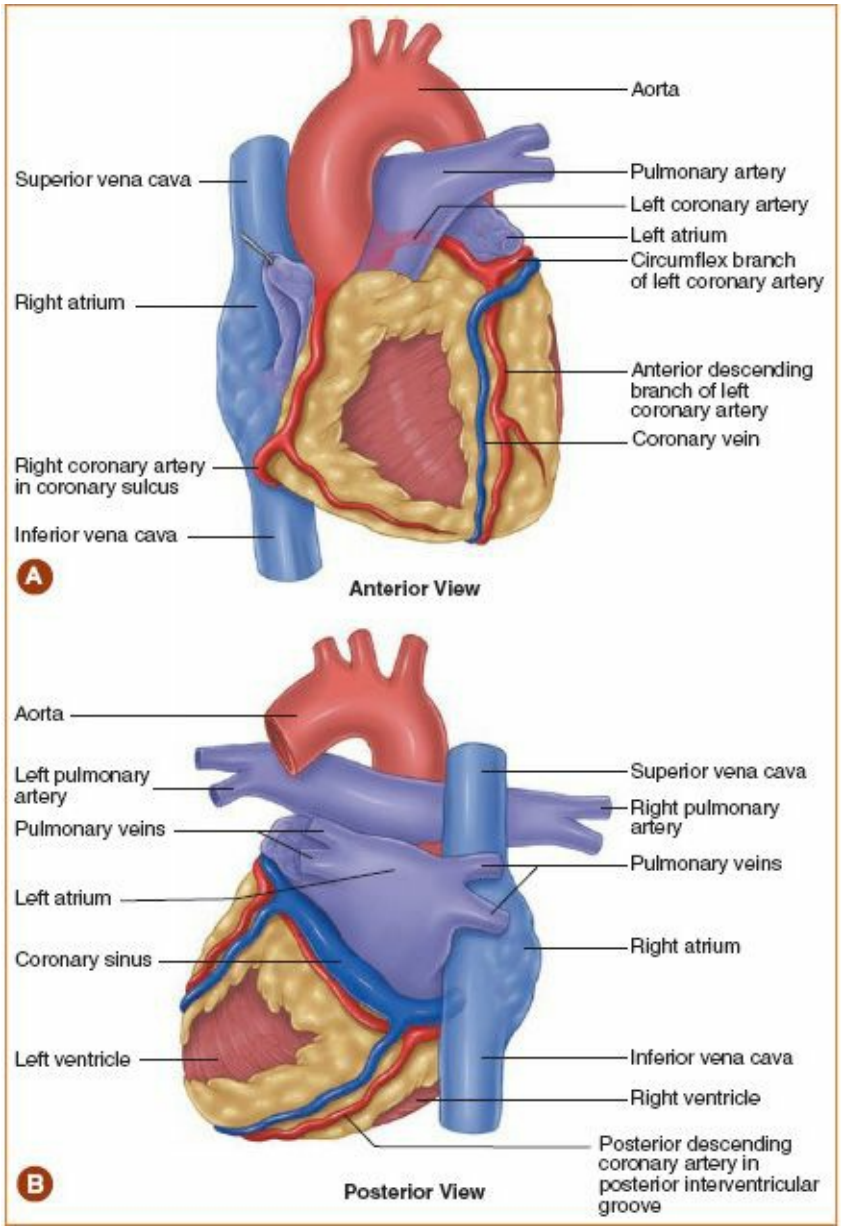


Figure 13-50 Coronary arteries. **A.** Anterior view, showing the takeoff point of left and right coronary arteries from the aorta. **B.** View from below and behind, showing the coronary sinus.

Blood Supply

Before proceeding further, a review of how blood circulates throughout the heart is given to aid understanding of the ECG changes seen in an MI. The heart is a muscle and requires an oxygen-enriched blood supply to survive. The blood supply to the heart is delivered through two main coronary arteries and their branches **Figure 13-50** during diastole. Areas that are supplied by more than one source are said to have collateral circulation. The right and left coronary arteries originate at the base of the aorta just above the aortic valve. The major branches of the RCA are the marginal, posterior (descending)

interventricular, and SA nodal branch. The RCA supplies the right atrium, right ventricle, and portions of the left atrium and left ventricle. The sinus node branch supplies the sinus node, and the AV nodal branch supplies blood to the AV node.

The major branches of the left coronary artery are the anterior interventricular, also known as the left anterior descending, and left circumflex branches. The left coronary artery supplies both ventricles, the interventricular septum, and the left atrium. The left anterior descending artery supplies the right and left bundle branches.

The deoxygenated blood is returned to the venous circulation through the coronary sinus. By understanding the coronary blood flow and the areas of the heart represented by the ECG, CCTPs can use ECG changes to identify which branch of the blood supply is affected. Note: There are slight variances in coronary artery distribution from patient to patient.

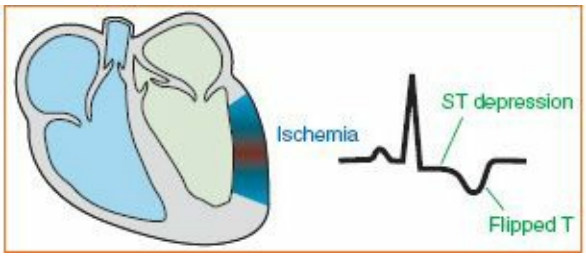


Figure 13-51 Ischemia.

Ischemia, Injury, and Infarction

Decreased blood flow to the heart can result from a chronic condition called coronary vascular disease or an acute injury in which a coronary artery or one of its branches is occluded by an embolus. The heart begins in a normal state in which it is receiving an adequate blood supply. Increased oxygen demand caused by an increased workload or a decrease in supply caused by coronary vascular disease or a blocked artery can lead to ischemia, injury, and, ultimately, infarction. Ischemia and injury are reversible, and when evidence of either is present, rapid intervention can often prevent infarction.

When ischemia develops, the tissue becomes more electrically negative compared with the unaffected surrounding tissue. This change causes a pattern of ST-segment depression. The T wave is also inverted (flipped) because of repolarization occurring along abnormal pathways **Figure 13-51**.

There are other causes of T-wave inversion, and they include ventricular hypertrophy with strain, BBB, and cerebral hemorrhage. To help distinguish ischemia from one of the other causes of T-wave inversion, the shape of the T wave may be helpful. With myocardial ischemia, the T wave inverts symmetrically. With other causes, the T-wave inversion is often asymmetric or “slurred.”

As ischemia continues uninterrupted, injury develops. The area that is injured does not repolarize completely, causing it to become more electrically positive than the unaffected area surrounding it. This change causes the ST segment to become elevated. The T wave remains inverted because it continues to repolarize through abnormal pathways. As the injury progresses, the elevation of the ST segment can increase

As ischemia and injury continue uninterrupted, infarction can occur. The infarcted tissue no longer generates or transmits electrical impulses. The area becomes “electrically neutral.” Because this tissue can no longer generate or transmit electrical impulses, no direct wave is recorded on the ECG. Electrical impulses are deflected away from the area of infarct and are represented by Q waves. Another way of looking at Q-wave formation is as a result of electrical impulses being generated by the other wall of the ventricle and passing unopposed away from the positive electrode. Because ischemia and injury continue to be present until the infarct process is completed, ST-segment elevation and inverted T waves are still

present **Figure 13-53**.

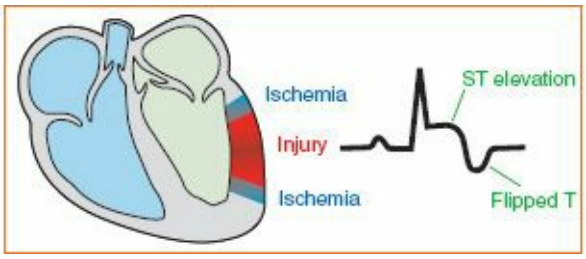


Figure 13-52 Ischemia and injury.

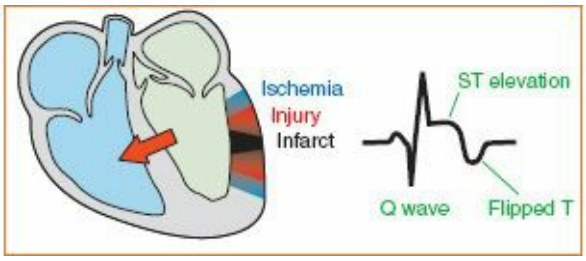


Figure 13-53 Ischemia, injury, and infarction.

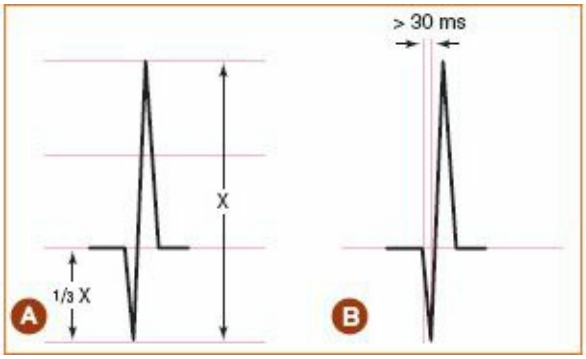


Figure 13-54 Q-wave significance. **A.** If the Q wave is more than one third the total height of the QRS, it is a pathological Q. **B.** If the Q wave is more than 30 milliseconds wide, it is a pathological Q.

Sometimes small Q waves are seen in the left lateral leads (I, aVL, V_5, and V_6) and occasionally in the inferior leads (especially II and III) of normal hearts. The normal small Q waves occur as a result of early left-to-right depolarization of the interventricular septum. For Q waves to be significant, indicating pathology, they must be more than one third the total height of the QRS they appear with and wider than 30 milliseconds in duration **Figure 13-54**.

An MI can occur with or without Q waves. Q waves can develop as discussed, especially when larger areas of myocardium are affected. With a smaller infarct, the cell-to-cell conduction of electrical impulses can hide the smaller resulting Q wave. Because ischemia and injury are always present in the setting of MI, ST-segment and T-wave abnormalities will still be present. When a patient has a history, signs and symptoms, and ECG changes suggestive of a cardiac event, a full workup is indicated. In this case, evaluation of cardiac enzymes can be the key to making the correct diagnosis.

Generally, STEMIs are associated with a higher incidence of congestive heart failure (CHF) and death because of the larger area of heart tissue damaged. In NSTEMIs, there is a higher incidence of long-term mortality because of life-threatening arrhythmias that develop out of the area of infarction.

Quick recognition of ischemia, injury, and infarction, followed by rapid intervention, can reduce the area of infarction and decrease the incidence of subsequent cardiac events that lead to death.

As mentioned earlier in this section, electrical activity in infarcted myocardial tissue becomes electrically neutral. As a result, the electrical current will be directed away from the area of infarction. The electrode that lies over the area of infarction will record a deep negative deflection, a Q wave. The electrical current that is directed away from the area of infarction causes **reciprocal changes** in leads located 180° from the site of infarction. Reciprocal leads must be 180° from the lead in question and lie in the same plane. Limb leads and precordial leads cannot be reciprocal. Limb leads lie in the frontal or vertical plane, and precordial leads lie in the horizontal plane. This apparent increase in electrical force moving toward the opposite lead will be recorded as tall, positive R waves. This applies not only to Q waves, but also to ST-segment and T-wave changes. For example, a Q wave, an ST-segment elevation, and a T-wave inversion recorded in leads II, III, and aVF will be recorded as a tall R wave, ST-segment depression, and an upright T wave in the reciprocal leads I and aVL **Figure 13-55 Table 13-8**.

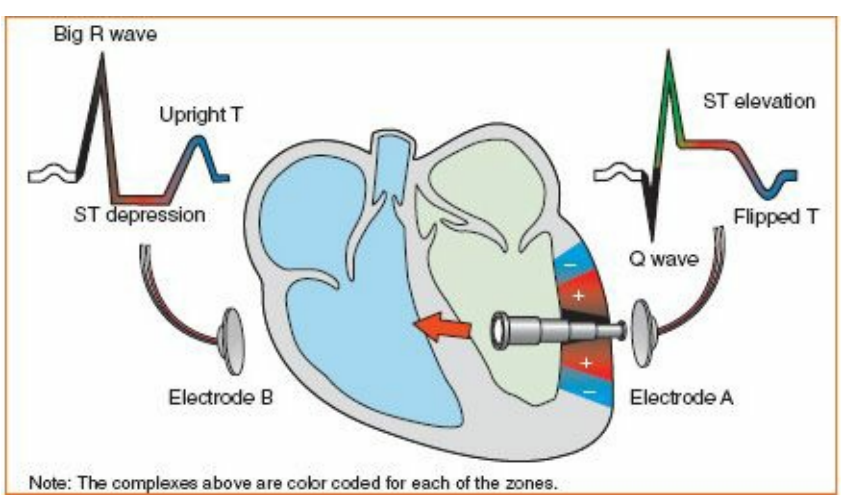


Figure 13-55 Reciprocal changes.

Localizing the Infarction

The area of myocardium that infarcts depends on which coronary arteries are occluded and the extent of collateral blood flow. It is important to localize where an infarction has occurred because the prognostic and therapeutic implications are determined largely by the area of the heart that has died. The review of coronary blood supply shows that each of the arteries perfuses more than one area in the heart. So, if one coronary artery becomes blocked, more than one region of the heart can become ischemic and proceed to injury and, ultimately, infarction. ECG findings, therefore, do not always fall neatly into one anatomic area of infarction. Acute MIs often involve more than one region of the heart; therefore, an inferior MI is often an inferoposterior MI, an anterior MI can become anteroseptal or anterolateral, lateral MIs are often posterolateral, and so forth **Figure 13-56**. Q waves from old infarctions can be present with findings of a new MI and can contribute to making the correct diagnosis from the ECG more challenging. In the critical care transport setting, the patient by definition is in serious condition and will likely have evidence of one or more underlying conditions. This makes it even more important to review an "old" ECG from the patient's chart, acquire a new 12-lead ECG before transport, and make serial evaluations along the way. To be sure an ECG change has occurred, CCTPs must know what was there before coming into contact with the patient.

With all this in mind, in general, infarctions can be grouped into several general anatomic categories **Table 13-9**.

Acute Anterior Myocardial Infarction

An anterior wall MI seldom occurs alone. It involves the anterior surface of the left ventricle and is

usually caused by occlusion of the left anterior descending artery **Figure 13-57**. Leads V_3 and V_4 are considered the anterior leads **Figure 13-58**. In an anterior MI, the normal pattern of R-wave progression may not occur. This situation is called **poor R-wave progression**. Even without significant Q-wave formation, poor R-wave progression may signify an anterior MI. Poor R-wave progression can also be seen in RVH and chronic lung disease and with improper lead placement. Alone, it is not specific for the diagnosis of anterior MI.

TABLE 13-8 Reciprocal Leads and Their Corresponding Locations

Location	Facing Leads	Reciprocal Leads
Anterior	V_3, V_4	None
Inferior	II, III, aVF	I, aVL
Lateral	I, aVL, V_5, V_6	II, III, aVF
Septum	V_1, V_2	None
Posterior	None	V1, V2

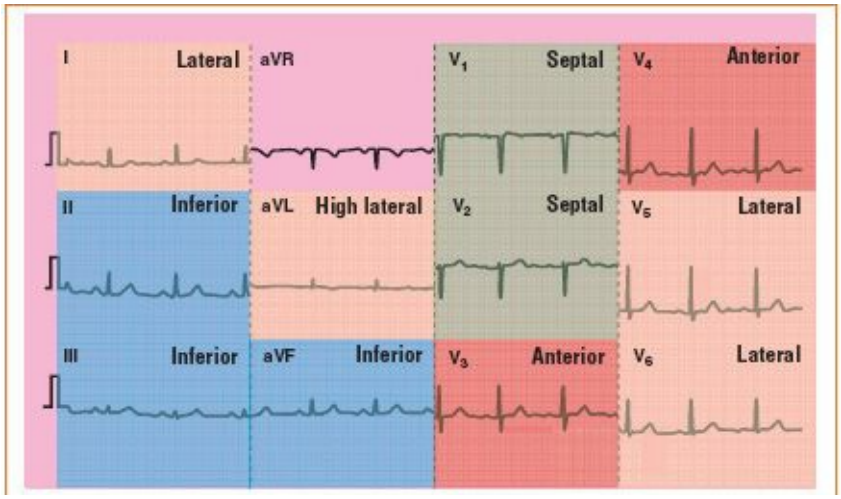


Figure 13-56 Correlation between areas of the heart and leads on the electrocardiogram.

TABLE 13-9 Coronary Artery Relationships to Area Perfused

Area of the Heart	Main Arteries That Perfuse the Area
Inferior	RCA (90%), LCx (10%)
Inferior right ventricle	Proximal RCA
Inferoposterior	RCA (90%), LCx (10%)
Isolated right ventricle	LCx
Isolated posterior	RCA (90%), LCx (10%)
Anterior	Left anterior descending
Anteroseptal	Left anterior descending

Anteroseptal-lateral	Proximal left anterior descending
Anterolateral, inferolateral, or posterolateral	LCx

Abbreviations: LCx, left circumflex artery; RCA, right coronary artery.

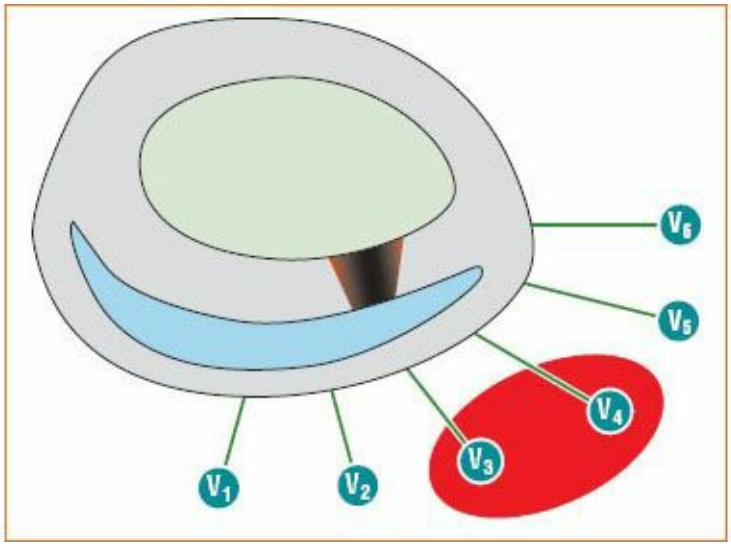


Figure 13-57 Anterior wall myocardial infarction.

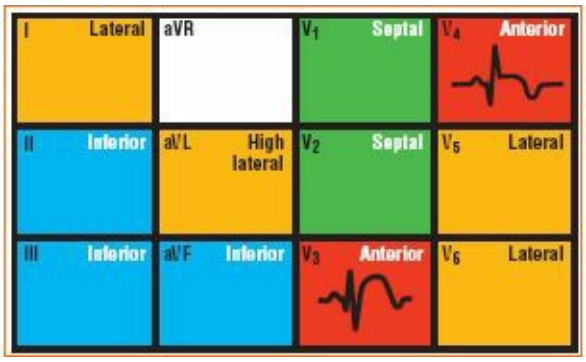


Figure 13-58 Anterior leads.

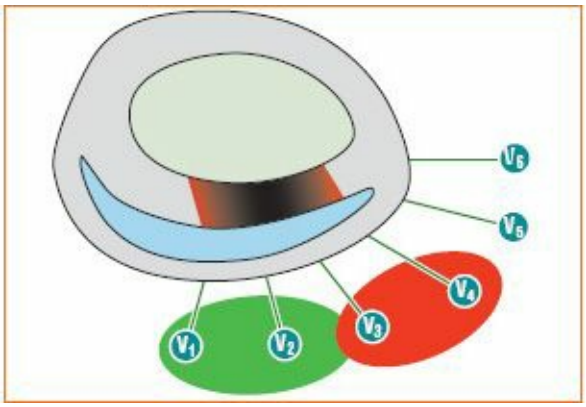


Figure 13-59 Anteroseptal myocardial infarction.

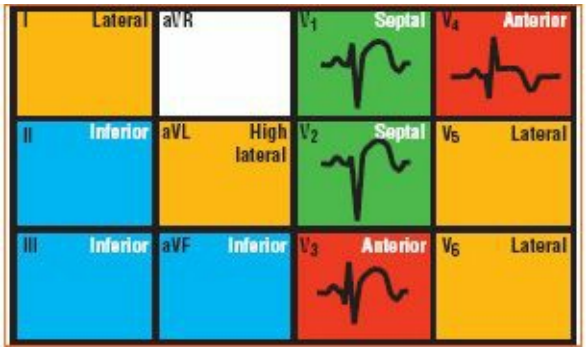


Figure 13-60 Anteroseptal leads.

Acute Anteroseptal Myocardial Infarction

An anteroseptal MI is common with an occlusion of the left anterior descending artery. As with all anterior MIs, this presentation is often associated with hemodynamic compromise and cardiogenic shock. The leads that best view this pattern of MI are the septal leads V_1 and V_2 and the anterior leads V_3 and V_4 **Figure 13-59** and **Figure 13-60**.

The ECG in **Figure 13-61** represents an acute anteroseptal MI. In the anterior and septal leads, V_1 through V_4, there is marked elevation and flattening of the ST segment, which is typical of this pattern of infarction. Further evaluation of the ECG shows some minimal elevation of the ST segment in lead aVL. All three areas are perfused by the left anterior descending artery. ST-segment depression is found in the reciprocal leads, II, III, and aVF.

Acute Anteroseptal Myocardial Infarction With Lateral Wall Extension

If the proximal left anterior descending artery is occluded, an acute anteroseptal MI with lateral wall extension may result. Changes can be seen in all precordial leads (specifically V_5 and V_6 for the lateral extension) and in leads I and aVL. With the loss of electrical impulses in the anterior infarcted tissue, there is not always significant Q-wave formation. Reciprocal changes can be found in leads II, III, and aVF **Figure 13-62** and **Figure 13-63**.

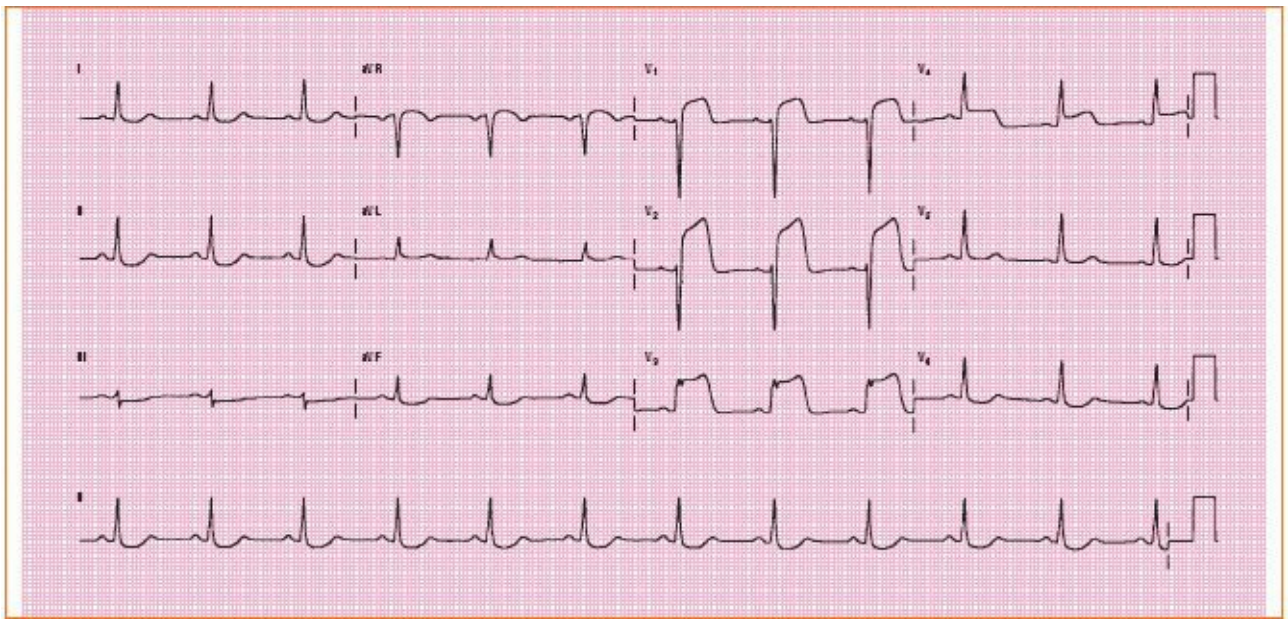


Figure 13-61 A 12-lead electrocardiogram showing an acute anteroseptal myocardial infarction.

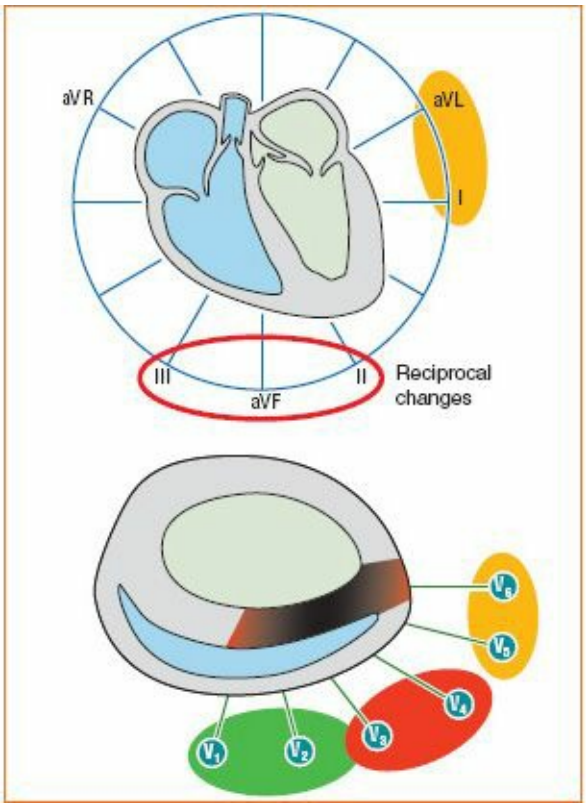


Figure 13-62 Acute anteroseptal myocardial infarction with lateral wall extension.

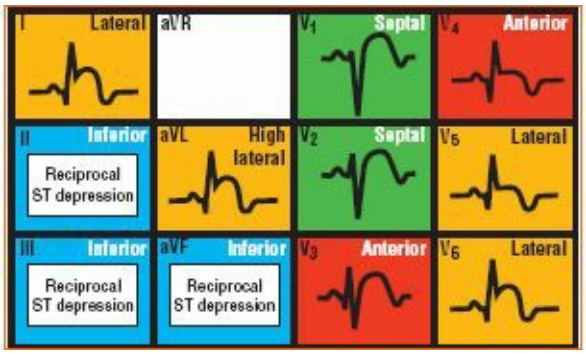


Figure 13-63 Electrocardiographic changes seen in an anteroseptal myocardial infarction with lateral wall extension.

The ECG in **Figure 13-64** represents an acute anteroseptal MI with lateral wall extension. There is ST-segment elevation in leads V_2 through V_6 that extends to leads I and aVL. Reciprocal changes are seen in leads III and aVF. In addition to the signs of ischemia and injury, there are Q waves forming in V_3 to V_6 and in leads I and aVL. In this ECG, the ST segments are very elevated and the T waves are tall and peaked, often referred to as hyperacute T-wave changes, indicating an early acute MI. The hyperacute T-wave changes occur only during the first 15 to 30 minutes of an acute MI. When they occur out of the hospital, they are seldom seen because responders usually arrive after the period when these changes can be seen. If these changes are found during transport, CCTPs are in a unique position to initiate care, even if it is only early notification of the receiving facility to decrease the door-to-treatment time. Early revascularization has a good outcome because the period of ischemia has been relatively short and less permanent damage has been done.

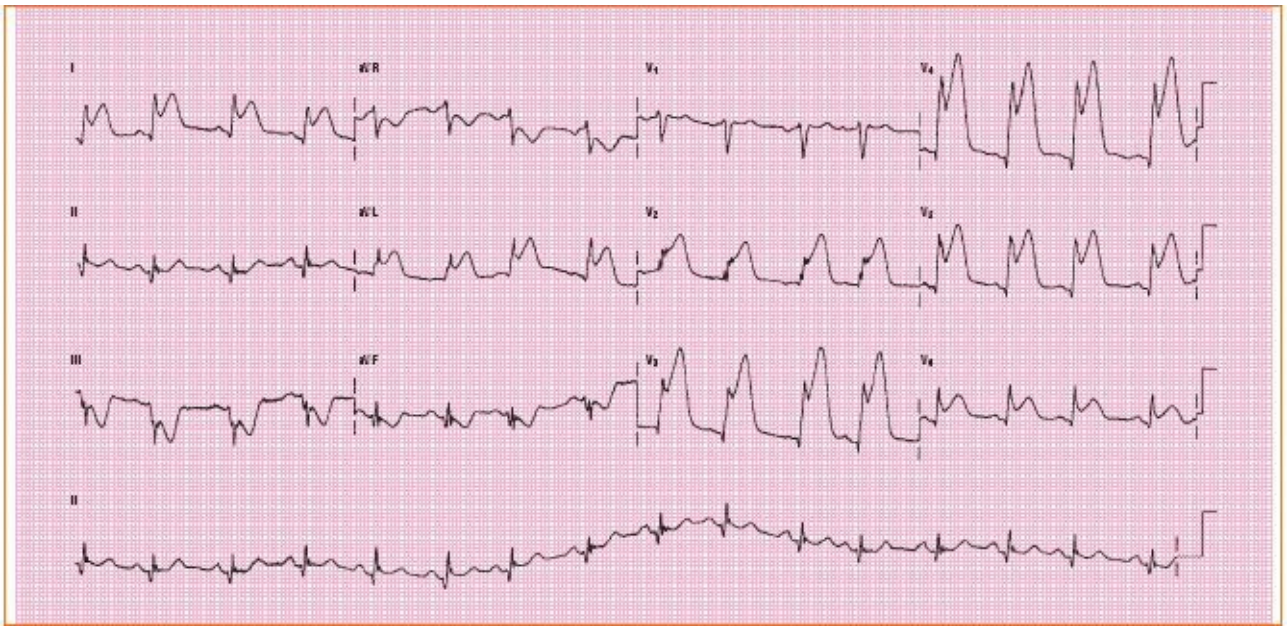


Figure 13-64 A 12-lead electrocardiogram showing an acute anteroseptal myocardial infarction with lateral wall extension.

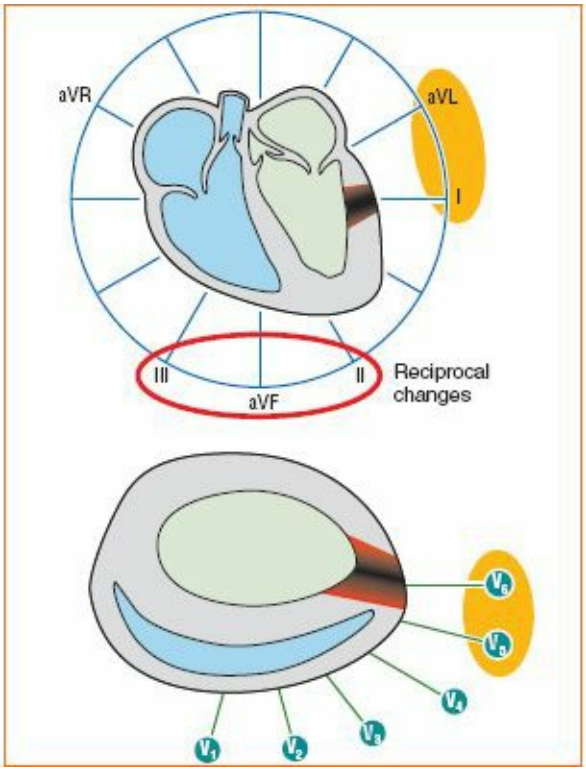


Figure 13-65 Lateral wall myocardial infarction.

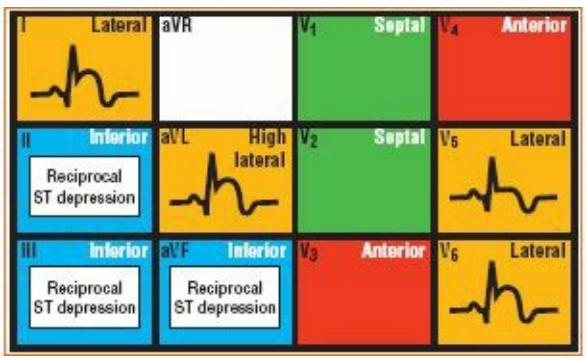


Figure 13-66 Electrocardiographic changes seen with lateral wall myocardial infarction.

Acute Lateral Wall Myocardial Infarction

An acute lateral wall MI involves the left lateral wall of the heart. It can occur alone or with other patterns of infarction. It is often the result of occlusion of the left circumflex artery. ECG changes often occur in the lateral leads, I, aVL, V_5, and V_6. Reciprocal changes may be seen in the inferior leads, II, III, and aVF **Figure 13-65** and **Figure 13-66**.

The ECG in **Figure 13-67** represents an acute lateral wall MI. There is ST-segment elevation in the lateral leads I and aVL and reciprocal changes in the inferior leads II, III, and aVF. Further evaluation shows T-wave inversions in the lateral precordial leads V_5 and V_6. Because the inverted T waves are symmetric, they are related to the acute lateral wall MI and not associated with LVH with strain. An LVH pattern shows an asymmetric inverted T wave.

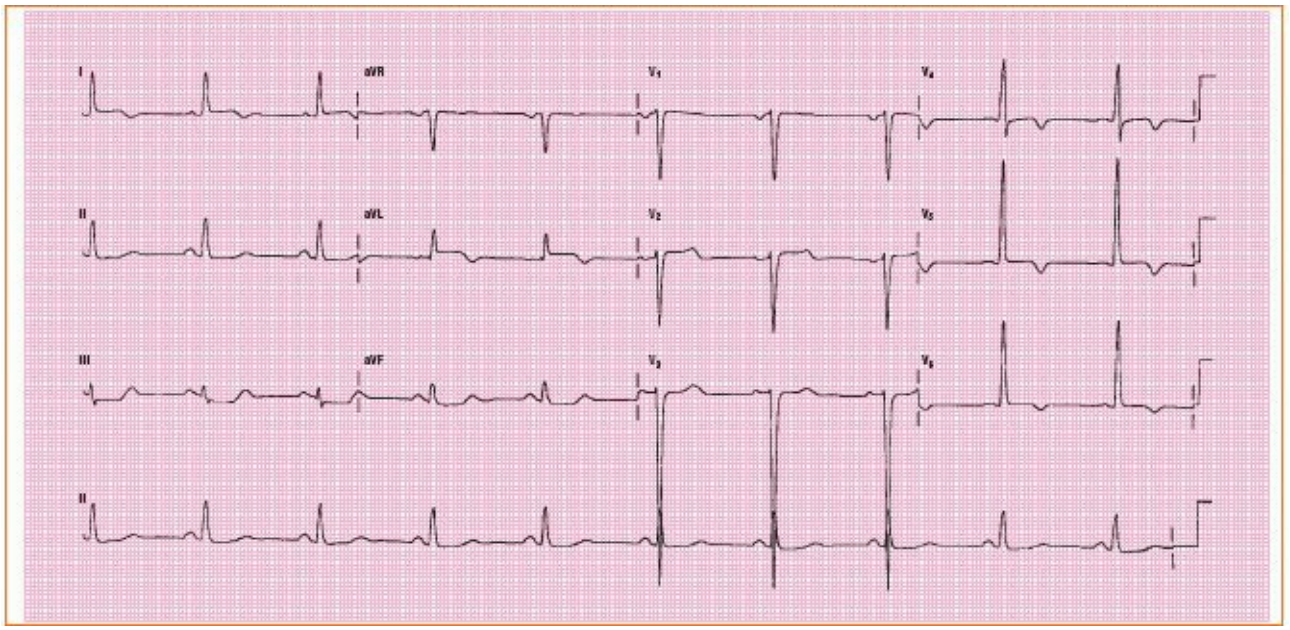


Figure 13-67 A 12-lead electrocardiogram showing an acute lateral wall myocardial infarction.

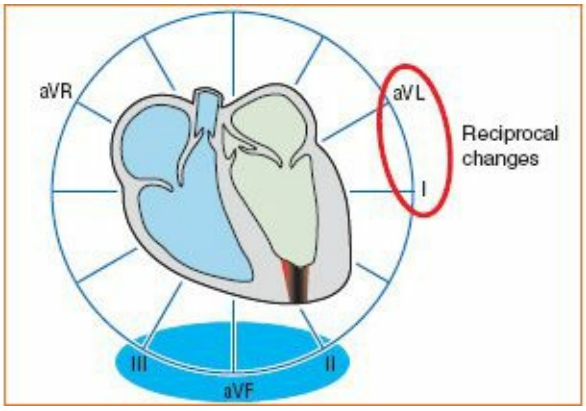


Figure 13-68 An inferior wall myocardial infarction.

Acute Inferior Wall Myocardial Infarction

An IWMI involves the diaphragmatic surface of the heart. It is caused by occlusion of the right coronary artery in 90% of patients and by occlusion in its descending branch or the left circumflex artery in 10% of patients. The characteristic ECG changes of infarction can be seen in the inferior leads II, III, and aVF.

Reciprocal changes are seen as ST-segment depression in leads I and aVL unless the high lateral wall is included in the infarction. An IWMI is commonly seen with patterns reflecting the lateral wall, the posterior wall, and RVIs. Q waves persist for the lifetime of the patient in most cases but not necessarily in inferior infarctions. Nearly 50% of inferior infarctions lose the criteria for significant Q waves within 6 months. Small Q waves in the inferior leads may suggest an old inferior infarction. Small inferior Q waves may be present in healthy hearts, so it is important to use the patient's clinical history in decision making **Figure 13-68** and **Figure 13-69**.

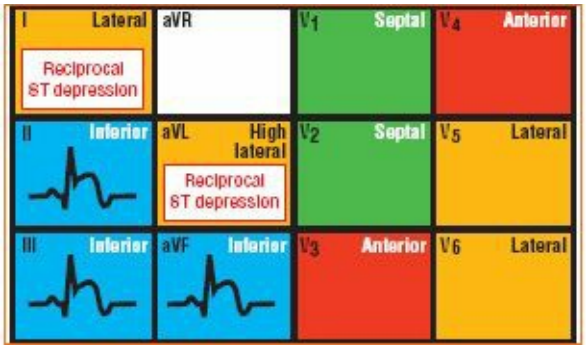


Figure 13-69 Electrocardiographic changes seen with inferior wall myocardial infarction.

The ECG in **Figure 13-70** represents an acute IWMI. Pathologic Q waves appear in leads II and aVF. There are no acute ST-segment or T-wave changes present consistent with an acute infarction. There are also no reciprocal changes found in leads I or aVL. Without signs of acute infarction, pathologic Q waves are considered age indeterminate because without a good patient history, there is no other way to know how old they are. There are also two premature ventricular ectopic beats present that are not necessarily related to the other findings.

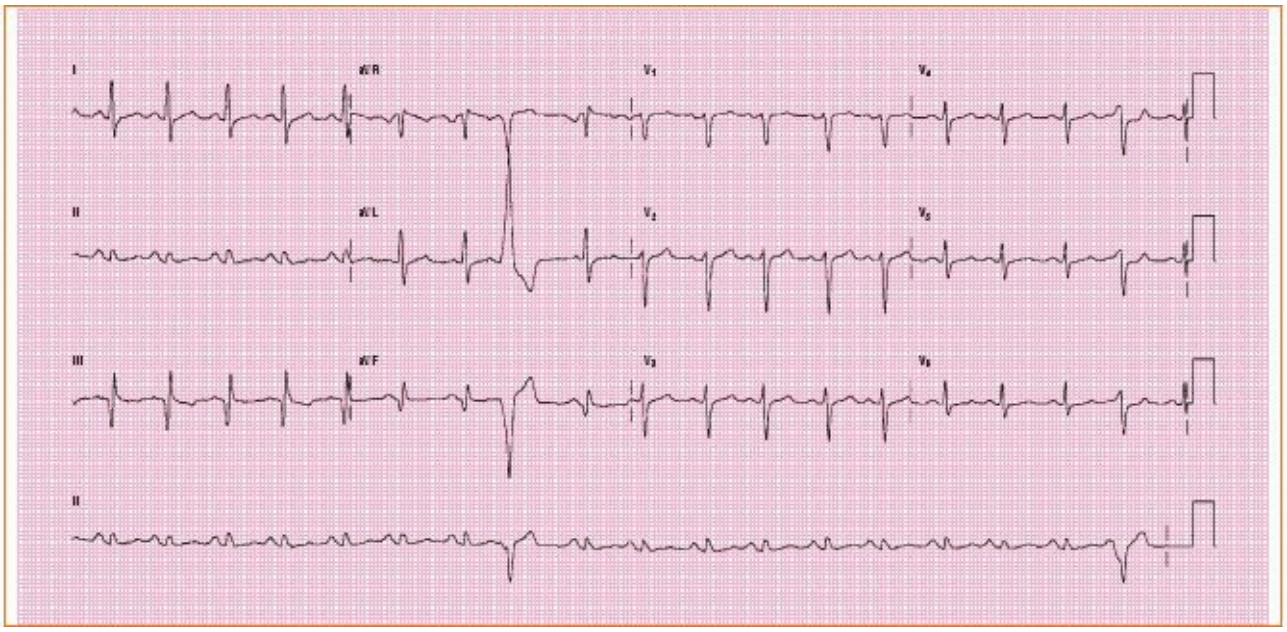


Figure 13-70 A 12-lead electrocardiogram showing an acute inferior wall myocardial infarction.

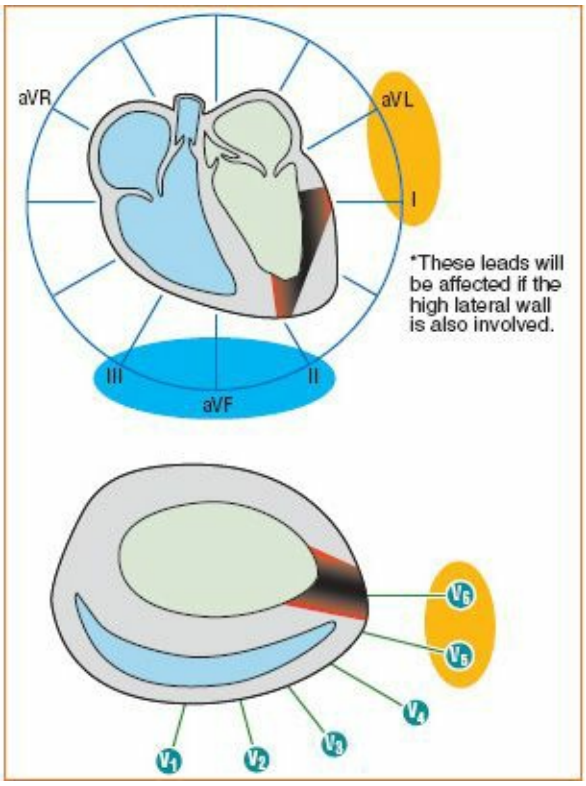


Figure 13-71 Inferolateral leads.

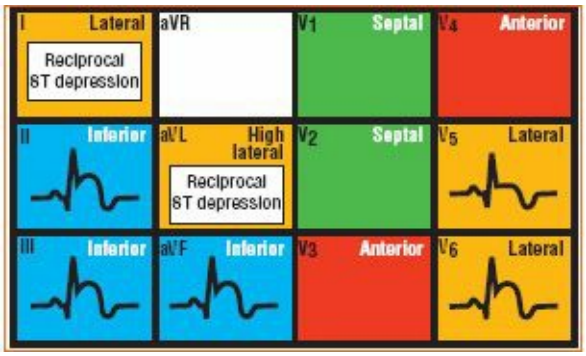


Figure 13-72 Electrocardiographic changes seen with acute inferolateral myocardial infarction.

Acute Inferolateral Myocardial Infarction

ECG changes found in the acute inferolateral MIs are seen in inferior leads II, III, and aVF and in lateral leads I, aVL, V_5, and V_6. ST-segment changes may occur and will be found in leads V_2 to V_4 if the infarct extends anteriorly. When anterior extension is present, classic ST-segment and T-wave changes will always be seen in the lateral precordial leads V_5 and V_6 **Figure 13-71** and **Figure 13-72**.

The ECG in **Figure 13-73** represents an acute inferolateral MI. The ECG shows ST-segment elevation in leads II, III, and aVF. Reciprocal findings can be found as ST-segment depression in leads I and aVF. There is also ST-segment elevation in leads V_3 through V_6. The lateral lead, aVL, shows a markedly downward-sloping ST-segment depression. This is often the first ECG change noted in an acute inferolateral MI, even before ST-segment elevation in the inferior leads.

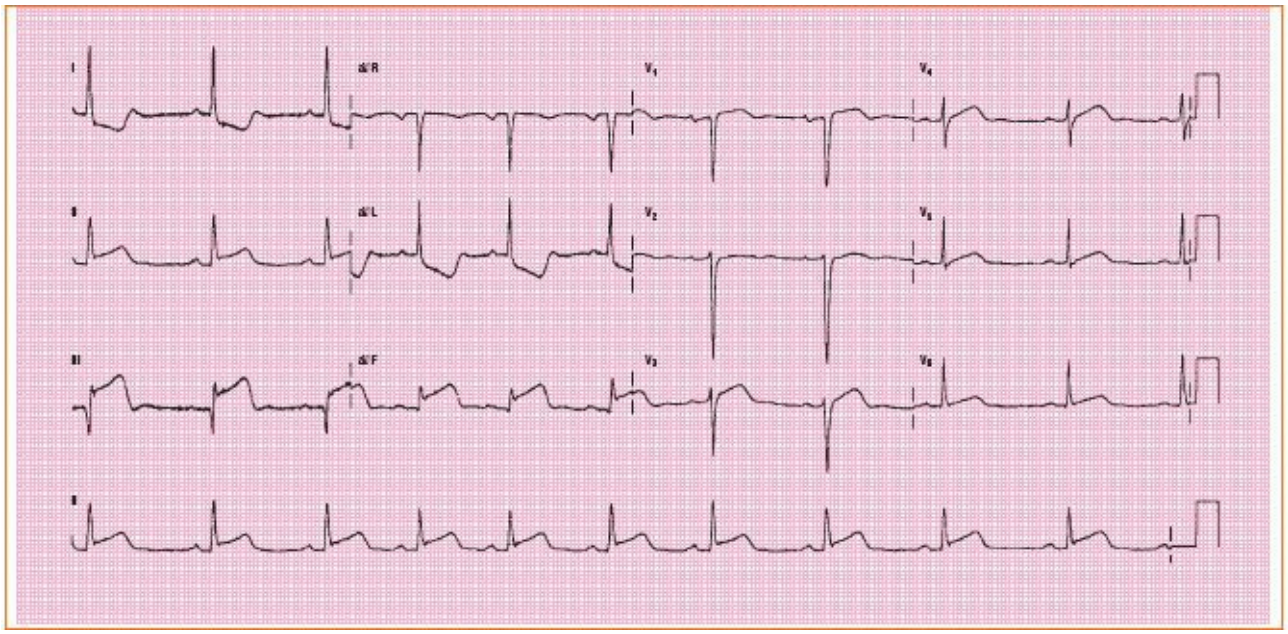


Figure 13-73 A 12-lead electrocardiogram showing an acute inferolateral myocardial infarction.

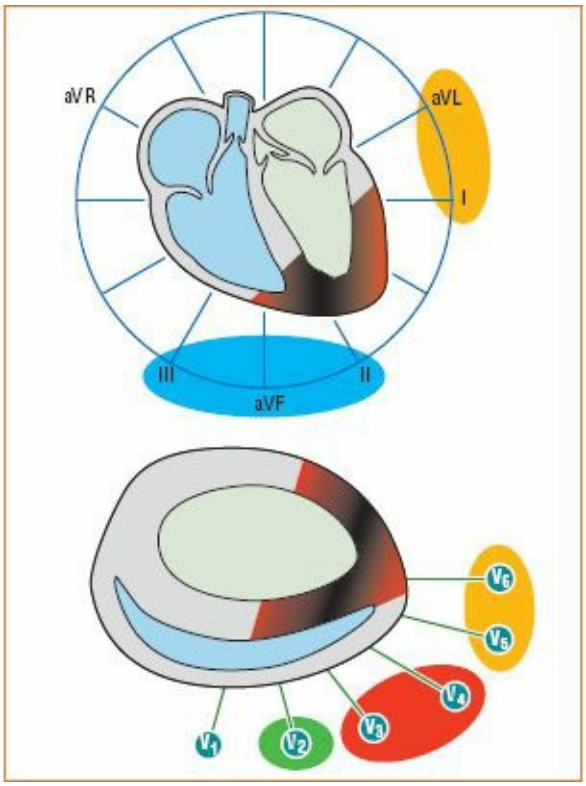


Figure 13-74 Acute apical myocardial infarction leads.

Acute Apical Myocardial Infarction

An apical MI is an extension of an inferolateral MI. It covers a large area of the inferior part of the heart and extends further anteriorly and laterally. The acute apical MI occurs in a patient with RCA dominance. Direct ECG changes can be found in inferior leads II, III, and aVF; lateral leads I and aVL; and precordial leads V_2 through V_6. Because of the diffuse ECG changes, an acute apical MI can be confused with pericarditis **Figure 13-74** and **Figure 13-75**.

The ECG in **Figure 13-76** represents an acute apical MI. Because of the large area affected, ECG changes are diffuse and can be found as ST-segment elevation in limb leads I, II, III, and aVF and

precordial leads V_2 to V_6. This widespread pattern of ECG changes must be distinguished from other conditions such as pericarditis. In pericarditis, there are no Q waves or reciprocal changes, and the T-wave inversion usually does not occur until the ST segment returns to baseline. In an infarction, the ST segment is elevated and the T wave is inverted at the same time. A very rare condition that also demonstrates diffuse ECG changes is aortic dissection, which occludes the coronary ostea (origin of the coronary arteries) on the aorta just distal to the aortic valve. This, among other things, causes a global infarction of the heart because it occludes the source of both main coronary arteries. To rule out some of the more serious differential diagnoses, complete medical histories and clinical evaluations are needed; and radiographic testing, echo-cardiograms, and more sophisticated tests such as cardiac catheterizations must be done.

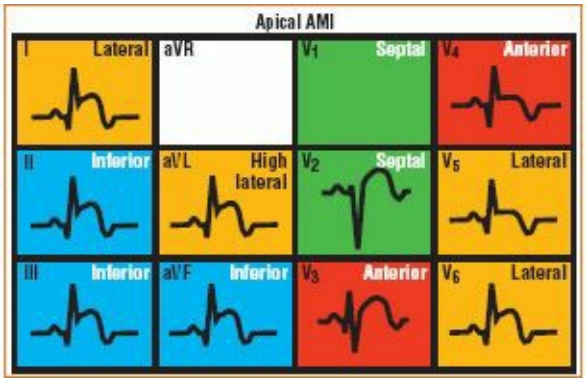


Figure 13-75 Electrocardiographic changes seen with an acute apical myocardial infarction.

Acute Right Ventricular Myocardial Infarction

This section predominantly deals with the criteria for identifying an RVI. Further study on RVI is suggested for a better understanding of the pathophysiology, clinical diagnosis, and management.

An RVI is strongly associated with IWMIs in 30% to 50% of the cases **Figure 13-77** because they are both most often perfused by the RCA and its branches. An occlusion of the RCA would likely affect both regions of the heart at the same time. In a small percentage of cases, the left circumflex artery can supply regions of the inferior wall and right ventricle; therefore, in these cases, an occlusion in the left circumflex artery could cause the same pattern of infarction. An additional 10% of anterior wall MIs are accompanied by RVIs. Because of the high coexistence of RVI with IWMI, a high index of suspicion and checking for other criteria are important.

The ST segment is greater in lead III than in lead II **Figure 13-78**. Because of the RVI, the electrical impulse flows unopposed from the interventricular septum and is directed anteriorly, inferiorly, and to the right. Because this impulse flow causes the vector to be moving toward the lead III electrode, it is recorded with greater amplitude than in lead II.

With ST-segment elevation in (septal) lead V_1 and (inferior) leads II, III, and aVF, RVI should be suspected. Because the electrical impulses are not being transmitted through infarcted tissue, they continue to travel unopposed in all other directions. The alteration in the wave of depolarization generally causes ST-segment elevation in V_1 and ST-segment depression in V_2. Depending on the extent of the MI in the involved heart tissue, ST-segment elevation can sometimes extend through V_5 or V_6.

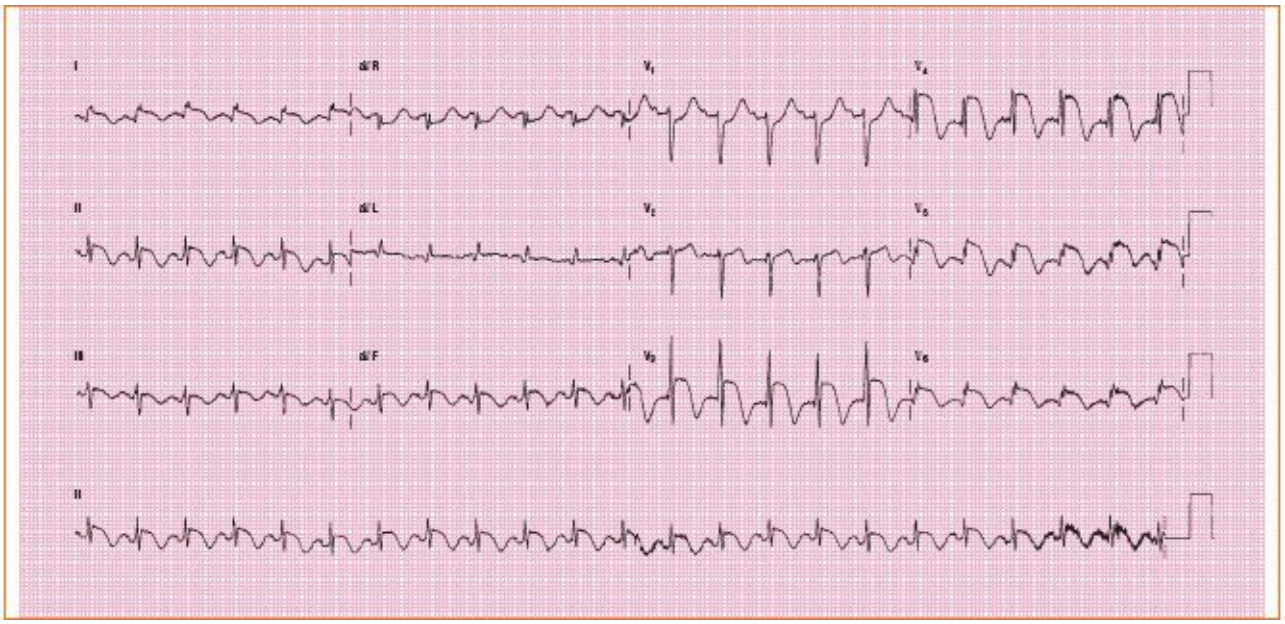


Figure 13-76 A 12-lead electrocardiogram showing an acute apical myocardial infarction.

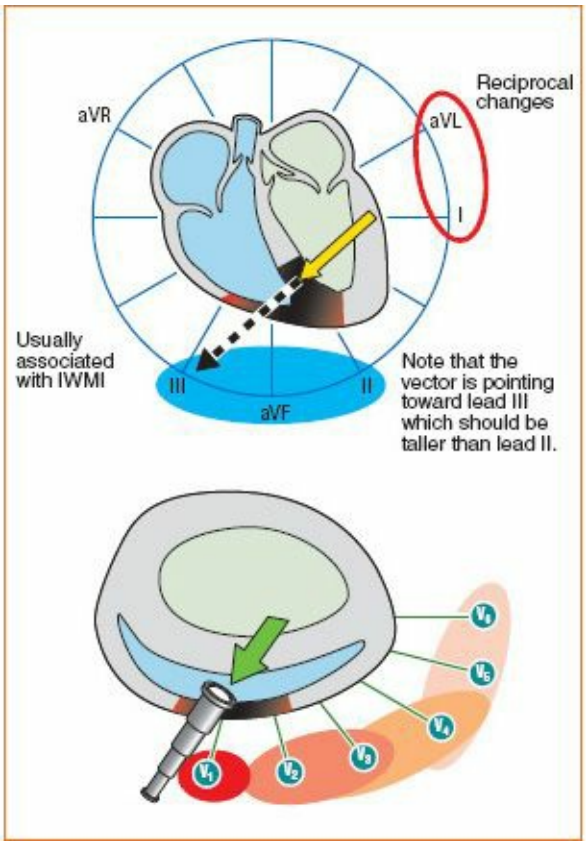


Figure 13-77 A right ventricular infarction.

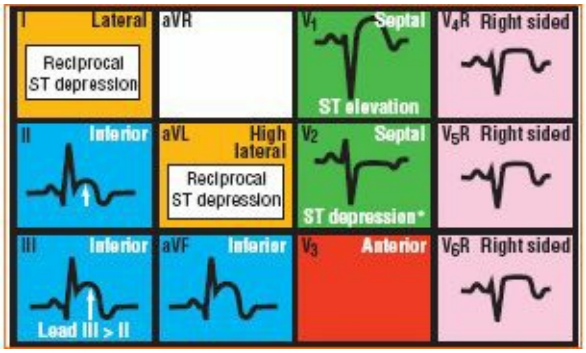


Figure 13-78 Electrocardiographic changes seen with a right ventricular infarction. Note ST elevation in lead V_1, but depending on how much of the RV is affected, it can extend up to V_6! In addition, the amount of ST depression has to be less than half of the ST elevation in aVF!

As mentioned, ST-segment elevation will occur in V_1, but it will usually be depressed in V_2. This difference is because the electrical impulse altered by the MI will cause the vector to travel more directly toward V_1 and past V_2.

An ST-segment depression in V_2 that is less than half the height of the ST-segment elevation in aVF indicates the infarct is localized to the inferior right ventricular region. An ST-segment depression in V_2 that is more than half the height of the ST-segment elevation in aVF indicates that the infarct is larger and includes the inferior wall, right ventricle, and posterior wall.

The use of nonstandard or additional leads on the right side of the chest is important for localizing an infarction. An ST-segment elevation of 1 mm or more in the right-sided chest leads, V_4R to V_6R, is the most specific indication for RVI. Evidence of IWMI (ST-segment elevation and T-wave inversion in leads II, II, and aVF) and also 1 mm or more ST-segment elevation in V_4R are diagnostic of an inferior RVI. If right-sided chest leads are being obtained, a complete set, including V_4R through V_6R, should be for a more complete view of the right ventricle. As this section on RVI suggests, in the presence of IWMI, right ventricular leads should always be recorded as well.

The criteria for RVI are as follows:

1. IWMI
2. ST-segment elevation greater in lead III than in lead II
3. ST-segment elevation in V_1 that could extend through V_5 or V_6
4. ST-segment depression in V_2 (unless the ST-segment elevation extends through V_5 or V_6)
5. ST-segment depression in V_2 not more than half the ST-segment elevation in aVF
6. 1 mm or more of ST-segment elevation in right-sided leads (V_4R to V_6R)

The ECG in **Figure 13-79** represents an RVI. The first notable changes on the standard 12-lead ECG are the classic findings of an IWMI and ST-segment elevation in leads II, III, and aVF with reciprocal findings of ST-segment depression in leads I and aVL. An RVI is suspected because of the IWMI; lead III shows an ST-segment elevation greater than in lead II. Additional criteria for RVI are met with ST-segment elevation in V_1 and ST-segment depression in V_2 that is less than half the ST-segment elevation in aVF.

With the criteria met for RVI in the standard 12-lead ECG, the additional right-sided chest leads V_4R to V_6R are indicated. The ST-segment elevation in lead V_4R completes the diagnostic criteria for RVI. (Note: This example shows only V_4R.)

Acute Posterior Wall Myocardial Infarction

A PWMI is caused by an occlusion of the RCA. The RCA bifurcates into the posterior descending artery and right posterolateral artery in 80% to 85% of the population. The posterior descending artery comes off of the left circumflex artery in the remaining 15% to 20% of the population. The posterior descending artery is sometimes called the posterior interventricular artery, and it supplies the inferior wall, ventricular septum, and posteromedial papillary muscle. The right posterolateral artery divides into

branches that supply the posterior surface of the left ventricle.

There are no leads that specifically look at the posterior surface of the heart on a standard 12-lead ECG. By using standard 12-lead ECG technology, the diagnosis is made by looking for reciprocal changes in leads V_1 and V_2. These changes include ST-segment depression, upright T waves, and tall R waves. Normally, the QRS complex in lead V_1 has a small R wave and a deep S wave. **Figure 13-80** shows the characteristic reciprocal changes of a PWMI as seen in leads V_1 and V_2. The addition of posterior leads can record a direct representation of electrical impulses through the posterior wall.

An R wave with greater amplitude than the corresponding S wave is highly suggestive of a PWMI. In RVH, there is also the presence of an R wave greater than the corresponding S wave in lead V_1. The difference is that RVH also requires the presence of an RAD, which is not present in a PWMI.

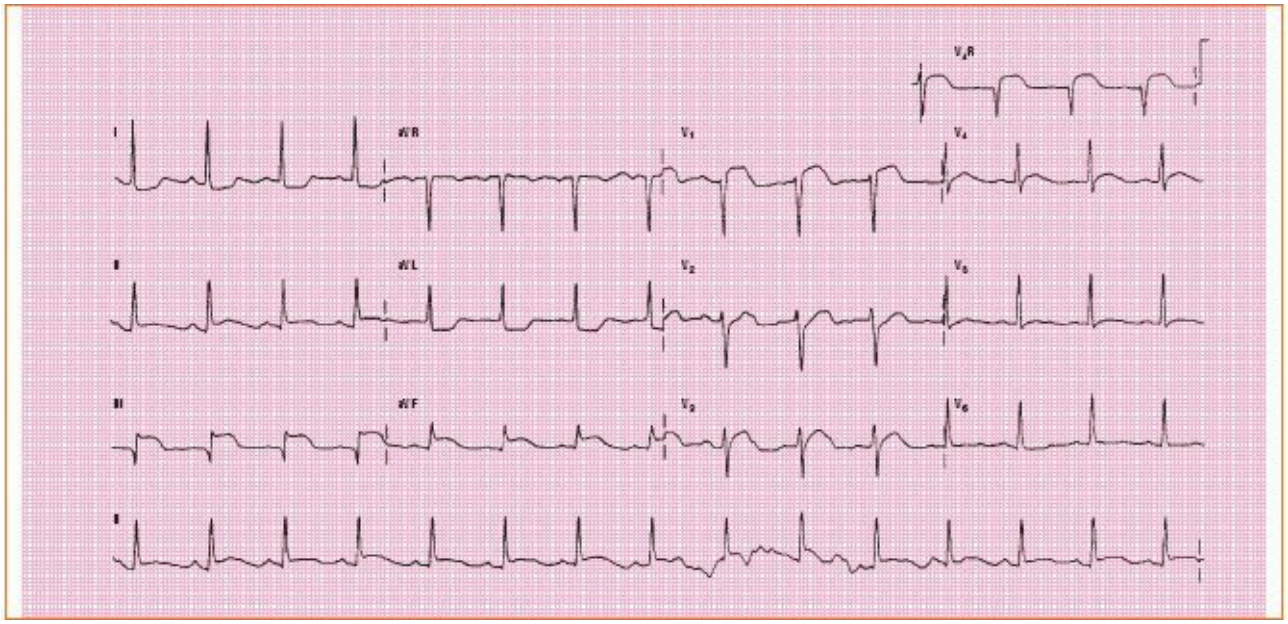


Figure 13-79 A 12-lead electrocardiogram showing a right ventricular infarction.

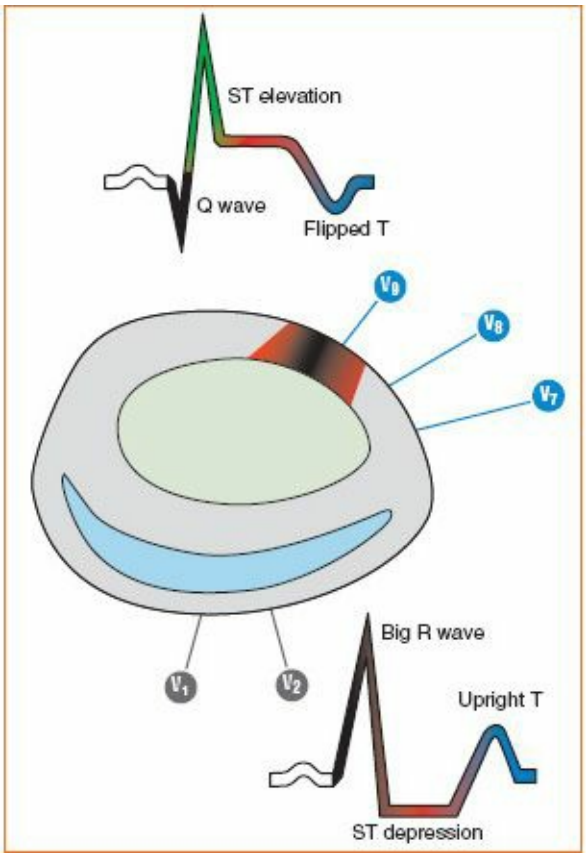


Figure 13-80 A posterior wall myocardial infarction.

Because the posterior descending artery branch of the right coronary artery also perfuses regions of the inferior wall, posterior and inferior wall MIs often occur together.

As discussed earlier, there are nonstandard or additional leads that can be used to provide a direct view of the posterior wall of the heart. When there is reciprocal evidence of a PWMI, a high index of suspicion should be maintained, and posterior leads (V_7, V_8, and V_9) should be recorded for comparison. A standard 12-lead ECG should be recorded first. Then, the additional posterior electrodes are added, the precordial leads are attached as described, and the posterior leads record and are "labeled" to avoid confusion. In the posterior leads, the electrical impulses have to travel through the larger muscle mass of the back, increasing electrical resistance. The complexes are typically positive but tend to have a lower amplitude.

Figure 13-81 and **Figure 13-82** permit comparison of the reciprocal changes of PWMI seen in leads V_1 and V_2 with those in posterior leads V_7, V_8, and V_9.

The ECG in **Figure 13-83** represents a PWMI with reciprocal changes seen on a standard 12-lead ECG. This ECG shows tall R waves in leads V_1 and V_2. There is also ST-segment depression with an upright T wave. These changes are all suggestive of a PWMI.

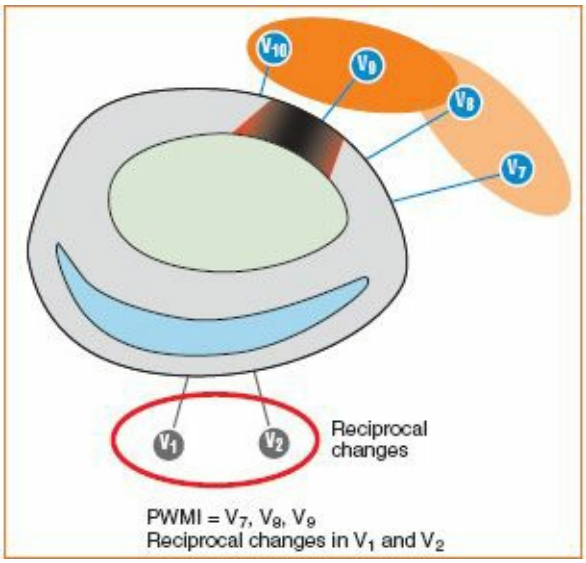


Figure 13-81 A posterior wall myocardial infarction.

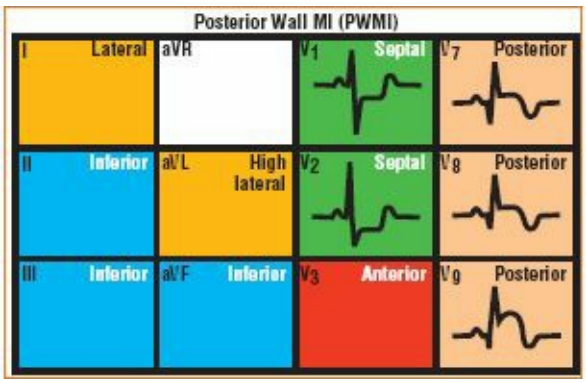


Figure 13-82 Electrocardiographic changes seen with a posterior wall myocardial infarction.

As mentioned, in the majority of the population, the coronary blood supply to the posterior wall is also shared by the inferior wall and right ventricle. This being so, patterns of infarction with changes

associated with inferoposterior and inferior-right ventricular-posterior are often seen.

Cardiomyopathy

Cardiomyopathy is a general term for diseases in which the myocardium becomes thin, flabby, dilated, or enlarged, ultimately progressing to heart failure, acute MI, or death. One variant, hypertrophic cardiomyopathy, is an autosomaldominant hereditary disease. The main feature of hypertrophic cardiomyopathy is an excessive thickening of the heart muscle (hypertrophy means to thicken or grow excessively). In addition, microscopic examination of the heart muscle shows that it is abnormal. Patients may have shortness of breath, chest pains, palpitations, or syncope; sudden cardiac death is also possible. In some patients, beta-blockers are effective treatment. Other patients require surgery or an ICD designed to deliver a shock to the heart when an arrhythmia occurs.

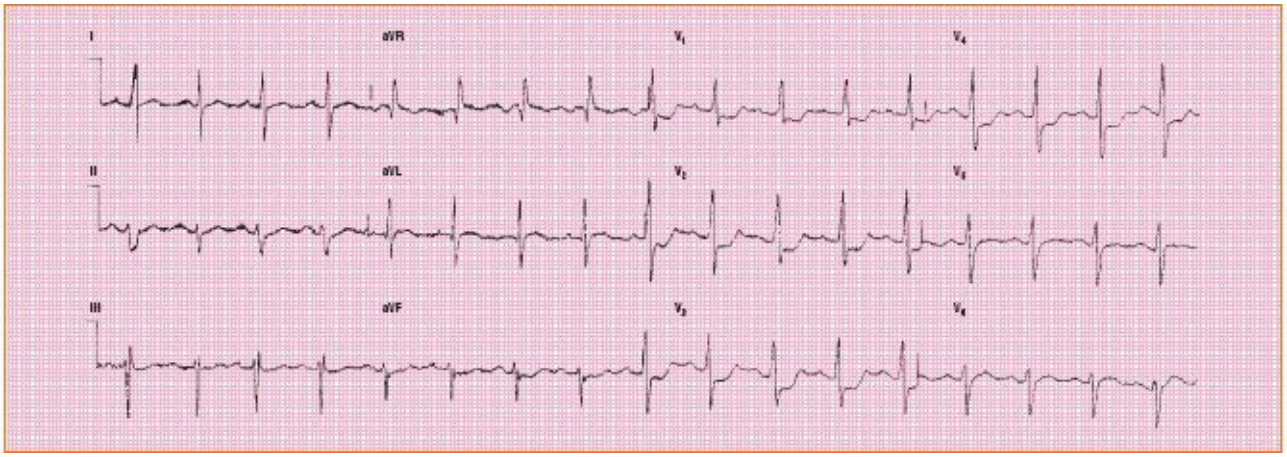


Figure 13-83 A 12-lead electrocardiogram showing a posterior wall myocardial infarction.

Signs and Symptoms

Cardiomyopathy

- Shortness of breath
- Chest pains
- Palpitations
- Syncope
- Sudden cardiac death

Congestive Heart Failure

Congestive heart failure (also known as chronic heart failure) occurs when the heart is unable, for any reason, to pump powerfully enough or fast enough to empty its chambers; as a result, blood backs up into the systemic circuit, the pulmonary circuit, or both. Although CHF may develop in situations other than acute MI, the basic principles of diagnosis and treatment are similar, whatever the precipitating factors.

More than 2 million people in the United States have CHF, and an additional 500,000 cases are diagnosed each year. Nearly half of the patients with CHF classified as severe die within 1 year of diagnosis. Severe cases are those involving acute pulmonary edema with marked limitation to physical activity; those involving cardiogenic shock are life threatening. Heart failure occurs when the heart is unable to pump a sufficient quantity of blood to meet the oxygen requirements of the body and the heart itself. Congestive heart failure can have a number of causes, such as the following:

1. Wall abnormalities (dilated and hypertrophic)
2. Valve failure (mitral, tricuspid, aortic, and pulmonic)
3. Damage as the result of infarctions
4. Conduction abnormalities

In most cases, failure begins with a major infarction involving one or more of the left ventricle walls (septum, anterior, or lateral). Major infarctions can often involve the right side and inferior wall. In any case, subsequent inadequate coronary artery oxygenation combined with cardiac damage often leads to conduction path disturbances, particularly AV nodal blocks requiring pacemaker implantation.

Another problem occurs around the areas of a major infarction. The new nerve fibers develop with an increased density that results in hyperinnervation that can lead to VT and ventricular fibrillation, requiring ICD implantation.

In cases of infarctions involving the septum, synchronization between the ventricles is lost and the ejection fraction is reduced. Patients often require biventricular pacing to restore synchrony.

Left-Sided Heart Failure

The left ventricle is most commonly damaged during an acute MI. Likewise, in chronic hypertension, the left ventricle tends to be affected by the long-term effects of having to pump against an increased afterload (constricted peripheral arteries). In both cases, the right side of the heart continues to pump relatively normally and to deliver normal volumes of blood to the pulmonary circulation. By comparison, the left side of the heart may no longer be able to pump the blood being delivered from the pulmonary vessels. As a result, blood backs up behind the left ventricle, and the pressure in the left atrium and pulmonary veins increases. As the pulmonary veins become engorged with blood, serum is forced out of the pulmonary capillaries and into the alveoli. The serum mixes with air in the alveoli to produce foam (in pulmonary edema).

When fluid occupies the alveoli, oxygenation is impaired. The patient experiences that impairment as shortness of breath (dyspnea), particularly in the recumbent position (orthopnea, as with **paroxysmal nocturnal dyspnea**). Paroxysmal nocturnal dyspnea is severe shortness of breath occurring at night after several hours of recumbency, during which fluid pools in the lungs; the person is forced to sit up to breathe. Paroxysmal nocturnal dyspnea is caused by left-sided heart failure or the decompensation of chronic obstructive pulmonary disease. If left ventricular failure is the result of chronic overload (as opposed to acute MI), the patient is likely to give a history of a week or two of paroxysmal nocturnal dyspnea. To compensate for the impairment in oxygenation, the patient's respiratory rate increases (tachypnea); even so, if the patient's condition is advanced enough, cyanosis may become evident. In some patients with pulmonary edema, especially elderly patients, Cheyne-Stokes respirations may be present.

Fluid from the pulmonary vessels also leaks into the interstitial spaces in the lungs, and increasing interstitial pressure causes narrowing of the bronchioles. Air passing through the narrowed bronchioles creates wheezing noises, whereas air bubbling through the fluid-filled alveoli produces crackles. Furthermore, the patient may cough up the edema fluid in the form of foamy, blood-tinged sputum. As the airways narrow and the lungs grow heavier from the accumulation of fluid, the work of breathing increases, which puts an even greater strain on an already floundering heart. Dyspnea and hypoxemia produce a state of panic, which induces the release of epinephrine from the adrenal glands. The heart is pushed even harder, and its oxygen demand is increased precisely when fluid in the alveoli is reducing the amount of oxygen available.

To make matters worse, the sympathetic nervous system response produces peripheral vasoconstriction: Peripheral resistance (afterload) increases, and the weakened, hypoxic heart finds itself

trying to push blood out into smaller and smaller pipes. Clinically, peripheral vasoconstriction is apparent as pallor and elevated blood pressure. The massive sympathetic discharge also produces sweating of the pale, cold skin.

It is not unusual for a patient with left-sided heart failure to become frantic from air hunger. He or she may pace or thrash about or may even be combative and struggle with the rescue team. Furthermore, hypoxemia results in inadequate oxygen supply to the brain, often manifested as confusion or disorientation. If hypoxemia is severe, cardiac arrest may follow quickly.

Signs and Symptoms of Left-Sided Heart Failure

The signs and symptoms of left-sided heart failure include extreme restlessness and agitation, confusion, severe dyspnea and tachypnea, tachycardia, elevated blood pressure, crackles and possibly wheezes, and frothy, pink sputum. Sometimes, it may be difficult to distinguish the wheezing of asthma from that of left-sided heart failure.

Signs and Symptoms

Left-Sided Heart Failure

- Extreme restlessness and agitation
- Confusion
- Severe dyspnea and tachypnea
- Tachycardia
- Elevated blood pressure
- Crackles and possibly wheezes
- Frothy, pink sputum

Transport Management

Left-Sided Heart Failure

- If the patient is conscious and is able to maintain an airway, apply CPAP.
- Intubate the patient if he or she cannot maintain the airway; deep suction the trachea with the largest suction catheter available.
- Assist the patient to breathe with a transport ventilator with the ability to provide PEEP.
- Use waveform capnography to monitor ventilation response.
- If the blood pressure is elevated, give IV nitroglycerin (Nitro-Bid IV, Tridil).
- If the blood pressure is too low, it must be elevated. Give furosemide (Lasix) or a vasopressor such as dopamine (Intropin) or dobutamine (Dobutrex).
- In case of severe valve failure, give combination therapy of low-dose nitroglycerin and dopamine or low-dose dopamine and dobutamine.
- Obtain a 12-lead ECG.
- Monitor for arrhythmias.
- Provide pain control (morphine, hydromorphone [Dilaudid], or fentanyl citrate).
- If the patient is experiencing an acute MI and a long transport is involved, give a heparin bolus, then

a heparin drip, and then an eptifibatide (Integrilin) drip.
- Monitor for signs of bleeding.

Management of Left-Sided Heart Failure

If a patient is being transported by a critical care transport team, the patient has probably experienced or is having an acute MI.

Pulmonary edema occurs as the result of reduced ejection fraction and CO of the left ventricle. Because the CO from the right ventricle is normal, pulmonary hydrostatic pressure is increased, causing fluids to cross the alveolar capillary membranes, thus filling the alveoli.

The priorities when managing left-sided heart failure are as follows:

1. Remove the fluid from the lungs.
2. Reduce afterload.
3. Increase oxygen supply to the heart.
4. Decrease myocardial oxygen demands.

From a critical care point of view, if the patient is conscious and is able to maintain an airway, apply CPAP, which provides a positive end diastolic pressure that opens the terminal bronchi, expands the alveoli sacs, and creates enough pressure to overcome the increased pulmonary hydrostatic pressure, forcing fluids back into the capillaries.

If the patient's level of consciousness is such that he or she cannot maintain the airway, endotracheal intubation is required. Once a successful intubation has been achieved, deep suctioning of the trachea is required with the largest suction catheter available. Then a transport ventilator with the ability to provide PEEP is applied. Waveform capnography should be used to monitor ventilation response.

Once the airway is controlled, the blood pressure must be managed, and the excess fluids must be removed from the body.

If the blood pressure is elevated, it should be controlled by IV nitroglycerin (Nitro-Bid IV, Tridil); IV nitroglycerin is most commonly mixed as 50 mL of nitroglycerin in 250 mL of D_5W or normal saline. Nitroglycerin is titrated to control blood pressure. Nitroglycerin given IV is preferential to sublingual or paste because it uniformly controls vasodilation and is not subject to the see-saw problems associated with repeated sublingual application. If IV nitroglycerin is used, all other forms of nitroglycerin must be discontinued. IV nitroglycerin is usually started at 10 μg and increased in 10-μg increments until the desired blood pressure is obtained.

If the blood pressure is too low, it must be elevated to provide adequate perfusion pressure. Low blood pressure is a problem in mitral valve failure. In this case, a vasopressor such as dopamine or dobutamine will be required to maintain adequate pressure. In some cases, patients with severe valve failure may be treated with a combination of low-dose nitroglycerin (2–3 μg) to reduce afterload and dopamine (Intropin) as the pressor agent or low-dose dopamine (2–3 μg/kg) to reduce afterload and dobutamine (Dobutrex) as the pressor agent. All of these medications must be administered via an IV pump.

Furosemide (Lasix) should be given to eliminate excess fluids from the system. The dose should be based on local protocol. Furosemide is usually given as 0.5 to 1 mg/kg, not to exceed 80 mg. Furosemide takes 20 minutes to reach full effect, and the systolic blood pressure must be greater than 100 mm Hg.

All patients with CHF or pulmonary edema should have a 12-lead ECG obtained and be monitored for arrhythmias.

For pain control, morphine can be used when blood pressure is not an issue. If the patient is

hypotensive, hydromorphone (Dilaudid) and fentanyl citrate are better choices because they do not lower blood pressure.

If the patient is having an acute MI and a long transport is involved, a heparin bolus, then a heparin drip, and an eptifibatide (Integrilin) drip may be indicated. First, contraindications to anticoagulant or antiplatelet treatment (such as bleeding or recent surgery) must be ruled out. Blood samples should be obtained if not previously done: a red tube (blood banking) and a blue tube (coagulation studies). An IV bolus of heparin is administered per direction of the physician (usually 4,000–5,000 U) and is followed with an IV drip via an infusion pump (usually 700–1,000 U/h). If needed, an eptifibatide drip is administered via an infusion pump per a physician's direction. Heparin is an anticoagulant, and eptifibatide is an antiplatelet drug. Both have short half-lives, less than 2.5 hours. During transport, frequent observation of the patient is needed for signs of major bleeding. Minor bleeding from around the nasal cannula or teeth and mouth is common and no cause for alarm. If any evidence of major bleeding is noted, the drug must be discontinued.

Right-Sided Heart Failure

Right-sided heart failure most commonly occurs as a result of left-sided heart failure. As blood backs up from the left side of the heart into the lungs, the right side has to work increasingly harder to pump blood into the engorged pulmonary vessels. Eventually, the right side of the heart is unable to keep up with the increased workload, and it, too, fails. Right-sided heart failure may also occur as a result of pulmonary embolism or long-standing chronic obstructive pulmonary disease, especially chronic bronchitis.

Signs and Symptoms

Right-Sided Heart Failure

- Edema

Transport Management

Right-Sided Heart Failure

- Provide supportive care.
- Place the patient in a comfortable position, preferably a semi-Fowler's position.
- Treat any signs of associated left-sided heart failure.

When right-sided heart failure occurs, blood backs up behind the right ventricle and increases the pressure in the systemic veins, causing them to become engorged. Distention can be seen in the veins visible on the surface of the body, such as the external jugular veins. Over time, as the pressure within the systemic veins increases, serum is forced out of the veins and into the surrounding tissues, producing edema. Edema is most likely to be visible in dependent parts of the body, such as the feet in a person who is sitting or standing or the lower back in a bedridden patient. Edema is also present in parts of the body that are *not* visible; a painful liver that is easily palpable in the right upper quadrant, for example, signals engorgement and swelling within that organ (hepatomegaly).

The development of right-sided heart failure can actually improve left-sided heart failure because the failing right side of the heart can no longer pump as much blood into the lungs. The decrease in output from the right side, in essence, amounts to a decrease in preload for the left side of the heart and may

lessen pulmonary congestion.

Right-sided heart failure, by itself, is seldom a life-threatening emergency. Usually it develops gradually over days to weeks; likewise, it requires days to weeks to reverse the process by slowly ridding the body of excess salt and water. Treatment in the field of a patient with right-sided heart failure, therefore, is simply to make the patient comfortable, preferably in the semi-Fowler's position. Monitoring is always indicated in any patient with significant cardiac disease. If signs of associated left-sided heart failure are present, they should be treated, as outlined in the previous section.

Pulmonary Edema

The intervention CPAP (discussed in Chapter 6) is most commonly used to treat pulmonary edema while the patient is conscious and able to maintain an airway. Increasing the expiratory pressure forces open the terminal bronchi and the alveoli and increases the pressure within the alveoli to greater than the pulmonary hydrostatic pressure to force fluid within the alveoli back into the capillaries. For transport purposes, a CPAP unit with adjustable PEEP is best. Once CPAP is initiated, treatment with nitroglycerin (Nitro-Bid IV, Tridil), to reduce blood pressure, and furosemide (Lasix), to remove excess fluids, should be undertaken. Initial PEEP settings should be higher (5–10 cm water) but may be reduced after the furosemide and nitroglycerin have taken effect.

Transport Management

Pulmonary Edema

- Use CPAP with adjustable PEEP if the patient is conscious and able to maintain the airway.
- Give nitroglycerin (Nitro-Bid IV, Tridil) to reduce blood pressure.
- Give furosemide (Lasix) to remove excess fluids.

Intubation for patients with acute respiratory distress resulting from asthma or chronic obstructive pulmonary disease should be seen as a last choice. Bilevel positive pressure is an excellent option for this population. Bilevel positive airway pressure is also appropriate for patients with pulmonary edema. Also, most bilevel positive airway pressure machines are large and rarely used during transport. They offer patient advantages, such as humidification, heated air, and exhalation pressure relief. They can be hooked up to oxygen or medical air. For short transports, CPAP may be used in place of bilevel positive airway pressure.

Cardiac Devices

Although ECG is used routinely in the field for diagnostic purposes, **cardiac electrophysiology** is performed in the hospital setting (not during transport). This cardiac specialty involves evaluation and management of cardiac rhythm disturbances. The cardiac electrophysiologist specializes in the diagnosis and treatment of life-threatening ventricular tachyarrhythmias, as well as supraventricular tachyarrhythmias (SVTs, including atrial fibrillation), bradycardias, AV blocks, and syncope of cardiac origin.

In the hospital setting, an electrophysiologist may conduct an electrophysiology study to assess electrical activity of the heart by stimulating and recording that activity from multiple catheters at several sites in the heart. The study records and maps electrical signals from the heart to determine the location of a heart block (AV node vs bundle of His), the origin of tachycardia (supraventricular or ventricular), and

other important parameters, such as the assessment of drugs and devices for the treatment of arrhythmias. These clinical data assist in the following:

- Determining whether the patient's symptoms are related to a tachyarrhythmia
- Assessing the risk for lethal tachyarrhythmias
- Developing and testing appropriate therapies, based on the induced tachyarrhythmia and its mechanism

The CCTP's role during transport is to anticipate the rhythm disturbance for which the patient is being transferred and to correct it per ACLS protocols.

Special Populations

Cardiovascular complications can be exacerbated by pregnancy. It is not uncommon for CCTPs to transport a pregnant patient to a tertiary center for aortic or mitral valve issues, coronary artery disease, thromboembolic events (including pulmonary embolism), or (although rare) aortic dissection.

Therapeutic Options for Patients With Arrhythmias

Compared with a decade ago, a wide array of safe and effective therapies are available for patients with arrhythmias, and more are appearing rapidly. The primary in-hospital arrhythmia therapies are catheter ablation, pharmacologic treatment, and implantable devices, such as pacemakers and ICDs.

Radiofrequency Catheter Ablation

Ablation (destruction) of discrete areas in the heart may reduce or eliminate susceptibility to arrhythmias in certain patients. Radiofrequency catheter ablation is highly effective for WPW syndrome and other SVTs, including atrial flutter, atrial tachycardia, AV node re-entrant tachycardia **Figure 13-84**, and some VTs. Improvements in mapping techniques have made a wider variety of arrhythmias eligible for ablation.

If an ablation corrects the arrhythmia, the patient will not require critical care transport, but if an ablation is unsuccessful, the patient may need critical care transport to a hospital with cardiac surgery capability.

Radiofrequency catheter ablation has completely changed the approach to the long-term management of patients with recurrent SVTs. Electrophysiologists are now able to convert arrhythmias during outpatient procedures. Ablative therapy is effective 90% to 98% of the time, frequently offers a permanent cure, and has a very low incidence of complications.

For many patients with SVT, ablation eliminates the need for long-term medical therapy, which is beneficial for several reasons. Long-term antiarrhythmic therapy can result in drug-induced adverse events and reduce quality of life. These problems often result in inadequate patient adherence to the medication regimen, limiting drug effectiveness. Patients who do not take prescribed medications or are reluctant to undergo long-term drug therapy may be candidates for catheter ablation. Drugs may also be used to interfere with electrical conduction or impulse formation at the AV node, accessory pathway, or origin of a focal tachycardia. Examples include digoxin (Lanoxicaps, Lanoxin), calcium antagonists, beta-blockers, class IA agents (procainamide [Procanbid, Pronestyl, Pronestyl-SR, Procan SR], disopyramide [Norpace, Norpace CR], quinidine gluconate, quinidine sulfate), class IC agents (propafenone [Rythmol], flecainide [Tambocor]), and class III agents (amiodarone [Cordarone], sotalol [Betapace, Betapace AF]). Long-term digoxin therapy is contraindicated in patients with WPW syndrome and patients with the capability of rapid **antegrade conduction** over the accessory pathway.

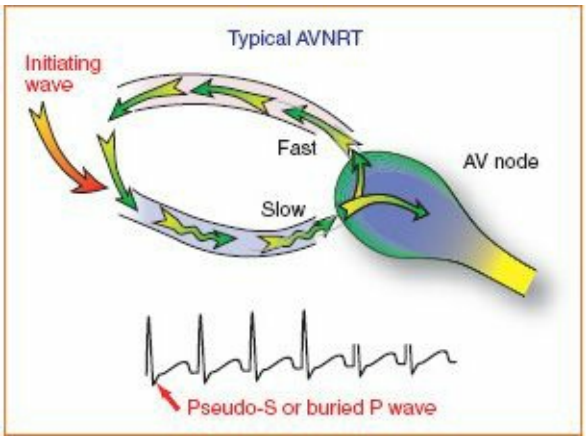


Figure 13-84 Atrioventricular (AV) node re-entrant tachycardia.

Cardiac Pharmacology

Cardiovascular drugs improve cardiac performance by altering the physiologic and biochemical events that control cardiac function. They may change heart rate, force of contraction, speed of conduction, excitability of cardiac tissue, phases of the action potential, and blood flow. They are used to halt or slow disease processes, augment heart rate reserves, control arrhythmias, and manage an overworked heart. **Table 13-10** summarizes the effects of cardiac drugs.

Implanted Pacemakers

An implanted pacemaker provides impulse generation in a diseased heart whose own impulses may be:

- Intermittent
- Irregular
- Occurring at an inappropriate rate for the patient's metabolic demand
- Absent

A block can occur at any point within the SA node, AV node, bundle of His, or distal conduction system.

Modern pacemakers support heart function in several ways. They provide effective and consistent cardiac depolarization, prevent unnecessary pacing by sensing cardiac activity, increase the rate to meet increased metabolic demand, and provide information about how the patient's heart and the implanted pacemaker are functioning.

Implantable Cardioverter-Defibrillators

New guidelines for **implantable cardioverter-defibrillator (ICD)** therapy reflect published data confirming the benefits of ICD therapy over antiarrhythmic drugs for the treatment of life-threatening ventricular tachyarrhythmias. ICDs have been proven to prevent sudden cardiac death and to reduce overall mortality considerably compared with antiarrhythmic drugs. Studies have also shown that ICDs are up to 99% effective in terminating life-threatening ventricular tachyarrhythmias.

Pacemakers

The first implantable pacemakers, developed in 1960, were asynchronous; they paced without regard to the heart's intrinsic action. The NBG (North American Society of Pacing and Electrophysiology [NASPE]/British Pacing and Electrophysiology Group [BPEG] Generic) codes appear later in this chapter in **Table 13-11**, which explains similar abbreviations related to pacemaker (also called pacer) function.

Single-chamber demand pacemakers appeared in the late 1960s. In 1979, the first **dual-chamber**

pacemaker was introduced. The first single-chamber, rate-responsive pacemaker came into use in 1985. Today, dual-chamber pacemakers use rate-responsive pacing to mimic the heart's rate response and meet metabolic needs, most recently using a combination of sensors to accomplish this.

In a normally functioning heart, the SA node stimulates depolarization of the ventricles. When the SA node becomes nonfunctional, arrhythmias affecting the life and functionality of the patient can occur. At this point, an artificial pacemaker must be installed. The primary indications for pacing are as follows:

1. Symptomatic sinus bradycardia refractory to atropine
2. Heart blocks (Mobitz II and third-degree heart block)
3. Sinus node disease (sick sinus)
4. Reflex syncope
5. Cardiac resynchronization
6. Chronotropic incompetence
7. Overdrive pacing for tachyarrhythmias

There are four major categories of cardiac pacemakers:

1. Transcutaneous
2. Implantable
3. Transthoracic or epicardial
4. Transvenous (external)

The most fundamental pacer is the transcutaneous pacemaker. It is the one most commonly used as an initial emergency pacer. Multifunction pads are attached to the patient's skin in the anterior-posterior positions over the patient's heart. The electrical pacing impulse is transferred to the heart through the skin and thoracic cavity. Although this is the quickest and most convenient method of pacing, it requires the most energy and is the least reliable owing to the quality of the skin-to-pad contact and the thoracic impedance that must be overcome. It is generally used as a temporary measure until a transvenous pacemaker can be placed. This pacemaker is the one most likely to be used by CCTPs if a patient requires emergency pacing en route.

Implantable pacemakers consist of a battery-operated pulse-generating module with attached pacer leads. An interventional cardiologist implants the lead or leads in the myocardial wall as needed. The pulse-generating module is then surgically implanted in the body, usually in a sac created between the pectoral muscle and the cutaneous layer of the skin. In some cases, the pacemaker electrode is screwed into the myocardial wall, and the pulse generator is installed below the xiphoid process.

TABLE 13-10 Cardiovascular Drug Actions and Uses

Drug	Action	Primary Uses
Drugs That Control Myocardial Ischemia		
Oxygen	Improves oxygen supply to tissue	Ischemic chest pain and cardiac arrest
Nitroglycerin (Nitro-Bid IV, Tridil)	Dilates blood vessels and improves coronary blood flow; reduces cardiac workload	Ischemic chest pain and acute MI
	Reduces cardiac workload and increases pain	

Morphine sulfate	threshold	Ischemic chest pain
Drugs That Control Heart Rhythm		
Lidocaine (Xylocaine)	Blocks the sodium channel and decreases excitability; suppresses ventricular ectopic activity	Persistent VT or fibrillation
Procainamide (Procanbid, Pronestyl, Pronestyl-SR, Procan SR)	Blocks the sodium channel and decreases conductivity; suppresses ventricular ectopic activity	Recommended when lidocaine treatment fails or is contraindicated
Beta-adrenergic blockers Propranolol (Inderal) Atenolol (Tenormin) Esmolol (Brevibloc)	Block the sympathetic response of the AV and SA nodes; reduce sinus activity and slow conduction	Atrial fibrillation and reduce the incidence of ventricular fibrillation after an MI
Atropine sulfate	Blocks the parasympathetic response of the SA node; increases heart rate and AV conduction	Symptomatic bradycardia, junctional escape rhythms, and second- or third-degree AV block, cardiac arrest
Isoproterenol (Isuprel)	Stimulates the heart's sympathetic response; increases heart rate and CO	Significant bradycardia refractory to other therapies; also denervated post–heart transplantation bradycardia
Verapamil (Calan, Covera, Isoptin, Verelan)	Blocks the calcium channel; slows sinus activity and suppresses AV conduction; slows heart rate, decreases blood pressure, decreases myocardial contractility; ultimately decreases myocardial oxygen demand; usually used as second-line drug after beta-blockers	Ischemic heart disease, LVH, lung disease, and hypertension
Diltiazem (Cardizem, Dilacor, Tiazac)	Blocks the calcium channel; slows sinus activity and suppresses AV conduction; slows heart rate, decreases blood pressure, decreases myocardial contractility; ultimately decreases myocardial oxygen demand; usually used as second-line drug after beta-blockers	Ischemic heart disease and LVH; atrial rhythms
Adenosine (Adenocard)	Reduces the sinus rate, slows AV conduction, and interrupts re-entrant pathways	Wide complex tachycardia, narrow complex tachycardia,

		conversion of SVT to normal sinus rhythm
Magnesium sulfate	Not completely understood; may increase systemic vasodilation and reduce myocardial oxygen demand	Polymorphic VT, pulseless VT, and ventricular fibrillation
Amiodarone (Cordarone)	Blocks the potassium channel and extends the refractory period; slows conduction	CHF and WPW syndrome
Sotalol (Betapace, Betapace AF)	Blocks the potassium channel and extends the refractory period; slows conduction	Ischemic heart disease
Drugs That Improve CO and Blood Pressure		
Epinephrine (Adrenalin)	Stimulates sympathetic response and increases heart rate and contractility	Cardiac arrest; also used in infusions to increase blood pressure
Norepinephrine (Levophed)	Constricts peripheral vessels and increases blood pressure	Cardiogenic shock and severe hypotension
Dopamine (Intropin)	Dilates blood vessels; increases heart rate and contractility at higher doses	Cardiogenic shock and hypotension
Dobutamine (Dobutrex)	Stimulates sympathetic response; exerts a positive inotropic effect	Refractory congestive heart failure and cardiogenic shock
Inamrinone (Inocor)	Dilates blood vessels; exerts a positive inotropic effect	Severe congestive heart failure that has not responded to other inotropic agents
Digitalis	Slows AV nodal conduction; exerts a positive inotropic effect; highly toxic	To control ventricular response rate in chronic atrial fibrillation
Drugs That Provide Diuresis or Prevent or Dissolve Blood Clots		
Diuretics Furosemide (Lasix) Bumetanide (Bumex)	Act on kidneys to increase urine output	Hypertension and pulmonary edema
Fibrinolytic agents TPA Streptokinase (Streptase) Tenecteplase (TNKase)	Dissolve blood clots and restore blood flow	Stop or limit MI

Reteplase (Retavase) Anistreplase (Eminase) Alteplase		
Anticoagulants Heparin (Liquaemin) Warfarin (Coumadin) Enoxaparin (Lovenox) Aspirin	Inhibit clot formation; heparin decreases thrombin and fibrin production; aspirin inhibits platelet aggregation	Prevent thromboembolism in patients at risk
Glycoprotein IIb/IIIa inhibitors Abciximab (ReoPro) Eptifibatide (Integrilin) Tirofiban (Aggrastat)	Inhibit platelet aggregation	ACS without ST-segment elevation

Implantable pacers can be unipolar or bipolar and come in a variety of types:

1. Atrial
2. Ventricular
3. Atrial and ventricular
4. Biventricular
5. Overdrive pacemakers for tachyarrhythmias

Pacemakers can operate on demand, or the patient's heart can be fully paced. A demand pacer intervenes when the patient's normal rhythm fails. In this case, the ECG will show the patient's normal rhythm with periodic pacer intervention. An ECG for a patient with a fully paced heart will show only the paced rhythm.

Most pacers today are atrial and ventricular, and many have an ICD built into them. Biventricular pacers are used in patients with ventricular asynchrony to restore synchrony and improve ejection fraction and CO. Pacer circuitry is less complicated than defibrillator circuitry and requires less power. Statistically, pacer circuits show a lower incidence of failure than ICDs. The most common sources of failure are the following:

1. Battery failure
2. Lead wire failure or detachment

Less common sources of failure are as follows:

1. Failure of the pulse-generator circuitry
2. Dislodgment from the myocardial wall

A third class of pacers is transthoracic or epicardial pacers. This type of pacing is done only by interventional cardiologists or cardiac surgeons and involves placing pacing electrodes directly into the heart through the thoracic cavity. This type of pacing is limited to the hospital and would only be encountered by the CCTP if called to move a patient post-operatively. Wires can be attached to the atria

and/or ventricles and are attached through a connector cable to the same type of temporary pulse generator used for transvenous pacing.

Finally, many patients are transported between facilities with transvenous pacemakers with an external pulse generator. The pacemaker lead wire is placed in the heart by an interventional cardiologist or an emergency department physician. This type of pacemaker is likely to be encountered by CCTPs performing interfacility transports and is discussed in more detail next.

Transvenous Pacemakers

Transvenous pacemakers are placed in a patient as a temporary method of pacing until the pacing problem is resolved or until a permanent pacemaker is implanted. The transvenous pacemaker is much move reliable than transcutaneous pacing. The pacer lead has direct myocardial contact, is located precisely in the most appropriate spot, and has an external pulse generator that allows direct control of the impulse parameters. The lead wires are placed by an interventional cardiologist or an emergency department physician, and the battery-operated pulse generator is externally connected to the pacing leads, as shown in **Figure 13-85**.

The external pulse generator provides the following controls:

1. Pulse rate
2. Current amplitude
3. Sensitivity
4. Automatic or manual mode

Figure 13-85 A temporary transvenous pacemaker.

The lead wires are externally connected to the pulse generator. Most transports require no adjustments; however, all of the previously mentioned controls can be changed as required by the patient's condition.

Generally, transvenous pacers require little attention and are quite reliable. It is recommended that CCTPs become familiar with the type of transvenous pacemaker used by local hospitals or that they get a tutorial on troubleshooting the device. Primary sources of problems are battery failure and lead disconnection at the generator. On long trips, having an additional battery and knowing how to install it are helpful. Before leaving the transferring facility, the pacer rate, capture current, and sensitivity settings should be noted and mechanical capture confirmed by taking a pulse. On occasion, a demand transvenous pacer may confuse peaked T waves or large P waves for the R wave and will not pace. In this case, the sensitivity should be lowered and mechanical capture should be confirmed by taking a pulse. If the pacer is in the automatic mode and capture is not happening, the manual mode should be used and the current (milliamps) adjusted until capture is achieved. It is important to note how the lead wires are attached so that they can be reattached if they become loosened during transport.

Pacing Fundamentals

The Pacing Impulse

Electricity is the flow of electrons along a conductive medium. A **pacing impulse** has current, voltage, and impedance. **Current** is the movement of electrons through an electrical circuit over time, measured in amperes. **Voltage** is the force that causes current to flow, measured in volts. Voltage in a pacing system, referred to as amplitude, reflects the strength or intensity of a pacing pulse.

Resistance to current, called **impedance**, is the sum of all factors that resist the flow of current along the conduction pathway, measured in ohms. One ampere is produced by 1 volt, acting through a resistance of 1 ohm. The main sources of resistance in a pacing circuit are the lead conductor, the electrode, and the concentration of electrically charged ions at the electrode-tissue interface (polarization). Electrode resistance improves pacing efficiency; lead conductor resistance and polarization do not. Review of the following definitions and formulas will help in understanding the basic concept of electricity as it pertains to pacing.

Ohm's law is an expression of the relationship between current (I), voltage (V), and resistance (R). Ohm's law states the following: Current = Volts/Resistance. In other words, I = V/R. Consequently, V = I × R and R = V/I. With these equations, volts, current, and resistance may be calculated if the other parameters are known. If the voltage drops by half, the current flow does also. Doubling impedance will also cut current flow in half. Voltage and impedance are important determinants of battery longevity.

A **joule** is a measurement of energy. Energy = Volts × Current × Time. Energy is then expressed in joules or microjoules, as appropriate for the device.

Pacing Circuit

A pacing system forms an electrical circuit in the patient's body by combining with body tissue and fluid. A **pacing circuit** consists of a power source, lead, cathode, anode, and body tissue, which form a conduction pathway along which electricity flows.

The pacemaker battery is the power source that generates electrical impulses. The lead conductor wire carries the impulses to the tissue. The **cathode** is an electrode with a negative charge that delivers the impulse to the myocardium. The impulses return through the **anode**, an electrode with a positive charge, after stimulating the heart. Body tissue and fluids between the anode and cathode are also part of the conduction pathway. Passage of the pacemaker's electrical impulse between the cathode and anode—through cardiac tissue and body fluids—is the event in the pacing circuit that stimulates cardiac depolarization. In a **bipolar system**, body tissue is part of the circuit only in the sense that it affects impedance at the electrode-tissue interface. In a **unipolar system**, contact with body tissue is essential to ground the **implantable pulse generator (IPG)**.

During pacing, an electrical impulse begins in the pacemaker battery, travels along the lead to the cathode, stimulates the heart, and then returns through body tissues to the anode to complete the pacing circuit **Figure 13-86**.

Pacemaker Components and Functions

Pacemakers stimulate cardiac depolarization, sense intrinsic cardiac activity, respond to metabolic need, and store diagnostic information. All pacemakers can provide fixed-rate pacing, in which the heart is paced at a predetermined rate. Most also provide rate-a daptive (rate-responsive) pacing, increasing and decreasing the pacing rate in response to input from rate sensors. This feature prevents the pacemaker from competing with normal cardiac function. In addition, modern pacemakers collect and store information about the patient's heart and the implanted pacemaker. This information allows clinicians to monitor pacing therapy, optimize programmed settings, and check battery and lead status. A basic pacing

system consists of the following:

- An IPG that contains a power source—the battery that generates the impulse—and sensing, timing, and output circuitry that controls pacemaker operation
- Leads—one or two insulated wires that deliver electrical impulses from the pulse generator to the heart and return electrical signals from the heart to the pulse generator
- Electrodes—conductors at the ends of the leads that deliver impulses to the heart

Most IPGs have a telemetry coil for sending and receiving programming instructions and receiving diagnostic data. Many have sensors that measure indicators of exertion and use the results to change heart rate.

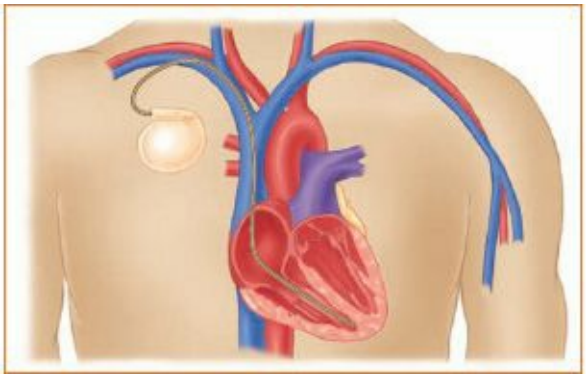


Figure 13-86 The pacing circuit. The electrical impulse travels along the lead, stimulates the heart, and returns through body tissues.

Leads

Lead insulation prevents contact between the conductor wire and body tissue, ensuring that stimulation occurs only at the tip of the electrode. About 95% of pacing leads are **endocardial (transvenous) leads**, threaded through veins—usually subclavian or cephalic—to the right atrium or ventricle **Figure 13-87**. Placement involves an introducer (hollow tube) and a stylet (stiff wire). The clinician uses imaging, usually fluoroscopy, to guide insertion and final placement. **Epicardial leads**, which attach to the external surface of the heart with sutures or another fixation device, account for the other 5%. Because epicardial placement requires a thoracotomy (chest incision), epicardial leads are desirable only when endocardial placement is not an option.

A pacing lead may be unipolar or bipolar. A **unipolar lead** has one conductor wire connecting to a single electrode. The cathode is in contact with the heart, and the IPG housing is the anode. A pacing pulse travels from the IPG to the tip of the electrode (cathode) to stimulate the heart and returns to the IPG housing through chest tissues to complete the pacing circuit. The current flows through a substantial part of the chest and forms a large current loop; the resulting pacing spike is easily visible on an ECG.

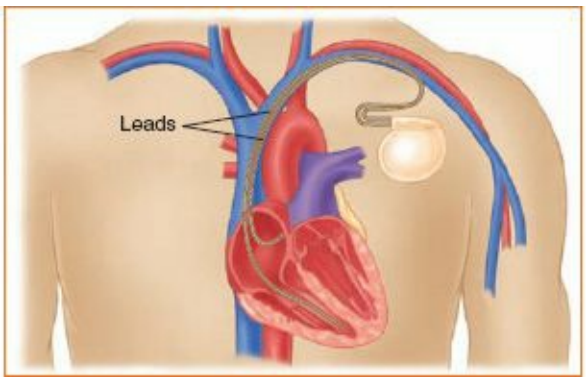


Figure 13-87 Endocardial (transvenous) pacing lead placement.

A **bipolar lead** has two conductor wires; both the anode and cathode are in contact with the heart. The anode, the ring electrode located 2 to 3 cm above the cathode, has a separate lead connecting it to the IPG. The pacing pulse travels from the IPG to the cathode and then through tissue to the ring electrode. It returns to the IPG by way of the second conductor wire. Because the current loop is small, passing through only a limited area of cardiac tissue, the pacing spike on the ECG is also small.

Electrodes

A cathode, an electrode that is in contact with heart tissue, is negatively charged when electrical current is flowing. The anode is the electrode that receives the electrical impulse after depolarization of cardiac tissue. It is positively charged when electrical current is flowing.

Small electrodes with a porous surface increase electrode resistance and reduce the effects of polarization. The porosity significantly increases the electrode surface area without increasing the radius and, thus, reduces the wasteful effects of polarization. The porous surface also promotes tissue ingrowth and improves sensing by providing a larger electrode-tissue contact area.

Steroid Elution

There are two types of transvenous pacemaker leads:

1. Passive fixation
2. Active fixation

The active fixation leads have higher stimulation than the passive fixation leads; however, they are preferred for the following reasons:

1. Passive fixation leads involve atrial appendage placement, which is difficult in patients with prior cardiac surgery
2. Active fixation leads allow for more placement areas
3. Active fixation leads reduce the incidence of dislodgement

To make the active fixation more beneficial, a steroideluting lead was developed. The steroid reservoir in the tip of the lead contained dexamethasone sodium phosphate. The inclusion of the steroid-eluting reservoir in the active fixation lead improved stimulation thresholds and was found to extend pulse-generator longevity.

Steroid elution reduces inflammation and the growth of fibrous tissue at the electrode site. Both inflammation and fibrosis reduce electrical excitability of cardiac tissue. Because steroids inhibit inflammation and fibrosis, steroid presence helps maintain a better electrode-tissue interface.

Steroid-eluting electrodes have a silicone plug that contains a small dose (<1 mg) of steroid. Body fluids seep into the electrode and gradually dissolve the steroid, which then flows in the body fluids to the electrode-tissue interface. Initially, the rate of steroid elution is high, but the rate decreases over time. The steroid typically lasts for several years.

Single-Chamber vs Dual-Chamber Systems

A single-chamber pacing system uses one lead that may be placed in the right atrium or right ventricle **Figure 13-88**. A dual-chamber pacing system uses two leads, one in each of these chambers **Figure 13-**

89. The dual-chamber approach compensates for two potential limitations of single-lead systems: the inability to coordinate atrial-ventricular timing (AV synchrony) and the lack of ventricular backup pacing in the absence of AV conduction.

NBG Codes

Developed by a joint effort of North American and British electrophysiology groups, the **NBG code** (NASPE/BPEG Generic) provides a succinct way to describe pacemaker functions and capabilities. These terms may be encountered in patient documentation; a general familiarity with them is very helpful in determining appropriate pacemaker function. **Table 13-11** lists the codes and their meanings.

The first letter in the NBG code represents the chamber where the pacing occurs. The options for where pacing can occur are the atrium, ventricle, both, none, or single (which can signify the atrium or ventricle). The second letter in the NBG code represents the chamber where the pacing is sensed. The options in this category are the same as those in the first category.

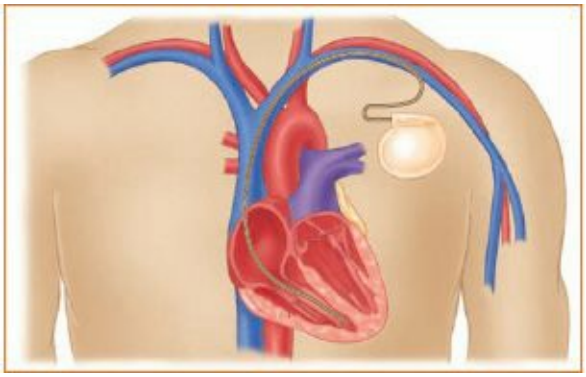


Figure 13-88 A single-chamber pacing system.

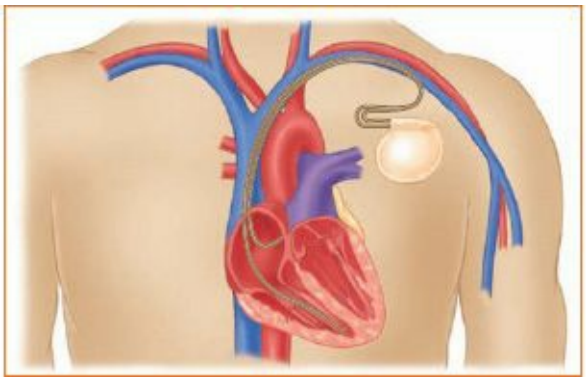


Figure 13-89 A dual-chamber pacing system.

TABLE 13-11 NBG Codes

I Chamber Paced	II Chamber Sensed	III Response to Sensing	IV Programmable Functions; Rate Modulation[a]	V Antitachyarrhythmia Functions
V: Ventricle	V: Ventricle	T: Triggered	P: Simple programmable[c]	P: Pace
A: Atrium	A: Atrium	I: Inhibited	M: Multiprogrammable[d]	S: Shock
D: Dual (A + V)	D: Dual (A + V)	D: Dual[b]	C: Communicating[e]	D: Dual[f]
O: None	O: None	O: None	R: Rate modulating	O: None
S: Single (A or V)	S: Single (A or V)		O: None	

[a] This sequence is hierarchical; it is assumed that if a pacemaker has rate modulation capabilities (R), it

also can communicate (C).

[b] Inhibited and triggered. In single-chamber mode, *triggered* means initiating a pacing pulse immediately after sensing an intrinsic event. In dualchamber mode, the term means that a sensed atrial event will initiate an AV delay.

[c] Rate and/or output.

[d] Rate, output, sensitivity, and so forth.

[e] Can send and receive information to and from the programmer.

[f] Pace and shock available.

The third letter indicates how the pacemaker responds to sensing. Options in this category include triggered, inhibited, dual, and none. The I setting, or inhibited response, indicates that the pacemaker is set to fire only under certain conditions. For example, the pacemaker may fire only when the patient's heart rate falls below a certain rate. In other words, when the pacemaker senses an impulse from the patient, it inhibits or does not fire its own impulse. The T setting, or triggered response, indicates that the pacemaker, after sensing an impulse from the patient, triggers a pacemaker impulse. D, for the dual setting, indicates that the pacemaker can perform in an inhibited and a triggered manner, as needed. The O setting, or no response, may be used when a technician wants to ignore sensed beats from the patient and fire the pacemaker regardless of any underlying patient rhythm.

The fourth letter represents the pacemaker's programmability and rate-modulation capability, which is simply a representation of how much programming can be done externally. Pacemakers may have many external programming options (M) or just a few (P). C, or communicating, indicates that the pacemaker can transmit data, as with telemetry. R, or rate modulation, means that the pacemaker can adjust its rate depending on the body's needs.

The fifth letter represents antitachyarrhythmia functions. P, or pacing, indicates that a tachyarrhythmia is converted by pacing. S, or shock, indicates that a tachyarrhythmia is converted by defibrillation administered by the pacemaker. D, or dual, indicates that the pacemaker can pace and administer a shock to address a tachyarrhythmia.

An example of an NBG code is a VVI pacemaker, in which pacing occurs in the ventricle, sensing occurs in the ventricle, and the response setting is inhibited. In day-to-day practice, only the first three letters of the NBG code are typically used.

Pacemaker Troubleshooting

Pulse-generator failure is a rare occurrence (0.46%, or 1.3 malfunctions per 1,000 persons annually). Pacemaker system malfunction occurs occasionally. The primary areas of failure are the following:

1. Battery failure
2. Problems at the lead electrode-tissue interface
3. Coiling or damage to the lead-generator interface
4. Insulation failure of the lead wires
5. Dislodgement of the lead at the implantation site (very rare)
6. Pacemaker programming
7. Pacemaker pocket stimulation (muscle tissue in the vicinity of the implanted pacemaker is stimulated by the pacer itself; more common in unipolar pacers than in other types)
8. Diaphragmatic stimulation
9. Electromagnetic interference (cell phones and other personal electronic devices)

Electromagnetic interference is the collective effect of high-frequency signals from environmental sources that produce electromagnetic fields in the frequency range of 50 to 60 Hz. Electromagnetic interference enters a pacemaker by conduction if the patient is in direct contact with the source or by way of radiation if the patient is in an electromagnetic field with the pacemaker lead acting as an antenna. Exposure to electromagnetic interference can affect pacers in various ways, all of which prevent it from functioning appropriately.

Table 13-12 details the four general types of in-hospital solutions available to correct abnormal pacing behavior; however, these cannot be done during transport except with temporary external pulse generators.

During transport, CCTPs must be aware of the signs of pacemaker failure. The cardiac monitor will show failure to capture, and a 12-lead ECG is helpful, but even more important is patient assessment. First, the CCTP should confirm that the patient's pulse corresponds to pacer presentation on the ECG and, next, should determine if the patient shows signs of decompensation, such as the following:

1. Syncope
2. Dizziness
3. Palpitations
4. Bradycardia
5. Heart blocks
6. Hypotension
7. Tachycardia
8. Extracardiac stimulation (sometimes shown by hiccoughs)

TABLE 13-12 Options for Correcting Pacer Malfunction

Solution	Use or Indication
Reprogramming	Correct oversensing, undersensing, and loss of capture As a temporary solution in some cases of an insulation break Adjust atrioventricular intervals and rate response to optimize pacing therapy
Repositioning the lead	A lead slips out of position and pacing and/or sensing is compromised Muscle, diaphragmatic, or phrenic nerve stimulation occurs Pacing the left side of the heart unintentionally (a lead may traverse the septal wall or innominate vein) Leads are reversed in the implantable pulse generator header block at implant
Replacing the lead or the pacemaker	Lead failure is confirmed The battery is depleted The patient is symptomatic (hemodynamically compromised) as a result of single-chamber pacing Passive fixation lead does not remain in place after attempts to reposition True circuit malfunction
Observation	Transient problems with the following: Lead maturation

Changes in drug therapy

If these conditions exist, alternative treatment should be initiated. First, look for obvious sources of malfunctions, such as cell phones or other personal electronic devices. If the patient is using such a device, it should be shut off and the patient should be reevaluated. If a device in the ambulance has just been turned on and the pacemaker malfunctions, the device should be shut off and the patient should be reevaluated. Another simple procedure that can be applied externally is to gently move the pulsegenerating module in the chest to attempt to uncoil the lead wires and improve lead-to-generator contact. If these quick fixes do not improve pacemaker performance, immediate treatment of the patient should begin. If bradycardia, heart blocks, or syncope exist, transcutaneous pacer (multifunction) pads should be applied, and transcutaneous pacing should be initiated. Hypotension should be treated with a fluid bolus or a vasopressor such as dopamine (Intropin). Tachycardia as the result of pacemaker runaway has virtually been eliminated, but if tachycardia is observed on the ECG, it should be confirmed by checking the pulse before considering treatment. Tachycardias that allow for adequate perfusion should be left as is, but tachycardias that result in inadequate perfusion should be treated per ACLS protocols.

Transport Management

Pacemaker Failure

- Shut off the patient's cell phone or other personal electronic device.
- If the pacemaker malfunction occurred after a device in the ambulance was turned on, shut off the device and reevaluate the patient.
- Gently move the pulse-generating module in the chest to attempt to uncoil the lead wires and improve lead-to-generator contact.
- If the malfunction persists, and the patient has bradycardia, heart blocks, or syncope, initiate transcutaneous pacing.
- Give a fluid bolus or a vasopressor (such as dopamine [Intropin]) to treat hypotension.
- Confirm tachycardia with a pulse reading; treat tachycardias that result in inadequate perfusion per ACLS protocols.

Identifying the exact cause of pacemaker failure is difficult for CCTPs without training in the use of an external diagnostic device. The easiest cause to diagnose is extracardiac stimulation evidenced by frequent hiccoughs. The hiccoughs are caused by pacing electrodes being placed too far into the apex of the ventricle, causing stimulation of the diaphragm.

Unfortunately, there is not much that can be done by CCTPs to correct implanted pacemaker malfunction because the device is not accessible to CCTPs. Even if the device were accessible, a CCTP would have to have had specific training in the repair of the particular device to initiate reprogramming. Some manufacturers provide a doughnut-type magnet that may be helpful if the problem is increased impedance at the lead-tissue interface. The magnet amplifies the current flow and may counteract the increased impedance.

Pacemakers and the ECG

Single-Chamber Systems

The sole electrode in a single-chamber system is implanted on the atrium or ventricle. Single-chamber systems require implantation of only one lead, but they do not provide AV synchrony or ventricular backup in the absence of AV conduction.

Pacing in the ventricular pacer (VVI/R) mode and loss of AV synchrony can lead to **pacemaker syndrome**, a variety of symptoms that result from hemodynamic deterioration and cause nonphysiologic timing of atrial and ventricular contractions. This asynchrony can result in an inability of the rate to respond to physiologic needs. Atrial pacemakers are appropriate only for patients who have proven AV conduction and have regular access to follow-up testing.

Atrial and Ventricular Tracings

Figure 13-90 shows an example of atrial pacing. The strip begins with an atrial pacemaker firing at a rate of 60 beats/min. A pacemaker spike precedes the p wave in these complexes. The ventricular rate is 60 beats/min. The paced p waves are conducted through the ventricles via a normal conduction pathway. Starting with the fourth complex, the intrinsic pacemaker begins depolarizing at a rate of 94 to 100 beats/min. There are no pacemaker spikes with these complexes. The pacemaker continues to sense the intrinsic atrial depolarizations. Implications for the CCTP depend on the patient's history (ie, what caused the intrinsic pacemaker to fail in the first place) and hemodynamic status.

Figure 13-91 shows an ECG from a patient with a ventricular pacer (VVI). Following the second paced ventricular beat, a capture beat appears and is conducted normally. The pacemaker does not fire during this capture beat because it is sensed as normal depolarization. When another capture beat fails to appear, the ventricular pacemaker senses this and fires, depolarizing the ventricles. It paces two beats, then another capture beat appears and is conducted. When this does not produce a sustained rhythm, the pacemaker senses this and begins firing again at a rate of 60 beats/min. The implication for the CCTP is that this patient's heart is probably failing, but still has the ability to generate electrical impulses periodically. Monitor the patient's hemodynamic status and treat accordingly.

Some of the newer pacers do not show a prominent spike; at times, several leads must be checked to identify the spike. It is a good idea to ask the sending nurse what type of pacer the patient has.

Dual-Chamber Systems

Dual-chamber systems feature two leads, one each in the atrium and the ventricle. This ensures AV synchrony and provides for ventricular depolarization even without AV conduction. This mode of pacing is known as atrial synchronous pacing, atrial tracking, or AV sequential pacing.

As discussed in Table 13-11, DDD and DVI are pacemaker acronyms that refer to dual-chamber pacemaker systems. DDD stands for dual sensed, dual paced, and dual mode (fixed or demand), and DVI stands for dual sensed, ventricular paced, and inhibited (demand) mode. DDD is the most common type of pacemaker in use today. Figures 13-92 through Figure 13-95 show samples of ECG rhythms from dual demand pacemakers.

Figure 13-92 shows a dual-chamber intrinsic atrial pacemaker depolarizing at a rate of approximately 60 beats/min. The pacemaker senses atrial electrical activity and does not need to provide atrial pacing. The atria depolarize but conduction is blocked and does not reach the ventricles. The ventricular mode of the DDD senses no conduction and fires to depolarize the ventricles at a rate of 60 beats/min. Pacemaker spikes precede each ventricular complex. This patient has a severe conduction problem with impulses not reaching the ventricles. CCTP implications would be to monitor the patient's hemodynamic status and treat accordingly.

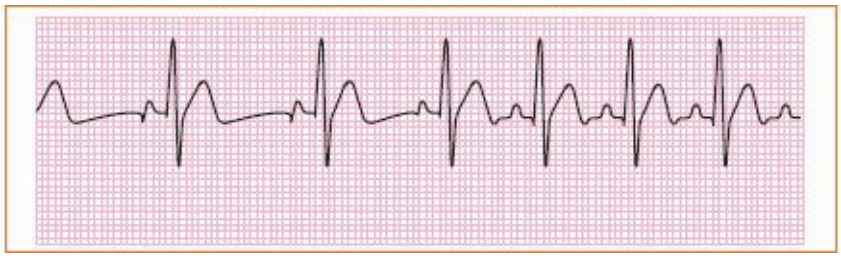


Figure 13-90 An electrocardiogram from a patient with an atrial pacer (AAI). Mode: AAI; rate setting: 60.

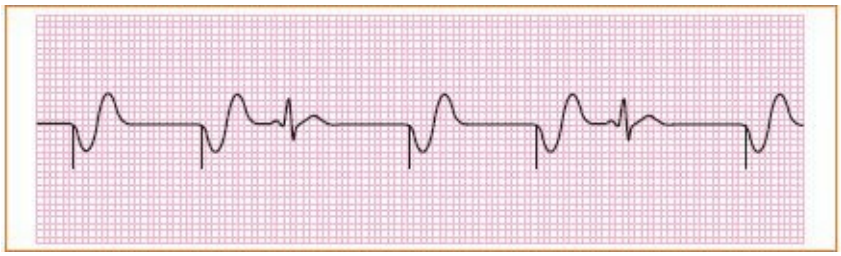


Figure 13-91 An electrocardiogram from a patient with a ventricular pacer (VVI). Mode: VVI; rate setting: 60.

Figure 13-93 shows a demand dual-mode (DDD) pacer sensing intrinsic pacemaker depolarizing at a rate of approximately 80 beats/min. The electrical activity is conducting from the atria to the ventricles without interruption. The underlying rhythm is a regular sinus rhythm at a rate of approximately 78 beats/min. Implications for the CCTP are to always compare ECG findings with the clinical presentation of the patient.

Figure 13-94 shows a dual-demand mode (DDD) pacemaker rhythm. This strip shows a demand DDD pacemaker that does not sense any intrinsic electrical activity and depolarizes the atria as indicated by a spike preceding each p wave at a rate of 60 beats/min. With no ventricular activity and the atrial pacing not being transmitted through to the ventricles, the ventricular mode of the DDD fires, depolarizing the ventricles at a rate of 60 beats/min. The implication for the CCTP is that the patient's cardiac electrical activity is depending solely on the pacemaker. As always, compare ECG findings with clinical findings and treat accordingly.

Figure 13-95 shows another demand dual-mode (DDD) pacemaker rhythm. The underlying rhythm appears to not be producing an atrial intrinsic pacemaker rhythm. The DDD pacer senses this and paces a rhythm at 60 beats/min. Note the pacemaker spike preceding each p wave. The electrical impulse generated by the pacemaker is transmitted through to the ventricles without interruption, allowing the ventricles to depolarize "normally" from above. This indicates that there is no AV block. The ventricular portion of the pacemaker continues to sense the ventricular conduction and does not need to fire. There should be no implications for the CCTP with this rhythm (unless this started after the transport began and could indicate a deteriorating condition).

Atriobiventricular Pacing

A CCTP may transport a patient with heart failure to a facility for pacemaker insertion. Heart failure, also known as CHF, is a condition that occurs when the heart is damaged and unable to pump blood adequately. Mortality and morbidity rates are high for patients with CHF.

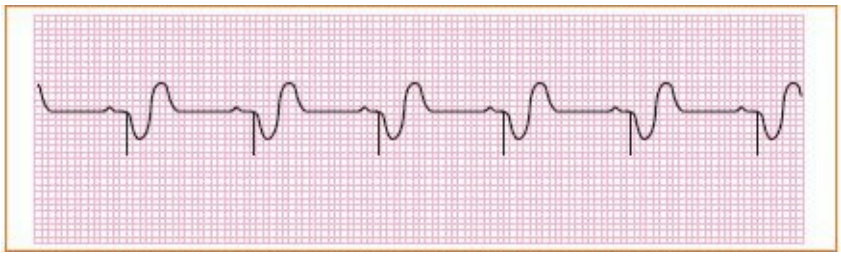


Figure 13-92 Mode DDD; lower rate setting, 60; upper rate setting, 120.

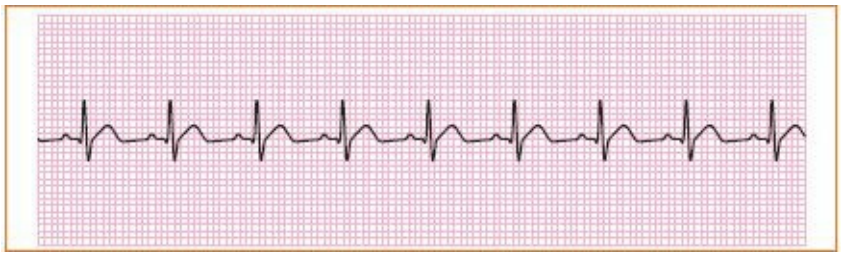


Figure 13-93 Mode DDD; lower rate setting, 60; upper rate setting, 120.

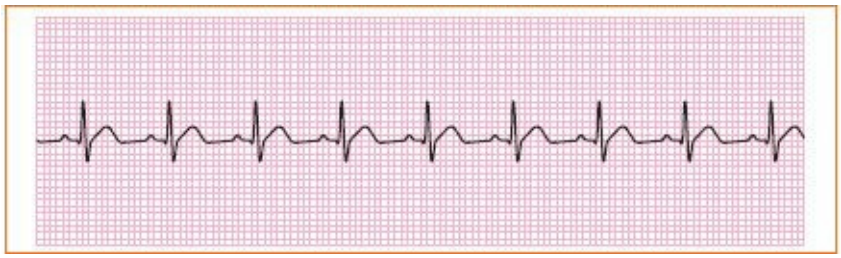


Figure 13-94 Mode DDD; lower rate setting, 60; upper rate setting, 120.

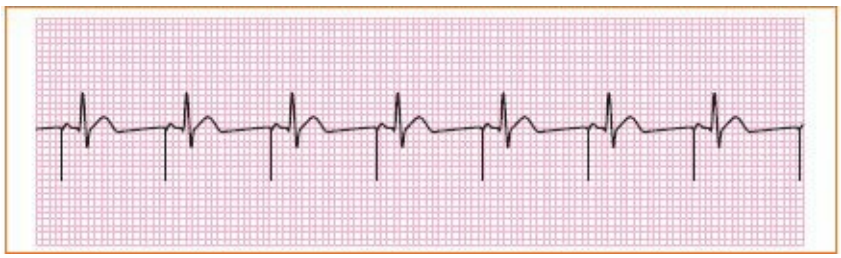


Figure 13-95 Mode DDD; lower rate setting, 60; upper rate setting, 120.

One effective new treatment for CHF uses a modified cardiac pacemaker or ICD to resynchronize the heart chambers. Once functioning in an organized manner, the heart pumps blood more efficiently and the effects of heart failure are reduced. The biventricular pacemaker coordinates the right and left ventricles of the heart. This coordination is accomplished by placing pacing leads in both ventricles and then optimizing their synchronization.

In a typical biventricular implant, one lead is placed in the atria and one lead is placed in each ventricle (right and left) **Figure 13-96**. A biventricular pacing waveform would be indistinguishable from that of a dual-chamber pacer on a typical monitor lead as the ventricles are paced simultaneously **Figure 13-97**. Analysis by 12-lead ECG and history would lead to a more definitive answer regarding the type of device implanted.

Implantable Cardioverter Defibrillators

Implantable cardioverter defibrillators automatically attempt to convert a fibrillating heart to sinus rhythm using a shock from voltage **Figure 13-98**. Since their introduction in 1985, the estimated number of implants has increased steadily to more than 50,000 each year. As the numbers rise, so does the frequency of patients with ICD-related issues.

Basic Function

Components

Each patient with an ICD has a custom ICD system consisting of a housing (can) and a variable number of pacing and high-voltage leads. The leads can be placed in the abdominal or pectoral region **Figure 13-99A**; the lead is itself often one of the electrodes in the system. Most ICDs are implanted with one or two transvenous leads used for pacing and shocking. The leads enter the venous system through the subclavian or cephalic vein, for placement into the right ventricle or the superior vena cava. Early ICD implants required that pacing leads and high-voltage patches be placed directly on the epicardial surface **Figure 13-99B**. Patients with mechanical heart valves are also limited to epicardial placement.

Sensing and Therapies

An ICD delivers therapy when it determines that the heart is in a tachyarrhythmia, based on the ventricular rate. The ICD counts the number of R-R intervals that are faster than a programmed threshold. When the count reaches the threshold, the device delivers electrical therapy. Because an ICD bases its decision on the ventricular rate alone, atrial arrhythmias and electromagnetic interference can lead to inappropriate shocks.

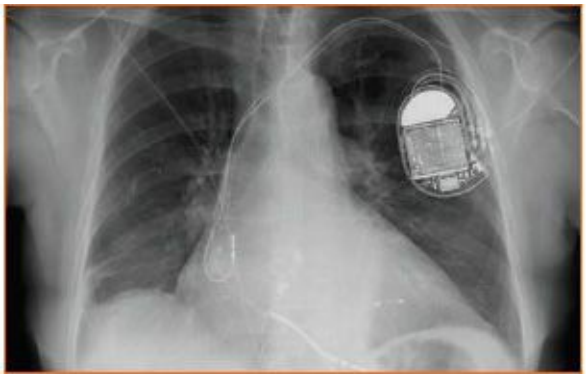


Figure 13-96 An implanted atriobiventricular pacing system.

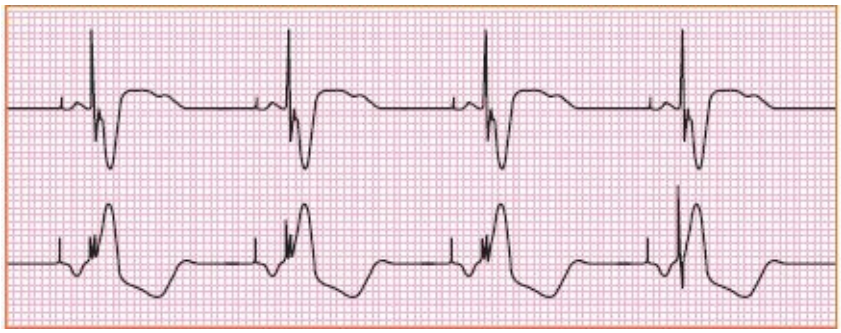


Figure 13-97 An example of a 12-lead electrocardiogram with atriobiventricular pacing.

For ICD purposes, fast heart rhythms are loosely classified into two groups: VT and ventricular fibrillation. An ICD may deliver antitachycardia pacing or cardioversion shocks for episodes of VT. Antitachycardia pacing is a series of 6 to 15 pacing pulses with cycle lengths of 70% to 95% of the

arrhythmia **Figure 13-100A**. In Figure 13-100A, when VT is sensed, antitachycardia pacing begins as indicated by pacemaker spikes (arrow at right). Cardioversion shocks are high-voltage shocks that are synchronized to an intrinsic R wave **Figure 13-100B**. The device delivers unsynchronized defibrillation shocks for episodes of ventricular fibrillation. In Figure 13-100B, when ventricular tachycardia is sensed, a single cardioversion shock is administered as indicated by the large spike. The rhythm is converted to a supraventricular rate of about 70 beats/min. There appears to be in a first-degree AV block with a prolonged P-R interval.

Implantable Cardioverter Defibrillator Troubleshooting

Because an ICD is an implantable device within the patient, it is not accessible to CCTPs. The device must be deimplanted (in the hospital) to correct the malfunction.

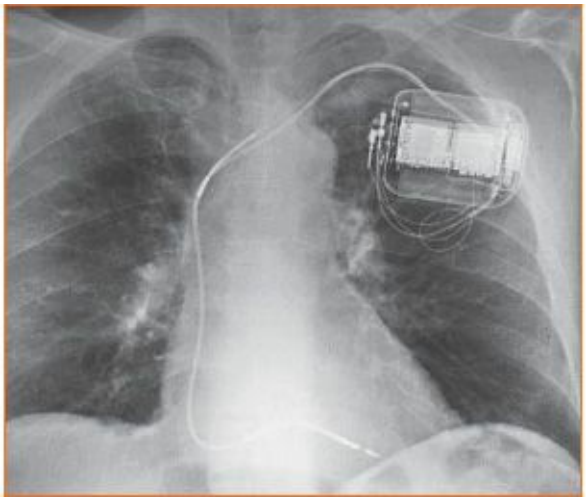


Figure 13-98 An implantable cardioverter defibrillator is attached directly to the heart and continuously monitors heart rhythm, delivering shocks as needed. The electricity from the device is so low that it has no effect on rescuers.

The most common sources of failure are the following:

1. Wire (lead) detachment or insulation failure
2. Battery failure or abnormalities
3. Pulse-generator failure
4. Movement of the wire tip in the ventricle

In addition, dual-chamber ICDs are subject to all of the problems described in the pacemaker section earlier in this chapter.

Information Gathering

If an ICD fires during the care of a patient, everything possible should be done to gather information that will help specialists confirm later whether the therapy was appropriate. After such an event, the patient's electrophysiologist will want to review the transport documentation and data from the ICD. Programming equipment that is unique to the ICD model makes it possible to retrieve detailed information from the device.

Many patients who have been shocked before recognize the symptoms of ventricular arrhythmias and can correctly identify appropriate and inappropriate therapies. A number of historical factors can have a role in ICD malfunction or offer clues about the nature of the problem. Among the most useful are the

following:

- Number of times shocked. Patients with VT "storms" have been known to have the device giving more than 100 shocks in a 24-hour period. When a patient has an atrial arrhythmia that conducts rapidly to the ventricle, it is common to deliver repeated unsuccessful therapies.
- Recent medical procedures. Many medical procedures can damage an ICD system, especially when directed to areas of the body in proximity to the ICD. Diathermy, lithotripsy, magnetic resonance imaging, and radiation treatments are among the most common offenders.

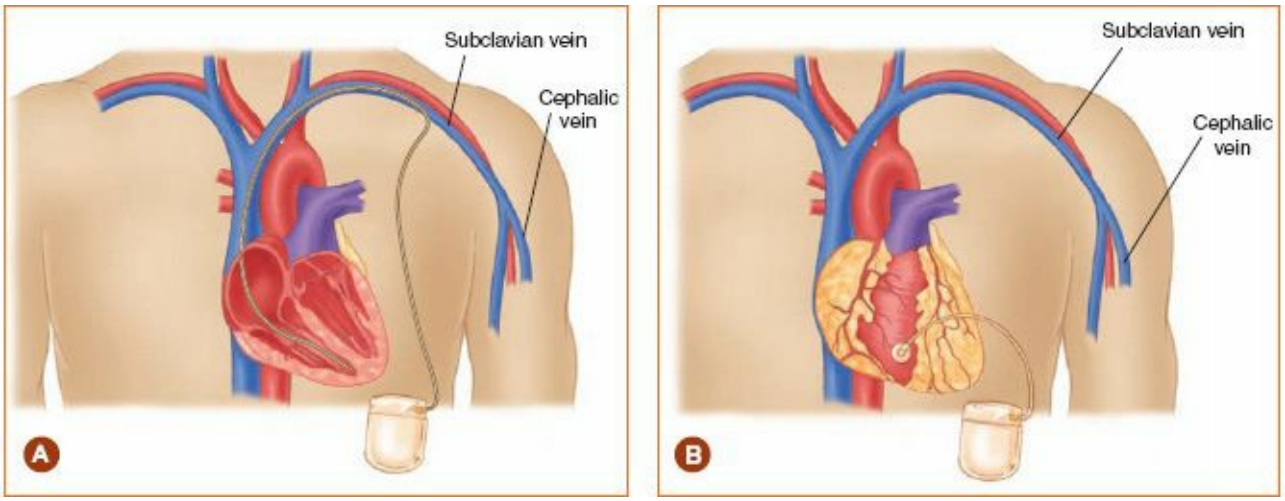


Figure 13-99 Typical implantable cardioverter defibrillator (ICD) systems. **A.** The ICD enters the heart through the abdominal or pectoral region. **B.** The ICD is directly on the epicardial surface.

- Recent changes in medical condition or in patient adherence to the medication regimen. It is not uncommon for patients to stop taking antiarrhythmic drugs if they have been arrhythmia free for several months or years.
- Coexisting pacemaker. Before the introduction of dual-chamber defibrillators in 1998, many patients received a dual-chamber pacemaker and a ventricular defibrillator. Interactions between the two devices can inhibit appropriate therapy or trigger inappropriate therapy.
- Activity at the time of shock. During exercise, the ICD may deliver inappropriate shocks to sinus tachycardia.
- Proximity to water or an electrical source. Poorly grounded water pipes can produce trickle electrical currents that the ICD may pick up as a rapid heart rate. Likewise, a nearby source of electromagnetic interference can cause the ICD to discharge inappropriately.

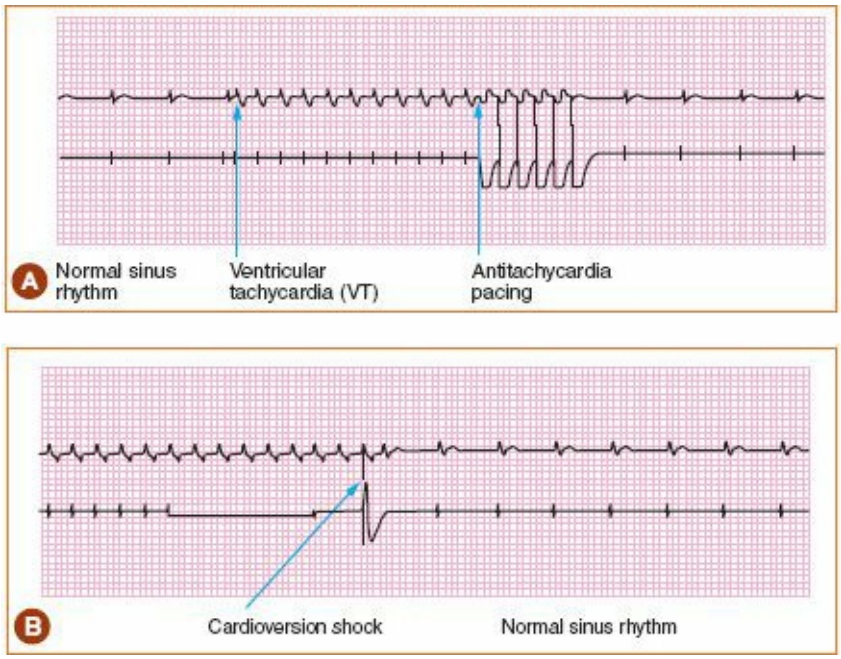


Figure 13-100 Implantable cardioverter defibrillator electrotherapies for ventricular tachycardia. **A.** Electrotherapy for tachycardias (antitachycardia pacing). **B.** Cardioversion.

Identifying the Problem

Occasionally, a patient with an ICD will experience multiple inappropriate defibrillator discharges as the result of defibrillator malfunction. This is uncomfortable and agitating to the patient and may cause damage to the myocardium if allowed to continue. The ICD can be temporarily disabled by a magnet to prevent further discharges. Manufacturers of ICDs can provide special magnets (usually doughnut shaped) that are placed directly over the pulse generator. The magnet creates a magnetic field that trips a reed switch in the ICD. When the magnet is applied, detection of tachycardia and therapy are discontinued. For most applications, a single magnet is all that is required; however, for obese or edematous patients, two magnets may be necessary. Before using the magnet, multifunction pads must be placed on the patient and hooked to the monitor; then the ICD can be deactivated. However, ICD manufacturers discourage deactivation of a malfunctioning ICD during transport. Certain ICD manufacturers make an external interrogation device that will help identify the source of the problem and, in some cases, allow reprogramming, but CCTPs should receive special training before using such a device.

In a cardiac arrest, external defibrillation can be delivered **Figure 13-101**, but the paddles or pads should not be placed directly over the ICD. CPR can also be performed on a patient with an ICD, with no danger to providers, the patient, or the implanted device. When delivering defibrillation, it is important to use A-P paddle placement and position the pads or paddles away from the ICD.

Figure 13-101 A commonly used monitor-defibrillator.

Figure 13-102 and Figure 13-103 illustrate some of the common problems with ICDs. The ICD stores these data during the episode for later retrieval with programming equipment.

In **Figure 13-102**, the ICD is oversensing the pacing spikes of a pacemaker. The VS markers align with each ventricular pacing spike. The underlying rhythm appears to be ventricular fibrillation. The top portion of the strip shows an ICD/DDD pacer firing atrial/ventricular spikes that are not capturing or correcting the rhythm. The bottom strip shows a single spike at a rate of around 80 beats/min that is not capturing. CCTP implications include first checking the clinical status of the patient, and if he or she is unconscious and unresponsive (which would be expected with this ECG), performing CPR, defibrillation, and medication administration per ACLS protocols.

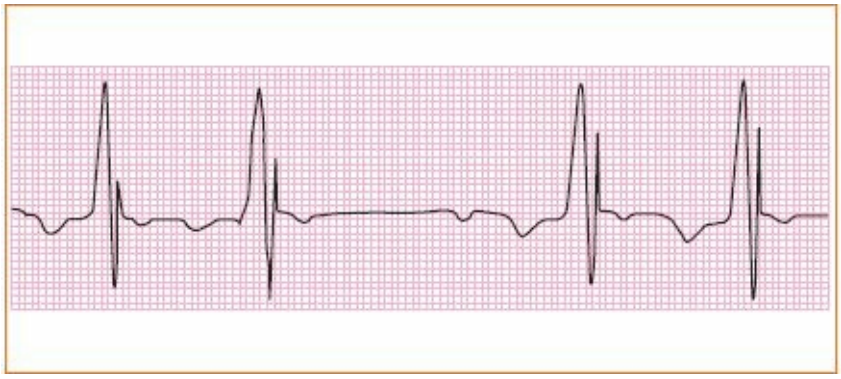


Figure 13-102 An electrocardiogram in which the implantable cardioverter defibrillator is oversensing the pacing spike.

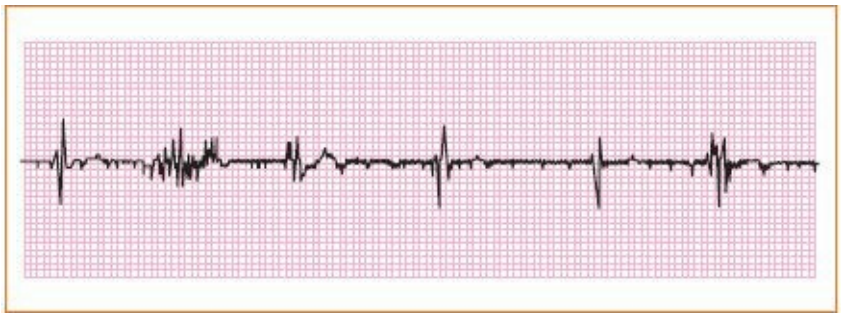


Figure 13-103 An example of pacemaker lead malfunction.

Figure 13-103 shows an example of lead malfunction. Intervals in the range of 120 to 150 milliseconds are usually not physiologic; they more likely indicate electromagnetic interference or a problem with the sensing lead. In this figure, there appears to be an irregular rhythm conducting at a rate of 60 to 80 beats/min. This ECG indicates the ventricular pacemaker is sensing at an irregular rate as well (malfunction). There are no clear pacemaker spikes indicating that it is firing appropriately. CCTP implications would include monitoring the patient for hemodynamic stability and initiating CPR and external pacing as indicated.

Atrial Tachycardia Implantable Cardioverter Defibrillators

Atrial fibrillation is one of the most common arrhythmias found. Significant risks associated with atrial fibrillation are thromboembolism and a resulting increased incidence of stroke. Conventional methods to convert atrial fibrillation have centered on antiarrhythmic medications and external cardioversion. Recently, internal cardioversion and/or antitachycardia pacing with an atrial ICD has been shown to be

the preferable mode of treatment. Advances in lead design and the use of biphasic energy waveform have made this possible. With biphasic waveform, lower energy levels may be used to effectively convert atrial fibrillation to a normal sinus rhythm.

Three modes for activating the atrial tachycardia ICD are generally use. In the patient-activated mode, a small handheld remote can be used by the patient to initiate therapy. This would occur after the device had notified the patient that atrial fibrillation was present. A second mode would involve the physician activating the device to provide appropriate therapy. In addition, the ICD may be programmed to automatically deliver therapy on detection of the arrhythmia. These ICDs commonly act as pacemakers also, and the atrial and ventricular leads may be dual purpose.

Transport Considerations for Cardiac Electrophysiology Devices

Devices to improve cardiac electrophysiologic performance are frequently encountered during critical care transport. Implantable cardioverter-defibrillators are currently the recommended therapy for the prevention of death from ventricular arrhythmias. Of all ICDs implanted in the world, 65% are done in the United States, despite the fact that only approximately 34% of the patients who should receive an ICD in the United States actually have one implanted. Once utilization improves, patients with ICDs will become increasingly common. When the epidemiology of pacemaker placement was first evaluated in the United States in 1995, there were more than 450,000 people with pacemakers in the general population. Annual pacemaker insertion procedures increased more than 20% in the following 5 years. CCTPs should expect to transport patients who have or need a pacemaker or ICD and should be prepared to manage any adverse situations related to either device.

Figure 13-104 shows an algorithm for online medical control during critical care transport and should assist in understanding the process. This algorithm can apply to cardiac transports and to other types of transports.

Preparation

The management of patients with a pacemaker or ICD begins well in advance of any transport request. CCTPs should receive training on any transthoracic, transcutaneous, or transvenous pacemaker device that the agency uses. In larger urban settings, CCTPs may encounter transthoracic, transcutaneous, and transvenous pacemakers from a wide variety of manufacturers at the different sending facilities, requiring additional scrutiny when picking up or dropping off patients at these facilities. In addition, agencies transporting critically ill cardiac patients should have a magnet capable of temporarily reprogramming an implanted pacemaker or ICD readily available. It may be necessary to review the instructions for a particular device while en route to a patient transport if transthoracic, transvenous, or transcutaneous pacing is anticipated and the CCTP has not used the particular equipment recently.

Table 13-13 lists information and assessments needed for critical care transport of cardiac critical care patients.

TABLE 13-13 Information and Assessments Needed for Transport of Critical Cardiac Patients

Information needed before	1. List of patient's current medications 2. Patient's allergies to medications 3. 12-lead electrocardiogram and rhythm history 4. History leading to present condition 5. Patient's past medical history

transport	6. Treatments administered to date 7. Problems encountered to date 8. Nurse's report before transport 9. Review with nurse any drips patient is receiving or may require. 10. Review with respiratory therapist any special respiratory requirements.
Overall assessment before transport	1. Level of consciousness, Glasgow Coma Scale 2. Anxiety (may need to be controlled) 3. Overall appearance 4. Pain (level, location, radiation, quality) 5. Blood pressure 6. Heart rate and pulse quality 7. Oxygen saturation 8. If the patient requires a ventilator: a. Breaths/min b. Tidal volume c. Positive end-expiratory pressure d. Peak inspiratory pressure e. Capnography carbon dioxide level and waveform f. Oxygen saturation as measured by pulse oximetry 9. If the patient has an arterial line: a. Check the French size. b. Check for bleeding or a hematoma at the catheter site. c. Check that there is sufficient pressure coming from the pressure bag into the arterial line. 10. Check all IV sites to be sure that they are patent. 11. If there are any drips being infused, ensure that the pumps are operable, note the drip rate, and ensure that there are sufficient fluids or medications. 12. Observe the cardiac monitor rhythm and rate. 13. Note and correct any gastric disturbances because they may induce subdiaphragmatic pressure. 14. All critical care transport patients should receive a thorough head-to-toe exam before transport; for cardiac patients, particular attention should be given to assessing jugulovenous distention, bruits, heart sounds (ie, S_3 and S_4), and valve closure sounds; all lung fields should be auscultated and chest rise should be noted.
Tests to know and review	1. Electrolytes, particularly potassium, sodium, magnesium, and calcium 2. Glucose 3. Coagulation tests, international normalized ratio if receiving warfarin (Coumadin) 4. Troponin, brain-type natriuretic peptide, D-dimer 5. Ejection fraction

	6. Cardiac index
Complications and interventions	1. Increased chest pain: increase the nitroglycerin drip rate; administer morphine sulfate, hydromorphone (Dilaudid), or fentanyl citrate. 2. Increased blood pressure: increase the nitroglycerin (Nitro-Bid IV, Tridil) drip rate; consider a beta-blocker (such as metoprolol [Lopressor, Toprol XL]).

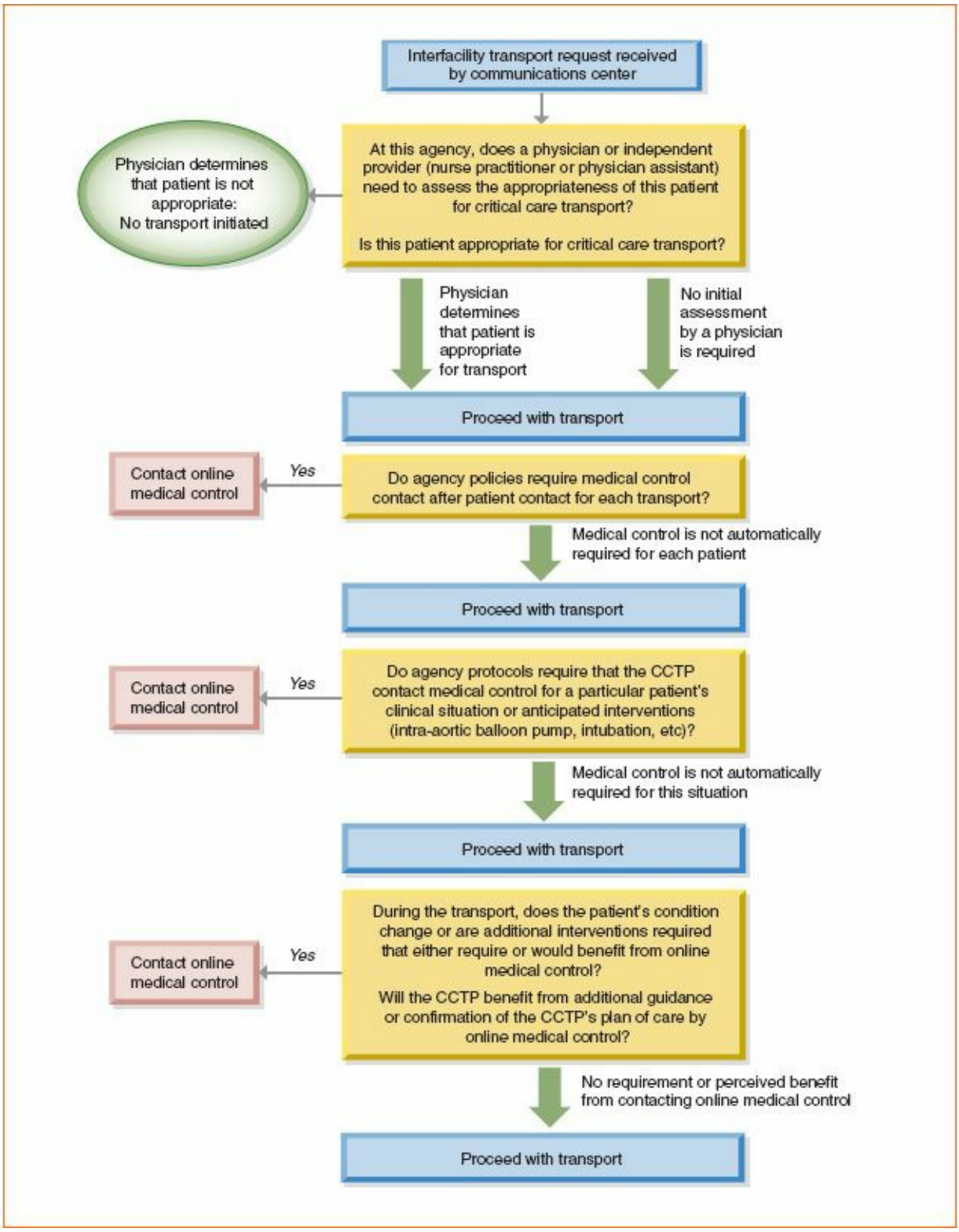


Figure 13-104 Algorithm for online medical control during critical care transport. CCT indicates critical care transport; IABP, intraaortic balloon pump; NP, nurse practitioner; PA, physician assistant.

Patient Acceptance

Accepting a patient from a sending hospital for critical care transport is often a complicated endeavor. CCTPs must obtain a patient report, typically from the sending nurse or physician, which includes the history of present illness, medical history, medications, allergies, hospital course, results of recent laboratory and diagnostic studies, current clinical status, relevant social information, presence of advance directives, and any anticipated changes or interventions during the transport. During patient acceptance, CCTPs must perform a physical examination of the patient appropriate for the particular medical condition, clinical status, and the duration and method of transportation. Any medication infusions, including concentrations and rate calculations, should be confirmed and double checked for accuracy with the sending nurse, physician, or other provider. Ventilator settings, monitoring equipment, indwelling devices, and fluid infusions must also be continued or modified as appropriate for the patient's condition and capabilities of the transport team. Copies of patient medical records, laboratory results, and diagnostic studies should accompany the patient for transport. All of these actions should ideally occur in the shortest time possible, facilitating prompt patient transfer to a higher level of care.

Any patient with a transthoracic, transvenous, transcutaneous, or implanted pacemaker device must have electrical and mechanical pacemaker capture assessed and confirmed. A CCTP may make a potentially lethal error if he or she mistakenly assumes that electrical capture present on an ECG tracing represents true mechanical capture. Mechanical capture is demonstrated by palpable pulses (or evidence of myocardial contraction on ultrasound) with each pacemaker complex on the ECG. It is possible for electrical capture to be present on the ECG without any actual contraction and pumping of the heart, disguising an underlying arrhythmia or cardiac arrest. Unrecognized lack of mechanical capture may have lethal consequences.

It is not always possible to continue using the same transcutaneous pacemaker machine or transvenous or transthoracic pacemaker pulse generator for transport that is being used at the sending facility. Switching from a device used in a sending facility to a device used by the transport team is often the source of anxiety for CCTPs and other providers. Transvenous and transthoracic pacemaker leads are typically compatible with pulse-generator devices from multiple, if not all, manufacturers. Care must be taken to ensure equipment compatibility and correctly match rate and current settings as the devices are switched. CCTPs must carefully check the patient for electrical and mechanical capture, in addition to other clinical indicators, when there has been a change from one pacemaker to another. If a CCTP does not have a compatible device available, it may be worth considering using the device from the sending facility for the transport and returning it promptly after the transport has been completed, although this may not be possible in many situations. Certain transcutaneous pacemaker machines have adapters to allow the use of pads from multiple manufacturers. If adapters are not available, it may be necessary to completely change transcutaneous pacing pads for transport.

CCTPs should also inquire about whether the patient has an ICD. The presence of an ICD would require an alternative site (not over the device) for transcutaneous pacing or defibrillation if needed during transport. An improperly functioning ICD has the potential to inappropriately discharge, causing patient discomfort, myocardial damage, and changes on the ECG. In a 2009 study, 11.5% of the subjects with ICDs had inappropriate shocks, and almost 8% of the subjects with ICDs *only* had inappropriate ICD discharges. CCTPs may use a pacemaker-type magnet to temporarily suspend cardioversion or defibrillation when an ICD administers continued inappropriate shocks.

A properly functioning ICD may terminate certain potentially lethal arrhythmias without the need for a CCTP to perform any external cardioversion or defibrillation during transport. It is still essential to have an external cardioverter-defibrillator immediately available. CCTPs may experience a mild sensation if they are in contact with a patient as an ICD discharges, but no serious injuries to bystanders or health care providers have been reported.

Patient Transport

The transport environment continually poses challenges for CCTPs providing patient care. Limited patient access, lack of supporting resources, adverse environmental conditions, and a myriad of safety concerns must be encountered with each transport. CCTPs must manage all of these factors when transporting critically ill cardiac patients with electrophysiologic devices **Figure 13-105**.

Two factors in the transport environment require special consideration for patients with pacemakers or ICDs. Electromagnetic interference is the major cause of pacemaker dysfunction, particularly in older pacemakers lacking adequate internal shielding. Modern chip-based pacemakers are less susceptible to electromagnetic interference. Neither small single-engine airplanes nor larger commercial aircraft pose an increased risk for electromagnetic interference affecting implanted pacemakers or ICDs. CCTPs should be aware that devices such as headphones and microphones with internal magnets may cause interference in older pacemakers when placed within 1″ to 2″ of the device. The American Heart Association recommends that any type of headphone remain more than 3 cm from the pacemaker to avoid the risk of electromagnetic interference. If patients are provided with headsets or intercom systems during air transport, CCTPs should take care to keep these items safely away from the pacemaker device, which is usually visible as a mass under the patient's skin.

Figure 13-105 The CCTP must manage the challenges of the transport environment when transporting a cardiac critical care patient.

Patient Turnover

CCTPs are expected to provide the receiving hospital with a thorough patient report, including the same elements outlined in the section on patient acceptance. In addition, CCTPs should provide the intensive care unit, catheterization laboratory, operating room, or other receiving unit staff with an accurate report of any patient changes and interventions provided during the transport. Certain high-risk items, such as endotracheal tube placement, central line status, IV sites for vasoactive medications, and the presence of mechanical pacemaker capture, should be specifically confirmed with the accepting provider on patient turnover to avoid any question that problems with these items occurred during patient transport. Oral patient report should be supplemented with a written document outlining the preceding information that is left with the receiving provider at the completion of the transport. CCTPs should carefully document the patient condition on acceptance and turnover; the conveyance of patient belongings, medical records, and diagnostic test multimedia; and the name(s) of the health care provider(s) who accepted the patient at the receiving facility. Although failure to accurately document this information may place a CCTP at significant legal, regulatory, or financial risk, should adverse events occur, the primary reason for documentation is continuity of care.

CCTPs should expect frequent encounters with patients who have or need a device to improve

cardiac electrophysiology. Proper training and education will improve a CCTP's ability to assess and manage these patients in the transport setting.

Flight Considerations

Pacemaker malfunction does not usually occur as a result of the flight environment. There are some isolated reports of helicopter vibration affecting pacemaker function; occasionally, rate-responsive pacemakers increase their firing rate as a result of vibration during helicopter transport. However, in a study involving helicopter transport of dozens of patients with pacemakers, no pacemaker malfunctions occurred. This does not usually occur in the fixed-wing environment, although it could occur if there is significant turbulence. CCTPs working on medical helicopters should be aware of this potential and should remain prepared to intervene if necessary.

Finally, always remember that as altitude increases, supplemental oxygen is important.

Summary

Providers' ability to diagnose and treat patients with cardiac arrhythmias has been greatly enhanced by advances in technology, and keeping up-to-date with the latest developments on this front is imperative for all CCTPs. This chapter reviewed how to interpret 12-lead ECGs in the context of the critical care transport setting. One of the most valuable skills that can be learned is the ability to better identify an early MI, relay this information to the receiving facility or treating physician, and decrease the time to treatment. In addition to identifying an MI, acquiring a diagnostic-quality 12-lead ECG in the field, even if it is normal, will provide the treating staff with a baseline to compare serial ECGs done in the follow-up evaluation of the patient.

Over the years, pacemakers and ICDs have evolved through a variety of designs with varying functions and capabilities. Although detailed information about a patient's implanted device is often available to CCTPs, general familiarity with basic concepts will always be helpful in determining whether these devices are functioning properly.

Pacers and ICDs share numerous basic components and functions. Modern devices all sense heart rate and use programmed circuitry to decide whether or how to respond. The exact types of sensing they perform and the therapies they deliver are where the types of devices differ the most.

Knowing the NBG codes is perhaps the single most valuable tool in a CCTP's approach to a patient with a pacemaker. Studying as many ECGs as possible of real patients with pacers and ICDs will help CCTPs prepare for identifying and managing problems in transport. In any case of suspected or certain malfunction, it will be important to the electrophysiologist that the CCTP obtain ECG recordings, ensure availability of data from the device, and provide detailed written documentation of the incident.

Case Study

JUST AS YOU ARE FINISHING YOUR LUNCH, dispatch advises you of an emergency transfer of a critical cardiac patient from Community Hospital to St Patrick's Hospital. Your crew, consisting of an EMT-B driver, yourself, and a fellow paramedic, responds in 12 minutes to the Community emergency department.

On arrival, you obtain a report from the transferring physician, Dr Jans, who states your patient is a 67-year-old, 111.36-kg Native American man who arrived in the emergency department approximately 45 minutes ago complaining of severe substernal chest pain and also had hypertension. The patient's 12-lead ECG revealed no acute ST-segment changes, but showed a new-onset left BBB when compared with a

prior ECG that was obtained during a routine visit to his primary care provider 4 months earlier. His medications in the emergency department included 324 mg of aspirin; 0.4 mg nitroglycerin, 2 doses; 3 mg of morphine IV; 300 mg of clopidogrel (Plavix); and 20 mg of eptifibatide (Integrilin). Presently, the patient is receiving a continuous infusion of nitroglycerin (Nitro-Bid IV, Tridil) at 20 µg/min and an eptifibatide infusion at 222 µg/min. The patient is being transferred to Dr Webb at St Patrick's catheterization laboratory for emergency cardiac catheterization.

As you assess the patient, you obtain the following readings: a heart rate of 94 beats/min, sinus rhythm; a respiratory rate of 18 breaths/min, clear and equal breath sounds bilaterally; a blood pressure of 174/110 mm Hg; a temperature of 99.1° F; pulse oximetry reading of 95% with 4 L/min of oxygen via nasal cannula; carbon dioxide, 42 mm Hg; heart tones normal with no rubs, gallops, or murmurs; and peripheral edema 1 + in the lower extremities.

Further interview of the patient reveals a past medical history of hypertension and hepatitis. Presently, he takes no medications and has no allergies. He stated that the pain started approximately 90 minutes ago and began radiating to his left arm. Initially, the pain was 10 on a 10-point scale, but after morphine was given, has decreased to 2 on a 10-point scale. As your partner secures the patient onto the stretcher, you transfer the infusions to your transport IV pump.

As you review the patient's lab studies, you note the following values of interest:

- Glucose: 98 mg/dL (normal range, 74 to 110 mg/dL)
- Chloride: 110 mmol/L (normal range, 98 to 107 mmol/L)
- Potassium: 4.1 mmol/L (normal range, 3.5 to 5.1 mmol/L)
- Calcium: 8.6 mg/dL (normal range, 8.5 to 10.1 mg/dL)
- Magnesium: 1.7 mEq/L (normal range, 1.5 to 2.0 mEq/L)
- Blood urea nitrogen (BUN): 13 mg/dL (normal range, 7 to 25 mg/dL)
- Creatinine: 1.3 mg/dL (normal range, 0.6 to 1.3 mg/dL)
- BUN/creatinine ratio: 10.0 calculated (normal range, 12.0 to 20.0 calculated)
- Prothrombin time: 14.1 seconds (normal range, 14.0 to 16.0 seconds)
- Activated partial thromboplastin time: 53.0 seconds (normal range, 45.0 to 55.0 seconds)
- Myoglobin: 24 ng/mL (normal range, 10 to 95 ng/mL)
- Troponin T: 1.6 ng/mL (normal range, 0.0 to 0.2 ng/mL)
- Brain-type natriuretic peptide: 140 µg/mL (normal range, 0.0 to 100.0 µg/mL)
- Alanine aminotransferase: 392 U/L (normal range, 30 to 65 U/L)
- Aspartate aminotransferase: 110 U/L (normal range, 15 to 37 U/L)

You load the patient into the ambulance and proceed on an emergency basis to St Patrick's Hospital, approximately 110 miles away. As you reassess the patient, your partner increases the nitroglycerin to 25 µg/min in an attempt to maintain the systolic blood pressure around 155 mm Hg. The patient appears to be in a moderate amount of distress; however, he denies having any discomfort. You opt to administer 3 mg of morphine IV, to which he replies "Thanks, that helps me relax and not hurt quite as bad."

Your partner repeats a 12-lead ECG to compare with the one that was done in the emergency department and the one from 4 months ago. The ECG remains unchanged from the one done in the emergency department, with no ST-segment elevation or depression. The remainder of the transport remains unremarkable, with the patient delivered to Dr Webb and the waiting catheterization laboratory team.

1. If this patient did not have any ECG changes, how are you able to determine that he was experiencing

an MI?

2. Why was this patient receiving nitroglycerin? Eptifibatide?
3. What are the side effects of these medications?
4. What are the possible complications that may be experienced en route with this patient?

Analysis

Because of your critical care training, you are able to recognize that this patient is experiencing an NSTEMI based on the given lab values. You know that myoglobin will be the first cardiac marker to increase (usually within 2 hours after muscle damage), and that troponin will increase 3 to 6 hours after infarction and is the most specific cardiac marker.

Nitroglycerin is indicated in MI situations owing to its vasodilatory properties on systemic and coronary vessels. The enhanced coronary perfusion, along with the decrease in cardiac workload, results in increased oxygen and blood supply to ischemic cardiac tissue. The possible side effects of using nitroglycerin for this patient are hypotension, reflexive tachycardia, and cardiac arrhythmias.

Eptifibatide is indicated in patients with unstable angina or NSTEMI to reduce the risk of acute ischemic events. Eptifibatide is an antiplatelet medication that selectively blocks the platelet glycoprotein IIb/IIIa receptor. Possible side effects of eptifibatide are uncontrollable bleeding, anemia, and thrombocytopenia.

Owing to the nature of this patient's situation and the different medications that have been administered, careful monitoring en route is essential. Some of the potential problems en route would be hypotension from inadvertent nitrate overdose, cardiogenic shock caused by pump failure, respiratory arrest due to narcotic potentiation, and, ultimately, cardiopulmonary arrest.

That evening as you are delivering yet another patient to the emergency department at St Patrick's, you see Dr Webb and ask about the status of the patient you brought in earlier. He replies that the patient underwent cardiac catheterization that revealed significant narrowing of the left main coronary artery. Owing to the severity of the narrowing, placing a stent was contraindicated, so the patient underwent an emergency coronary artery bypass graft in which a section of the left internal mammary artery was removed and grafted to the left anterior descending artery. The patient is expected to make a complete recovery and be released in 7 to 10 days.

Prep Kit

Ready for Review

- Oxygenated blood reaches the heart through the coronary arteries, which are vulnerable to blockage and narrowing by plaque formation, a condition called atherosclerosis.
- The major heart sounds are the two normal sounds, S_1 and S_2, and the two abnormal sounds, S_3 and S_4.
- S_1 (the "lub" sound) occurs near the beginning of ventricular contraction (systole), when the tricuspid and mitral valves close. Any delay in the closing of these two valves, heard as a split sound, is considered abnormal.
- S_2 (the "dub" sound) occurs near the end of ventricular contraction (systole), when the pulmonary and aortic valves close. The two valves can close simultaneously or with a slight delay between them under normal physiologic circumstances.

- S_3 (“dub-lub-dub”) is the result of the end of the rapid filling period of the ventricle during the beginning of diastole. In older adults, it often signifies heart failure.
- S_4 (“lub-dub-dub”) coincides with atrial contraction at the end of ventricular diastole. If heard at any other time, the patient may have resistance to ventricular filling.
- The cardiac cycle comprises one complete phase of atrial and ventricular relaxation (diastole), followed by one atrial and ventricular contraction (systole).
- During diastole (normally 520 milliseconds), the left atrium fills passively with blood under the influence of venous pressure, and approximately 80% of ventricular filling occurs.
- In systole (normally 280 milliseconds), ventricular filling is completed, the AV valves snap shut, the two ventricles contract (ventricular systole), and the semilunar valves are forced open. Blood squeezed out of the right ventricle moves into the pulmonary arteries; blood from the left ventricle is pushed into the aorta.
- If the right side of the heart fails, blood backs up behind the right atrium, evidenced by distention of the external jugular veins, and fluid leaks into the surrounding tissues, causing edema in the subcutaneous and internal tissues.
- If the left side of the heart fails, blood backs up behind the left atrium, and fluid is squeezed into the alveoli, producing pulmonary edema, evidenced by dyspnea, bubbling crackles, and frothy sputum.
- Blood pressure is influenced not only by the output of the heart and the volume of blood present in the system, but also by the relative constriction or dilation of arteries, which is controlled by the autonomic nervous system.
- Key parameters associated with the heart’s functioning include cardiac output (the amount of blood that is pumped out by either ventricle), stroke volume (the amount of blood pumped out by either ventricle in a single contraction), and heart rate (the number of cardiac contractions per minute).
- Changes in the heart’s contractility (the degree of contraction of its muscle without changing the stretch on the muscle) may be induced by medications that have a positive or negative inotropic effect.
- The heart can generate its own electrical impulses without stimulation from nerves (automaticity) and has specialized conduction tissue that can rapidly propagate electrical impulses to the muscular tissue of the heart.
- The dominant pacemaker in the heart is the sinoatrial (SA) node; if the right coronary artery is occluded, as in a myocardial infarction (MI), the SA node will become ischemic.
- Depolarization of the heart—the process by which muscle fibers are stimulated to contract—occurs because of an influx of sodium and calcium ions into the myocardial cells. It results in a “wave of depolarization” as the electrical charges pass in all directions from one cell to another.
- Repolarization occurs when the sodium and calcium channels close and potassium channels open; thus, sodium ions move out of the cell and potassium ions flood back in.
- In the absolute refractory period, the heart muscle has been drained of energy; it will not contract during this period. In the relative refractory period, the heart is partially charged, albeit not strongly enough to create a full contraction.
- The electrical conduction events in the heart can be recorded on an ECG as a series of waves and complexes. Depolarization of the atria produces the P wave; as the electrical impulse slows through the atrioventricular (AV) node and AV junction, it can be seen on the ECG as the PR interval; depolarization of the ventricles produces the QRS complex; repolarization of the atria and ventricles produces T waves.

- A U wave may be present after the T wave, although there is no definitive agreement as to what it represents.
- The 12-lead electrocardiogram (ECG) graphically represents electrical activity in cardiac tissue.
- Today's cardiac monitor/defibrillators typically combine continuous 3-lead ECG monitoring, 12-lead electrocardiography, semiautomatic and manual defibrillation, external pacing, capnography, pulse oximetry, blood pressure, end-tidal carbon dioxide, cardiac output, and other capabilities.
- Assessment of the patient's cardiac status—which takes place before monitoring—should include analysis of the cardiac rhythm and 12-lead ECG to determine any underlying electrical disturbances caused by changes in the heart's structure or function as the result of disease, ischemia, injury, or infarction.
- Cardiac monitoring—the continuous observation of the patient's condition in relation to his or her cardiac rhythm—typically uses lead configurations for viewing from one to three leads simultaneously.
- A "gold standard" for 12-lead ECG interpretation is the ability to perform serial evaluations for comparison; however, it is important to use consistent lead placements to ensure that all providers compare "apples to apples."
- The electrode is the sensing device that connects directly to the skin; the lead is the designated position of the electrode (that is, limb and precordial leads). The cable is the physical wire that connects the electrode to the ECG monitor.
- The six limb leads (I, II, III, aVR, aVL, and aVF) provide a view of the heart in a vertical plane (the frontal plane); the six precordial leads (V_1 to V_6), or chest leads, show a horizontal plane. The combination of limb leads and precordial leads provides a three-dimensional view of the heart.
- If the flow of electrical activity proceeds unimpeded toward a positive lead, it appears as a positive deflection on the ECG. If the flow of electrical activity flows away from a positive lead, it is deflected negatively. An electrical impulse that crosses perpendicular to a positive lead is called isoelectric.
- If the heart's electrical impulse is deflected by a change in structure or function, it will be less positive, aberrantly conducted, or deflected away from the selected electrode.
- Placement of additional or nonstandard right-sided heart leads V_4R, V_5R, and V_6R and posterior heart leads V_7, V_8, and V_9 is indicated in suspected right ventricular and posterior MIs and when a patient has clinical findings suggestive of acute coronary ischemia, but the standard 12-lead ECG is normal or nondiagnostic.
- Before running the ECG with right-sided leads, the standard 12-lead ECG should be completed. Then, using the precordial (chest) leads, the additional leads are attached as follows: V_4R, V_5R, V_6R, V_7, V_8, and V_9.
- Confirm placement of leads before transport and several times during transport.
- A "new" 12-lead ECG should be recorded and evaluated before transport and repeated at regular intervals, when any changes in the ECG are noted, and on arrival at the receiving facility.
- The transport of a patient with ST-segment elevation MI to definitive care should not be delayed for the sake of obtaining additional ECGs.
- The supine position is recommended for obtaining ECGs; however, an adequate ECG can be obtained with the patient in a semisitting (semi-Fowler's) position.
- Before obtaining a 12-lead ECG, standardization or calibration of the device at 1 mV = 10 mm high

and 200 milliseconds wide is necessary.

- In the limb leads, the P wave is typically upright in leads I, II, aVL, and aVF. It is frequently biphasic in lead III and is purely negatively deflected in lead aVR.
- In the precordial leads, the P wave is typically upright in leads V_5 and V_6. Lead V_1 is biphasic, and leads V_2 and V_4 are variable.
- The PR interval spans the time from the start of atrial depolarization to the start of ventricular depolarization; it also includes the delay in conduction that occurs at the AV junction. It usually lasts from 120 to 200 milliseconds.
- The QRS complex is a representation of ventricular depolarization. Septal depolarization is not always seen on the ECG; when it is, a small Q wave may be seen in leads I, aVL, V_5, and V_6.
- The keys to being able to quickly read an ECG are experience and consistency. Interpretation of 12-lead ECGs is a skill that must be practiced repeatedly using a standard method of analysis.
- Interpretation of rhythms and arrhythmias is a fundamental building block for ECG analysis.
- The 12-lead ECG shows groupings of complexes that are recorded simultaneously by each lead. The rhythm strip, which typically appears at the bottom of the ECG, records for the entire time the 12-lead ECG is being obtained.
- Each cell in the heart can produce its own electrical impulse (vector), which varies in intensity and direction. The axis is the direction of the wave of depolarization as it passes through the heart.
- The sum of all the electrical impulses is called the mean electrical axis. It should progress in a downward and leftward direction; if there is a change in the patient's normal or preexisting axis, a change has occurred in the structure or function of the heart.
- To calculate the direction and intensity of the electrical axis, the hexaxial system, which relies on a circular diagram oriented in the frontal (coronal) plane, can be used.
- Before transporting a critically ill patient, a baseline ECG should be acquired and analyzed on the cardiac monitor and abnormalities should be noted, including in the mean electrical axis. An axis that deviates outside the normal range or is different from the patient's preexisting axis usually has clinical implications. In such a case, the ECG should be checked for corresponding changes suggestive of the underlying cause of the deviation.
- Cardiac causes of axis deviation include ischemia, infarction, left or right ventricular hypertrophy, ectopic beats and rhythms, dextrocardia, and conduction defect of the left posterior-inferior fascicle of the left bundle branch.
- Other conditions that are not cardiac in origin can also contribute to a shift in axis—for example, anatomy (such as tall, thin stature), young age, chronic obstructive pulmonary disease, obesity, and pregnancy.
- A disruption of the electrical impulse in the right and left atria will result in a change in the shape of the P wave.
- An injury to the tricuspid and mitral valves or disruption of the electrical impulse in the AV node can result in lengthening or shortening of the PR interval.
- Injuries in the bundle of His, the left and right bundle branches, the fascicles, and the Purkinje fibers can result in a bundle or fascicular block.
- With a right bundle branch block (BBB), the representative complex displayed on the ECG includes a prolonged QRS interval of 120 milliseconds or more; a slurred S-wave in leads I and V_6; and the

RSR' pattern in V_1, with the R' wave taller than the R wave.

- There is no specific treatment required for isolated right BBB, but the patient should be seen for periodic follow-up evaluations.
- With a left BBB, the representative complex displayed on the ECG includes a prolonged QRS interval of 120 milliseconds or more; wide R waves in leads I and V_6 with no Q wave; and wide, deep S waves in lead V_1 and possibly a small r wave.
- Left BBB usually means that there is a serious cardiac problem within the conduction system or ischemia from coronary artery disease. Patients with this condition require complete cardiac evaluation.
- A hemiblock is a block in the electrical conduction of one of the two fascicle branches of the left bundle branch. It causes little to no widening of the QRS complexes.
- The criteria for diagnosing left anterior hemiblock are left axis deviation within –30° to –90°; a qR complex or large R wave in lead I; and an rS complex in lead III (likely to also appear in lead II and aVF).
- The criteria for diagnosing left posterior hemiblock are right axis deviation of +90° to 180°; rS wave in lead I and qR wave in lead III; and exclusion of right atrial enlargement and/or right ventricular hypertrophy.
- A bifascicular block (ie, two conduction pathways blocked at the same time) includes concurrent findings of right BBB with left anterior hemiblock or left posterior hemiblock. A new onset of bifascicular block with ischemia is not stable and often deteriorates into a complete heart block, especially in the presence of an acute MI.
- When transporting a critically ill cardiac patient with an existing bifascicular block consisting of right BBB and left anterior hemiblock, if signs of ischemia or infarction develop, the patient will likely need acute pacemaker placement, especially in the presence of second- or third-degree AV block. Medical control should be contacted, the patient should be prepared for external cardiac pacing, and the receiving facility should be notified to be urgently prepared for pacemaker placement.
- A trifascicular block is the combination of a bifascicular block (right BBB with a block in the left anterior or left posterior fascicle) that occurs with a first-degree heart block (prolonged PR interval); its presence after an MI implies extensive cardiac damage. True trifascicular blocks require immediate temporary pacing followed by the placement of a permanent pacemaker.
- Hyperacute T waves that appear as a result of ischemia and infarction appear only in the leads that view the area of ischemia and infarction.
- In addition to presence after an MI, ST-segment elevation is observed with left BBB, ventricular rhythms, left ventricular hypertrophy, pericarditis, and early repolarization. A persistent ST-segment elevation may indicate the formation of a ventricular aneurysm.
- The combination of hypertrophy and dilation means that the heart has a larger muscle mass in the affected area. The larger the muscle mass, the higher the concentration of electrical impulses or vectors.
- Left atrial enlargement produces a P wave with a notched double-humped appearance. The characteristic findings in right atrial enlargement are tall, peaked P waves in the inferior leads II, III, and aVF that have an amplitude of 2.5 mm or more. The tall, peaked P waves are known as P pulmonale.
- Patients with left ventricular hypertrophy (LVH) have a higher risk for coronary artery disease, making

them more susceptible to the sequelae of that disease, and are more susceptible to increased mortality than patients who experience an MI without LVH. Increased R-wave amplitude in the leads that look at the left ventricle forms the basis of ECG diagnosis of LVH.

- Patients with right ventricular hypertrophy exhibit a right shift in axis in leads I and aVF and in precordial leads V_1 and V_2 for increased R-wave deflection.
- When hypertrophy is uncomplicated by ischemia, the changes in QRS complexes are used to make the diagnosis. When subendocardial ischemia is present as well, changes to the ST segment and T wave appear as a strain pattern.
- It is important to distinguish between the strain pattern and the ST-segment and T-wave changes seen in ischemia and infarction because the ST-segment and T-wave changes can be immediately life threatening.
- In people with Wolff-Parkinson-White syndrome (WPW), the bundle of Kent (accessory pathway) does not have the same ability to slowly conduct electrical impulses as it does through the AV node, which puts the person at risk for extremely fast heart rates that cause hemodynamic instability. On the ECG, WPW is signaled by the presence of a slurred upstroke known as a delta wave.
- The two tachyarrhythmias that occur most often in WPW are paroxysmal supraventricular tachycardia and atrial fibrillation. CCTPs should be aware of the potential for a wide complex tachycardia developing that is difficult to distinguish from ventricular tachycardia (VT).
- Treatment of unstable WPW tachyarrhythmias may require electrical cardioversion; management with drug therapy produces unpredictable results.
- Pericarditis (inflammation of the pericardium) may cause ST-segment elevation, T-wave flattening or inversion, and the appearance of low voltage in all leads.
- Long QT syndrome is characterized by a prolonged QT interval on ECG and has a tendency to deteriorate into ventricular tachyarrhythmias, including torsade de pointes, which can lead to syncope and sudden cardiac death. The rhythm is too fast to maintain effective cardiac output, so the patient loses consciousness—often without warning.
- The deterioration of long QT syndrome poses a management challenge for CCTPs. Carefully evaluating the ECG, reviewing the patient's medications, and obtaining a medical history will help provide clues to the diagnosis and alert CCTPs to the possibility of a tachyarrhythmia developing during transport.
- Treatment for torsade de pointes includes correcting the underlying cause, overdrive pacing, and administering magnesium sulfate. Other therapies for patients in unstable condition may include electrical cardioversion and defibrillation.
- Wide complex tachycardia is any cardiac rhythm of more than 100 beats/min with a QRS duration of 120 milliseconds or more. This rhythm can be of ventricular or supraventricular origin; identifying the origin of the arrhythmia can lead to the correct diagnosis and proper therapeutic interventions.
- The most useful ECG criterion in establishing the diagnosis of VT is the presence of AV dissociation with more ventricular than atrial events.
- In a patient with right BBB with a QRS duration of greater than 140 milliseconds, VT is the likely diagnosis. In left BBB, a QRS duration of greater than 160 milliseconds indicates VT.
- If the electrical axis is 180° to –90°, the wide complex tachycardia is not likely to be supraventricular tachycardia (SVT). Electrical concordance across the precordium (all QRS complexes pointing in the same direction in ECG leads V_1 through V_6) is more likely VT and is rarely consistent with SVT.
- Patients with wide complex tachycardia who have low blood pressure, pulmonary edema, severe chest

pain, or other evidence of poor perfusion should be promptly treated using synchronized cardioversion.

- When a patient in wide complex tachycardia remains in hemodynamically stable condition, more time can be spent reviewing the history, physical examination, and ECG findings, to make the correct diagnosis.
- When a patient has wide complex tachycardia of an uncertain cause, yet remains in hemodynamically stable condition, amiodarone (Cordarone) is the drug of choice because it can terminate SVT and VT. It is also useful in patients with a poor ejection fraction.
- Procainamide (Procanbid, Pronestyl, Pronestyl-SR, Procan SR) can be used with caution in a patient with wide complex tachycardia who remains in hemodynamically stable condition, although this drug can cause a sudden drop in blood pressure with rapid IV administration and is contra-indicated in patients with complete heart block.
- Lidocaine (Xylocaine) is useful for VT of ischemic origin but does not terminate SVT.
- If SVT is suspected, adenosine (Adenocard) is a useful and diagnostic medication. When irregular wide complex tachycardia is the result of atrial fibrillation and preexcitation over an accessory pathway, however, adenosine may lead to ventricular fibrillation.
- Following termination of wide complex tachycardia, preventive treatment for maintaining a stable rhythm should be initiated—usually in the form of an antiarrhythmic drug (eg, continuation of the drug that terminated the wide complex tachycardia).
- In patients presenting with a wide complex tachycardia rhythm, BLS is the first step in patient management, closely followed by ACLS practices. If the patient is in hemodynamically unstable condition, wide complex tachycardia should be managed as if it were VT until proven otherwise, with synchronized cardioversion the initial therapy of choice.
- Polymorphic or pulseless VT should be treated as ventricular fibrillation: give immediate defibrillation; secure the airway; provide CPR; perform endotracheal intubation; support ventilations; and administer an antiarrhythmic medication.
- When changes in electrolytes—especially potassium and calcium—occur, their effects are often seen on the ECG.
- Hyperkalemia—the most dangerous of all electrolyte abnormalities—can cause death and prevent some drugs used in resuscitation efforts from being effective. On the ECG, hyperkalemia can cause changes in the appearance of all waveforms, and it can cause virtually any arrhythmia. Rapid recognition and correction of this electrolyte disturbance is essential.
- Hypokalemia does not cause the dramatic changes on the ECG, but rather is associated with mild, nonspecific changes such as ST-segment depression, a slightly decreased amplitude of the T waves, minimal prolongation of the QRS interval, and the presence of a prominent U wave.
- ECG changes associated with hypercalcemia include a shortened ST segment, a shortened QT interval, a prolonged PR interval, and flattened or inverted T waves. A variable degree of heart block may also develop.
- Hypocalcemia that occurs acutely can be the result of medications or surgical effects. It causes the opposite effects of hypercalcemia.
- There is a higher incidence of adverse effects with calcium channel blockers and beta-blockers, but the mortality rate is higher in digoxin toxicity. The margin between toxic and therapeutic doses of digoxin is small.

- At toxic levels, the excessive increase of intracellular calcium associated with digoxin elevates the resting potential and predisposes the heart to arrhythmias. The most common ECG changes in digoxin toxicity include AV, junctional, or ventricular ectopic beats; first-degree AV block; slow ventricular response in atrial fibrillation; and an accelerated AV or junctional rhythm. More severe arrhythmias may include severe bradycardia, high-degree heart blocks, and malignant ventricular rhythms.
- Early recognition and aggressive treatment of digoxin toxicity are lifesaving.
- If one of the coronary arteries becomes blocked, the muscle it supplies will be deprived of oxygen (ischemia). If this oxygen supply is not quickly restored, the ischemic area of heart muscle will eventually die (undergo infarction).
- In atherosclerosis, plaque infiltrates the arterial wall and decreases its elasticity; it also narrows the arterial lumen and interferes with blood flow through the vessel. The narrowed, roughened area of the arterial intima provides a locus for the formation of a fixed blood clot (thrombus) that may obstruct the artery, and calcium may begin to precipitate from the bloodstream into the arterial walls, causing arteriosclerosis.
- Atherosclerosis is rarely the primary cause of medical emergencies but is a major contributor to other conditions that may become medical emergencies.
- Arterial bruits (turbulence or "swishing" sounds heard with a stethoscope placed over the carotid arteries) signal the presence of atherosclerosis and contraindicate the use of carotid sinus massage.
- Other peripheral vascular disorders associated with atherosclerosis include claudication, a severe pain in the calf muscle caused by narrowing of the arteries in this muscle and leading to a painful limp, and phlebitis, inflammation, swelling, and pain along the veins that can lead to the formation of blood clots and thrombophlebitis, which is venous inflammation associated with a thrombus (blood clot).
- The most dangerous complication of peripheral vascular disorders is pulmonary embolism, in which a thromboembolus travels to the heart and through its right side, lodging in the pulmonary arterial tree.
- Signs of peripheral vascular occlusion may include pain, redness, swelling, warmth, and tenderness in the extremity; claudication; and arterial bruits.
- If a blockage or potential embolus is suspected, the CCTP should administer IV heparin, monitor the affected limb via ultrasound or Doppler (if available), and maximize blood flow to the extremity by using warm compresses.
- A patient being transported over a long distance with a blood clot that has the potential to become a pulmonary embolus should have a baseline ECG before transport and must undergo frequent 12-lead ECG monitoring. ST-segment depression in the limb and precordial leads accompanied by an increase in right axis deviation may indicate a pulmonary embolus.
- Acute coronary syndrome comprises any group of clinical symptoms consistent with acute myocardial ischemia. Acute myocardial ischemia typically presents as chest pain as the result of insufficient blood supply to the heart muscle because of coronary artery disease.
- Acute coronary syndrome includes conditions that cause an episode of ischemic discomfort (chest pain) as the result of disruption of plaque within a coronary artery. These substances promote a chain reaction of platelet activation, adhesion, aggregation, thrombin formation, and, ultimately, thrombus formation, with the end product being complete occlusion of the coronary artery or one of its branches.
- The 12-lead ECG is used to categorize two groups of patients who have experienced acute coronary syndrome: patients with ST-segment elevation MI (STEMI) and patients without STEMI (NSTEMI, which is further classified based on the presence or absence of unstable angina).

- A 12-lead ECG should be performed within 10 minutes of contacting a patient with chest discomfort or other signs and symptoms suggestive of STEMI. If the findings are not conclusive, but the patient remains symptomatic and there is have a high degree of suspicion, serial 12-lead ECGs should be performed while maintaining continuous ST-segment monitoring.
- All patients with acute coronary syndrome (ACS) should receive antithrombin and antiplatelet therapy, regardless of the presence or absence of ST-segment elevation. Patients with STEMI are candidates for prompt reperfusion therapy, pharmacologic or catheter based, to restore blood flow in the occluded artery; patients with NSTEMI should receive anti-ischemic therapy and be considered for catheter-based therapy.
- Marked ST-segment depression in leads V_1 through V_4, with tall R waves in the right precordial leads and upright T waves, is suggestive of a true posterior MI. Placement of additional electrodes in the posterior position to form leads V_7, V_8, and V_9 is recommended to confirm the diagnosis because primary percutaneous coronary intervention may be appropriate in patients with true posterior MI.
- Lethal ventricular arrhythmias may develop in patients with STEMI, so continuous ECG monitoring is required.
- Most patients whose ECG displays ST-segment elevation will ultimately have a STEMI.
- In case of ACS, the role of the critical care transport team is to transport the patient from a basic hospital to a cardiac hospital with the least possible decline in the patient's condition and to prevent further loss of heart muscle. If transport times will be long or the degree of blockage is severe to the point of being life threatening, tissue plasminogen activators may be required.
- Angina occurs when the supply of oxygen to the myocardium is insufficient to meet the demand. When obtaining a history from a patient with chest pain, it is important to distinguish between stable angina (follows a recurrent pattern) and unstable angina (follows no pattern).
- In the critical care transport environment, nitroglycerin (Nitro-Bid IV, Tridil) should be given intravenously to patients with stable angina, especially for long transports.
- Unstable angina is characterized by noticeable changes in the frequency, severity, and duration of pain and often occurs without predictable stress. It is often a warning sign of an impending MI.
- An MI occurs as a result of the cardiac muscle being deprived of coronary blood flow long enough for myocardial tissue death to ensue. Causes include narrowed vessels, coronary artery occlusion, spasm of a coronary artery, and reduction of overall blood flow.
- Acute MI is the leading cause of death in the United States. The majority of infarctions involve the left ventricle.
- The infarcted tissue is invariably surrounded by a ring of ischemic tissue—an area that is relatively deprived of oxygen but is still viable. The ischemic tissue is often the source of cardiac arrhythmias.
- The three initial components for the diagnosis of an MI are history and physical examination, testing for cardiac enzymes, and interpretation of ECG changes associated with an MI.
- Biomarkers for MI include troponin I, troponin T, creatine kinase, and myoglobin; all of these biomarkers increase in the setting of MI.
- Increased oxygen demand caused by an increased workload or a decrease in supply caused by coronary artery disease or a blocked artery can lead to ischemia, injury, and, ultimately, infarction. Ischemia and injury are reversible, and rapid intervention when they occur can often prevent infarction.
- On the ECG, findings associated with ischemia include ST-segment depression and an inverted T wave. With MI, the T wave inverts symmetrically; when other causes (such as ventricular hypertrophy

with strain and BBB and cerebral hemorrhage) are to blame, the T-wave inversion is often asymmetric or "slurred."

- If ischemia continues uninterrupted, the injured area does not repolarize completely, leading to an elevated ST segment.
- If ischemia and injury continue uninterrupted, infarction can occur; the infarcted tissue no longer generates or transmits electrical impulses. Thus, no direct wave is recorded on the ECG, and electrical impulses are deflected away from the area of infarct and are represented by Q waves.
- For Q waves to be significant (ie, indicating pathology), they must be more than one third the total height of the QRS they appear with and wider than 30 milliseconds in duration.
- The prognosis and therapeutic implications for a patient who has had an MI are determined largely by which area of the heart has died.
- Q waves from old infarctions can be present with findings of a new MI and can contribute to making the correct diagnosis from the ECG more challenging. For this reason, CCTPs should review an "old" ECG from the patient's chart, acquire a new 12-lead ECG before transport, and make serial evaluations along the way.
- In anterior wall MI, the normal pattern of R-wave progression may not occur, a phenomenon called poor R-wave progression.
- An anteroseptal MI is common with an occlusion of the left anterior descending artery and is often associated with hemodynamic compromise and cardiogenic shock. This pattern of MI may be identified by analyzing the septal leads V_1 and V_2 and the anterior leads V_3 and V_4.
- In an acute anteroseptal MI with lateral extension, changes can be seen in all precordial leads (specifically V_5 and V_6 for the lateral extension) and in leads I and aVL; there is not always significant Q-wave formation, but reciprocal changes can be found in leads II, III, and aVF.
- Hyperacute T-wave changes occur only during the first 15 to 30 minutes of an acute MI. If these changes are found during transport, the CCTP is in a unique position to initiate care, even if it is only early notification of the receiving facility to decrease the door-to-treatment time.
- In an acute lateral wall MI, ECG changes are often seen in the lateral leads I, aVL, V_5, and V_6. Reciprocal changes may be seen in the inferior leads II, III, and aVF.
- With an inferior wall MI, characteristic ECG changes can be seen in inferior leads II, III, and aVF. Reciprocal changes are seen as ST-segment depression in leads I and aVL unless the high lateral wall is included in the infarction.
- Without signs of acute infarction, pathologic Q waves are considered age indeterminate because without a good patient history, there is no other way to tell how old the patient is. In an acute inferolateral MI, ECG changes are seen in inferior leads II, III, and aVF and in lateral leads I, aVL, V_5, and V_6. ST-segment changes will be found in leads V_2 to V_4 if the infarct extends anteriorly, and classic ST-segment and T-wave changes will be evident in the lateral precordial leads V_5 and V_6.
- In an acute apical MI, direct ECG changes can be found in inferior leads II, III, and aVF; lateral leads I and aVL; and precordial leads V_2 through V_6. Because of the diffuse ECG changes, this type of MI can be confused with pericarditis. Right ventricular infarction is strongly associated with an inferior wall MI, so when it is present, CCTPs should keep a high index of suspicion and check for other criteria. Right ventricular infarction should be suspected with ST-segment elevation in (septal) lead V_1 and (inferior) leads II, III, and aVF.

- With a posterior wall MI, the diagnosis is made by looking for reciprocal changes in leads V_1 and V_2—specifically, ST-segment depression, upright T waves, and tall R waves.
- When there is reciprocal evidence of a posterior wall MI, there should also be a high index of suspicion, and posterior leads (V_7, V_8, and V_9) should be recorded for comparison.
- Patients with cardiomyopathy may have shortness of breath, chest pains, palpitations, or syncope; sudden cardiac death is also possible.
- Congestive heart failure occurs when the heart cannot pump powerfully enough or fast enough to empty its chambers; as a result, blood backs up into the systemic circuit, the pulmonary circuit, or both.
- In left-sided heart failure, the left side of the heart is no longer able to pump the blood being delivered from the pulmonary vessels. Blood backs up behind the left ventricle, the pressure in the left atrium and pulmonary veins increases, and serum is forced out of the pulmonary capillaries and into the alveoli.
- Pulmonary edema occurs as the result of reduced ejection fraction and cardiac output of the left ventricle when serum mixes with air in the alveoli.
- The priorities when managing left-sided heart failure are to remove the fluid from the lungs, reduce afterload, increase oxygen supply to the heart, and decrease myocardial oxygen demands.
- Right-sided heart failure most commonly occurs as a result of left-sided heart failure, but may also occur as a result of pulmonary embolism or long-standing chronic obstructive pulmonary disease, especially chronic bronchitis.
- The edema associated with right-sided heart failure is most likely to be visible in dependent parts of the body (eg, the feet in a person who is sitting or standing or the lower back in a bedridden patient). It is also present in parts of the body that are not visible, such as the liver.
- Electrophysiology studies are not done during transport, but in the hospital setting may be performed to assess electrical activity of the heart.
- The primary in-hospital arrhythmia therapies are catheter ablation, pharmacologic treatment, and implantable devices, such as pacemakers and implantable cardioverter-defibrillators.
- The transcutaneous pacemaker is most commonly used as an initial emergency pacer (a temporary measure). It is the quickest and most convenient method of pacing, but also requires the most energy and is the least reliable owing to the quality of the skin-to-pad contact and the thoracic impedance that must be overcome.
- Many patients are transported between medical facilities with transvenous pacemakers accompanied by an external pulse generator. Generally, these transvenous pacers require little attention and are quite reliable; the primary sources of problems are battery failure and lead disconnection at the generator. Voltage (the force that causes current to flow) and impedance (resistance to current) are important determinants of battery longevity in a pacemaker.
- The main sources of resistance in a pacing circuit are the lead conductor, the electrode, and the concentration of electrically charged ions at the electrode-tissue interface. Electrode resistance improves pacing efficiency; lead conductor resistance and polarization do not.
- Passage of the pacemaker's electrical impulse between the cathode and the anode—through cardiac tissue and body fluids—is the event in the pacing circuit that stimulates cardiac depolarization.
- Pacemakers stimulate cardiac depolarization, sense intrinsic cardiac activity, respond to metabolic needs, and store diagnostic information. All pacemakers can provide fixed-rate pacing; most also provide rate-adaptive (rate-responsive) pacing, increasing and decreasing pacing rate in response to

input from rate sensors.

- Endocardial (transvenous) leads are threaded through veins—usually subclavian or cephalic—to the right atrium or ventricle. Epicardial leads are attached to the external surface of the heart with sutures or another fixation device.
- Steroid-eluting electrodes have a silicone plug that contains a small dose of steroid, which reduces inflammation and the growth of fibrous tissue at the electrode site.
- Pacemaker system malfunction may occur as a result of battery failure, problems at the lead electrode-tissue interface, coiling or damage to the lead-generator interface, insulation failure of the lead wires, dislodgement of the lead at the implantation site (very rare), pacemaker programming, pocket stimulation, diaphragmatic stimulation, or electromagnetic interference.
- If pacemaker failure occurs during transport, the cardiac monitor will show failure to capture a signal; a 12-lead ECG (compared with the patient's pulse) and patient assessment (signs of decompensation) may then be used to confirm the pacing failure.
- Pacing in the ventricular pacer mode and loss of AV synchrony in a single-chamber pacemaker can lead to pacemaker syndrome, which results from hemodynamic deterioration and causes nonphysiologic timing of atrial and ventricular contractions.
- Atriobiventricular pacemakers, which coordinate the right and left ventricles of the heart, may be used as a treatment for congestive heart failure to resynchronize the heart's chambers.
- Implantable cardioverter-defibrillators (ICDs) automatically attempt to convert a fibrillating heart into a sinus rhythm using a shock from voltage. Each patient with an ICD has a custom ICD system consisting of a housing (can) and a variable number of pacing and high-voltage leads.
- Most ICDs are implanted with one or two transvenous leads used for pacing and shocking. The leads enter the venous system through the subclavian or cephalic vein for placement into the right ventricle or the superior vena cava.
- An ICD bases its decision to deliver a shock (to treat tachyarrhythmias) on the ventricular rate alone; thus, atrial arrhythmias and electromagnetic interference can lead to inappropriate shocks.
- If an ICD fires during care of a patient, the provider should gather the following information: the number of times shocked, recent medical procedures, recent changes in medical condition or changes in patient adherence to the medication regimen, presence of a coexisting pacemaker, patient's activity at the time of shock, and proximity to water or an electrical source.
- If a patient with an ICD experiences multiple inappropriate defibrillator discharges as the result of defibrillator malfunction, the CD can be temporarily disabled by use of a magnet to prevent further discharges.
- In a cardiac arrest, a CCTP can deliver external defibrillation to a patient with an ICD, although the paddles or pads should not be placed directly over the ICD. Providers can also perform CPR on a patient with an ICD.
- Significant risks associated with atrial fibrillation are thromboembolism and resulting increased incidence of stroke. Methods to convert atrial fibrillation include antiarrhythmic medications, external cardioversion, and (the preferred method) internal cardioversion and/or antitachycardia pacing with an atrial ICD.
- CCTPs should expect to transport patients who have or need a pacemaker or ICD and should be prepared to manage any adverse situations related to either device. They should receive training on any transthoracic or epicardial, transcutaneous, or transvenous pacemaker device that their agencies use

and should be prepared to use a magnet to temporarily reprogram an implanted pacemaker or ICD if their agencies transport critically ill cardiac patients.

- When accepting a patient from a sending hospital for critical care transport, any patient with a transthoracic or epicardial, transvenous, transcutaneous, or implanted pacemaker device must have electrical and mechanical pacemaker capture assessed and confirmed by a CCTP.
- Switching from a device used in a sending facility to a device used by the transport team is often the source of anxiety for CCTPs and other providers. If the CCTP does not have a compatible device available, it may be worth considering using the device from the sending facility for the transport and returning it promptly after the transport has been completed.
- The presence of an ICD would require an alternative site (not over the device) for transcutaneous pacing or defibrillation if either of those measures is needed during transport. It is essential to have an external cardioverter-defibrillator immediately available when transporting a patient with an ICD.
- Electromagnetic interference is the major cause of pacemaker dysfunction, particularly in older-style pacemakers lacking adequate internal shielding; they may be vulnerable to interference from headsets and intercom systems in air transport.
- Rate-adaptive pacemakers appear to be sensitive to helicopter vibrations—a factor that CCTPs working on medical helicopters should take into consideration.
- During patient turnover, high-risk items such as endotracheal tube placement, central line status, IV sites for vasoactive medications, and the presence of mechanical pacemaker capture should be specifically confirmed with the accepting provider.

Vital Vocabulary

ablation Removal of a pathway or function by electrocautery or radiofrequency.

absolute refractory period The early phase of cardiac repolarization, wherein the heart muscle cannot be stimulated to depolarize.

acute coronary syndrome (ACS) The term used to describe any group of clinical symptoms consistent with acute myocardial ischemia.

anode The electrode in a pacing circuit that is positively charged when current is flowing.

antegrade conduction Conduction in the normal direction between cardiac structures.

antiarrhythmic A type of medication used to treat and prevent cardiac rhythm disorders.

bifascicular block The combination of a right bundle branch block and a block of one of the fascicles of the left bundle, the left anterior or left posterior fascicle.

biphasic A term used to describe a wave with negative and positive components; usually used in conjunction with P and T waves.

bipolar lead A conduction lead comprising two electrodes attached at specific body sites with different polarity used to examine electrical activity by monitoring changes in the electrical potential between them.

bipolar system A closed system consisting of bipolar leads and a module to generate impulses and measure response.

cable The physical wire that connects the electrode to the ECG monitor.

cardiac electrophysiology The cardiac specialty that involves evaluation and management of rhythm

disturbances.

cardiac monitoring The continuous observation of the patient's condition in relation to his or her cardiac rhythm.

cathode The electrode in a pacing circuit that is negatively charged when current is flowing.

chronotropic A type of medication that affects the rate of contraction of the heart.

current The movement of electrons through an electrical circuit over time, measured in amperes.

delta wave The slurring of the upstroke of the first part of the QRS complex that occurs in Wolff-Parkinson-White syndrome.

depolarization The process of discharging resting cardiac muscle fibers by an electrical impulse that causes them to contract.

dextrocardia A congenital cause of right axis deviation, in which the heart develops in a right-facing position, creating a mirror image of the normal left-facing heart.

digoxin A cardiac glycoside that produces positive inotropic and negative chronotropic activity in the heart and is primarily indicated in the treatment of chronic heart failure and to control the ventricular rate in atrial tachyarrhythmias.

discordant A term that describes T waves that are in the opposite direction from the terminal portion of the QRS complex in bundle branch blocks.

dromotropic Affecting the velocity of conduction.

dual-chamber pacemaker An artificial pacemaker with two leads (one in the atrium and one in the ventricle) so electromechanical synchrony can be achieved.

electrical alternans An ECG pattern in which the QRS vector changes with each heartbeat. This pattern is pathognomonic of cardiac tamponade.

electrode In the context of a 12-lead ECG, an electrical sensor placed on the chest to record the bioelectrical activity of the heart. In the context of a pacemaker, a conductor in contact with cardiac tissue at the end of a pacing lead; it delivers impulses to that tissue.

endocardial (transvenous) leads Pacemaker leads guided by angiography and attached to the endocardium.

epicardial leads Pacemaker leads attached to the epicardium (outer surface of the myocardium); placement and troubleshooting are done in the hospital and require surgery and anesthesia.

hemiblocks Blocks of one of the fascicles of the left bundle branch, the left anterior or left posterior fascicle.

hexaxial system The system developed to describe the coronal plane that is created by the limb leads (I, II, III, aVR, aVL, and aVF).

hypercalcemia An increased level of calcium in the blood.

hyperkalemia An increased level of potassium in the blood.

hypertrophy An increase in the size of the cells as the result of synthesis of more subcellular components, leading to an increase in tissue and organ size.

hypocalcemia A low level of calcium in the blood.

hypokalemia A low blood serum potassium level.

impedance Resistance to the flow of current along an electrical pathway, measured in ohms.

implantable cardioverter-defibrillator (ICD) A small, battery-powered electrical impulse generator that is implanted in patients at risk for sudden cardiac death as the result of ventricular fibrillation or pulseless ventricular tachycardia.

implantable pulse generator (IPG) The largest implanted element in a pacemaking system, containing the battery and control circuitry.

inotropic An effect on the contractility of muscle tissue, especially cardiac muscle.

intra-atrial conduction delay Delayed conduction within one of the atria, often associated with left or right atrial enlargement.

intra-atrial pathways The anterior or Bachman bundle, middle bundle, and posterior internodal system, through which the electrical impulse passes after the SA node; represented by the P wave on the ECG; also called intranodal pathways.

isoelectric When referring to a wave, the wave is neither positive nor negative.

joule A measurement of energy.

lead In the context of the 12-lead ECG, the designated position of the electrode, or the name of the electrode placement. In the context of a pacemaker, an insulated wire that carries signals in a pacemaking system between the implantable pulse generator and the heart tissue.

left anterior fascicle The portion of the electrical conduction system responsible for innervating the anterior and superior areas of the left ventricle. It is a single-stranded cord terminating in the Purkinje cells.

left posterior fascicle The portion of the electrical conduction system responsible for innervating the posterior and inferior areas of the left ventricle. It is a widely distributed, fanlike structure terminating in the Purkinje cells.

limb leads The ECG lead electrodes attached to the limbs that form the hexaxial system, dividing the heart along a coronal plane into the anterior and posterior segments.

long QT syndrome A prolonged QT interval on the ECG primarily caused by a congenital disorder and that under certain conditions tends to deteriorate into ventricular tachyarrhythmias and can lead to syncope or sudden cardiac death; the patient loses consciousness, often without warning.

mean electrical axis The sum of all electrical impulses.

NBG code A code of five letters used to categorize pacemakers by their functions and capabilities, developed by a joint effort of North American and British electrophysiology groups.

Ohm's law The principle given by the equation $V = IR$, which states that applied voltage is equal to the current times the resistance of the circuit.

pacemaker syndrome The occurrence of symptoms relating to the loss of atrioventricular synchrony in ventricularly paced hearts or symptoms caused by inadequate timing and ventricular contractions in paced hearts.

pacing circuit The conduction pathway along which the pacing impulse flows; formed by a power source, one or two lead-electrode pairs, and body tissue.

pacing impulse The electrical impulse sent to the heart to stimulate the heart to beat.

paroxysmal nocturnal dyspnea Severe shortness of breath occurring at night after several hours of recumbency, during which fluid pools in the lungs; the person is forced to sit up to breathe; caused by left-sided heart failure or decompensation of chronic obstructive pulmonary disease.

paroxysmal supraventricular tachycardia A supraventricular tachycardia that starts and ends abruptly.

pericarditis An inflammatory process involving the pericardium.

P mitrale A double-humped, M-shaped P wave that is 120 milliseconds wide or greater with the tops of the humps 40 milliseconds apart or greater; found in limb leads I, II, and III; represents left atrial enlargement.

poor R-wave progression An abnormal R-wave pattern; one of the factors that may signify anterior infarction.

P pulmonale A tall P wave that is 2.5 mm high or greater, found in leads II and III; indicates right atrial enlargement.

precordial leads Another term used to describe the chest leads in an ECG.

PR interval The interval of time that occupies the space between the beginning of the P wave and the beginning of the QRS complex.

QRS complex Deflections in the ECG produced by ventricular depolarization.

reciprocal changes An ECG pattern in which a lead shows a pattern that is the opposite of the one shown in the lead located 180° from the other; for example, the electrode over the area of infarction records ST-segment elevation, whereas the electrode over the lead that is 180° away records ST-segment depression.

refractory period A short period immediately after depolarization in which the myocytes are not yet repolarized and are unable to fire or conduct an impulse.

relative refractory period The period in the cell-firing cycle at which it is possible but difficult to restimulate the cell to fire another impulse.

repolarization A state in which the cell becomes more negative, moving away from equilibrium with the extracellular fluid; this is an active process.

single-chamber demand pacemaker A pacemaker with the pacing lead placed in only one chamber of the heart, in which the generator stimulus is inhibited by a signal derived from the heart's depolarization, thus minimizing the risk of pacemaker-induced fibrillation.

ST-elevation myocardial infarction (STEMI) A myocardial infarction that shows ST-segment elevation on the ECG; patients with STEMI have a high probability of coronary thrombus occlusion.

strain pattern An ECG pattern that involves ST-segment changes and flipped, asymmetric T waves associated with right or left ventricular hypertrophy.

ST segment The section of the ECG complex from the end of the QRS complex to the beginning of the T wave; represents the period of inactivity between ventricular depolarization and repolarization; mechanically, represents the time that the myocardium is maintaining contraction.

torsade de pointes An undulating sinusoidal rhythm in which the axis of the QRS complexes changes from positive to negative and back in a haphazard manner.

trifascicular block The combination of bifascicular block (right bundle branch block with a block in the left anterior fascicle or left posterior fascicle) that occurs with a first-degree heart block (prolonged PR interval).

T wave The upright, flat, or inverted wave following the QRS complex of the ECG, representing ventricular repolarization.

unipolar lead A lead in which one of the electrodes is placed in the heart and the other lead is placed in

an area of zero potential.

unipolar system A type of pacemaker system in which contact between the pacemaker itself and the body tissue actually forms the ground lead for the implantable pulse generator.

voltage The force that causes current to flow in a circuit, measured in volts; also called *amplitude* in a pacing system.

wide complex tachycardia A cardiac rhythm of greater than 100 beats/min with a QRS duration of 120 milliseconds or greater; can be of ventricular or supraventricular origin.

Wolff-Parkinson-White (WPW) syndrome A syndrome characterized by short PR intervals, delta waves, nonspecific ST-T wave changes, and paroxysmal episodes of tachycardia caused by the presence of an accessory pathway.

Bibliography

American Heart Association. Devices that may interfere with pacemakers. Available at: http://www.americanheart.org/presenter.jhtml?identifier=32. Accessed April 19, 2009.

Bocka JJ. External pacemakers. *emedicine.* 2008. Available at: http://emedicine.medscape.com/article/780639-overview. Accessed April 19, 2009.

Brown DW, Croft JB, Giles WH, Anda RF, Mensah GA. Epidemiology of pacemaker procedures among Medicare enrollees in 1990, 1995, and 2000. *Am J Cardiol.* 2005;95:409–411.

Cevik C, Perez-Verdia A, Nugent K. Implantable cardioverter defibrillators and their role in heart failure progression [published online ahead of print April 8, 2009]. *Europace.* 2009;11:710–715. doi:10.1093.

De Rotte AA, Van Der Kemp P. Electromagnetic interference in pacemakers in single-engine fixed wing aircraft: a European perspective. *Aviat Space Environ Med.* 2002;73:179–183.

Ellenbogen KA, Wood MA, eds. *Cardiac Pacing and ICDs,* 5th ed. Malden, MA: Blackwell Publishing; 2008.

Ellenbogen KA, Wilkoff BL, Kay N, Lau CP. *Clinical Cardiac Pacing, Defibrillation and Resynchronization Therapy.* Philadelphia, PA: WB Saunders; 2007.

Essebag V, Halabi AR, Churchill-Smith M, Lutchmedial S. Air medical transport of cardiac patients. *Chest.* 2003;124:1937–1945.

French RS, Tillman JG. Pacemaker function during helicopter transport. *Ann Emerg Med.* 1989;18:305–307.

Fromm RE Jr, Taylor DH, Cronin L, McCallum WBG, Levine RL. The incidence of pacemaker dysfunction during helicopter air medical transport. *Am J Emerg Med.* 1992;19:333–335.

Furman S, Hayes DL, Holmes DR. *A Practice of Cardiac Pacing,* 3rd ed. Mount Kisco, NY: Futura Publishing; 1993.

Garcia T, Holtz N. *12-Lead ECG: The Art of Interpretation.* Sudbury, MA: Jones and Bartlett Publishers; 2001.

Garcia T, Holtz N. *Introduction to 12-Lead ECG: The Art of Interpretation.* Sudbury, MA: Jones and Bartlett Publishers; 2003.

Garcia T, Miller G. *Arrhythmias: The Art of Interpretation.* Sudbury, MA: Jones and Bartlett Publishers; 2004.

Hayes DL, Lloyd MA, Friedman PA. *Cardiac Pacing and Defibrillation: A Clinical Approach.* Elmsford, NY: Blackwell Publishing, Futura Division; 2000.

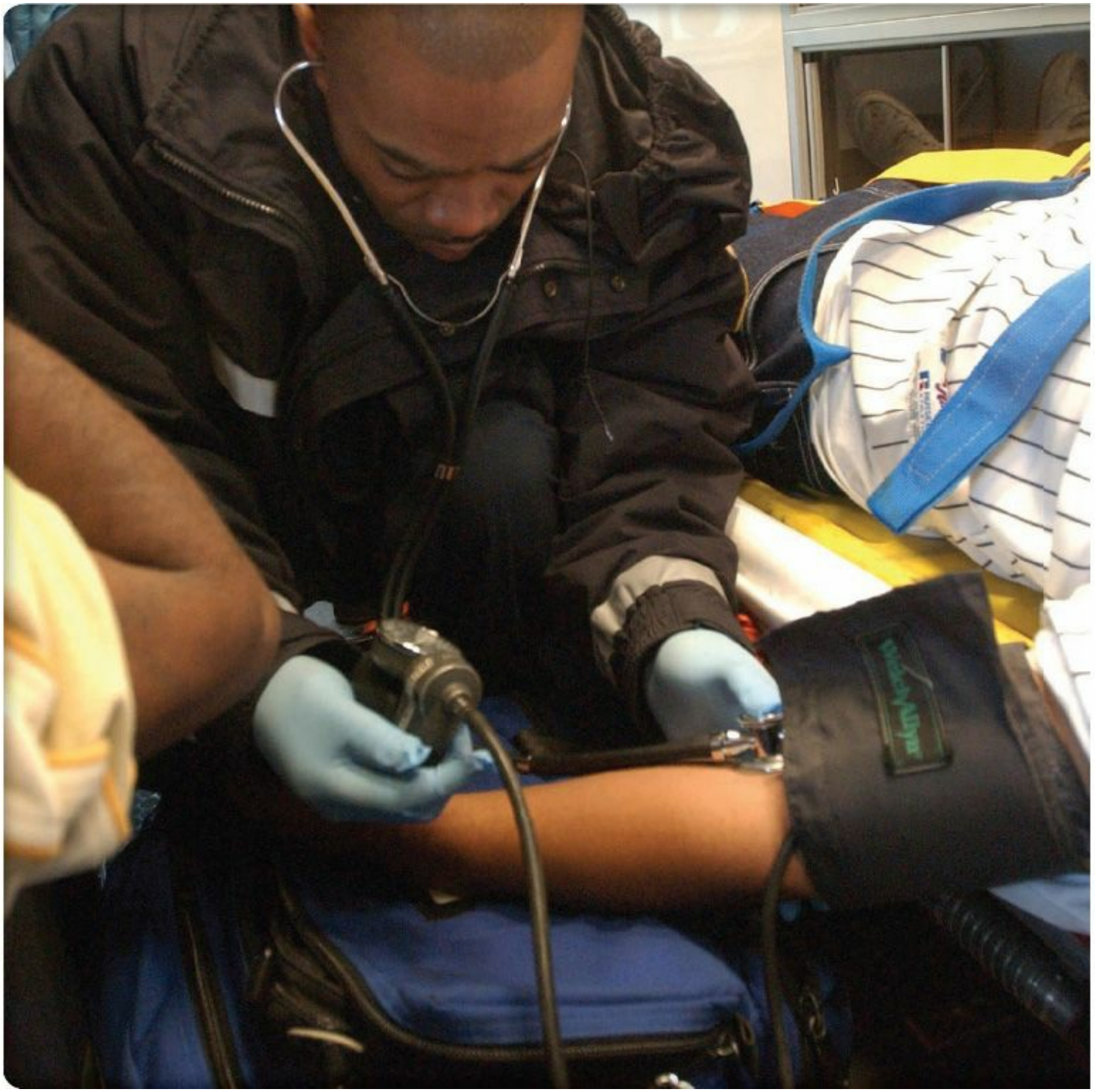
Otto LA, Aufderheide TP. Evaluation of ST segment elevation criteria for the prehospital electrocardiographic diagnosis of acute myocardial infarction. *Ann Emerg Med.* 1994;23:17–23.

Ruskin JN, Camm AJ, Zipes DP, Hallstrom AP, McGrory-Usset ME. Implantable cardioverter defibrillator utilization based on discharge diagnoses from Medicare and managed care patients. *J Cardiovasc Electrophysiol.* 2001;13:38–43.

Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol.* 1996;28:1328–1428.

Silverman BG, Gross TP, Kaczmarek RG, Hamilton P, Hamburger S. The epidemiology of pacemaker implantation in the United States. *Public Health Rep.* 1995;110:42–46.

Vasilyadis M. Pacemaker and automatic internal cardiac defibrillator. *emedicine.* 2006. Available at: https://emedicine.medscape.com/article/780825-overview. Accessed April 18, 2009.



Hemodynamic Monitoring

Authors

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Christopher Handley, BS, EMT-P

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Objectives

1. Discuss the principles of and indications for invasive hemodynamic monitoring (p 563–564).
2. Interpret the following hemodynamic values and discuss their meaning:
   - Central venous pressure (CVP)
   - Systolic blood pressure (SBP)
   - Diastolic blood pressure (DBP)
   - Mean arterial pressure (MAP) Pulse pressure
   - Pulmonary artery pressures (systolic, diastolic, and mean)
   - Pulmonary capillary wedge pressure (PCWP)
   - Cardiac output (CO)
   - Cardiac index
   - Pulmonary vascular resistance (PVR)
   - Systemic vascular resistance (SVR)
   - Stroke volume (SV)
   - Mixed venous saturation (Svo_2) (p 576–583)
3. List normal hemodynamic values for the various types of invasive hemodynamic measurements (p 576).
4. Describe the significance of each pressure reading used in patient management (p 576–583).
5. Describe how a central venous line works and list the sites where it may be placed (p 564–570).
6. Describe the types of catheters used for central venous lines (p 565–566).
7. Discuss indications, contraindications, and complications for central venous lines (p 564–565).
8. Describe the equipment and steps for inserting a central venous line (p 566–570).
9. Explain how to troubleshoot problems encountered when inserting a central venous line (p 569).
10. Describe how an arterial line works and list the sites where it may be placed (p 570).
11. Describe the types of catheters used for arterial lines (p 573–574).
12. Discuss the indications, contraindications, and complications for arterial lines (p 570).
13. Describe the equipment and steps for inserting an arterial line (p 571).
14. Discuss the concepts of preload, afterload, and the Frank-Starling curve (p 577, 581).
15. Demonstrate the proper placement of and zeroing procedure for a pressure transducer (p 584–585).
16. Describe proper transport procedures for invasive hemodynamic monitoring (p 586).
17. Describe troubleshooting procedures for common problems with invasive hemodynamic monitoring

during transport (p 587).

18. Discuss flight considerations related to invasive hemodynamic monitoring (p 588).

Introduction

Two major developments led to the advancement of the intensive care unit (ICU) in the last 40 years. First, in the late 1960s, the value of delivering mechanically assisted ventilation was discovered. Second, in the 1970s, the introduction of the bedside pulmonary artery catheter (PAC) revolutionized hemodynamic monitoring in the ICU. These catheters are used not only in the ICU for hemodynamic monitoring, but also during ground and air transport by CCTPs.

Invasive hemodynamic monitoring methods attempt to assess the physiologic condition of the three principle components of the cardiovascular system: heart, vascular network, and fluid volume. The core hemodynamic assessments center on the capability of the patient's heart to pump the requisite amount of blood to the body. Other hemodynamic assessments allow CCTPs to ascertain the compliance, tone, and resistance of the vascular network. Further assessments can be performed to gauge the fluid status of the patient.

Invasive hemodynamic monitoring is one of the cornerstones of care for critically ill patients. Assessment of a patient's hemodynamic status builds on patient monitoring. The CCTP must be familiar with these critical care assessments to provide adequate interhospital care. Hemodynamic monitoring can aid CCTPs in assessing the severity of a patient's condition, the benefits of blood product and vasoactive drug administration, the effectiveness of ventricular-assist devices and intra-aortic balloon pump support, and, most important, the early warning signs of possible deterioration in a patient's condition.

Invasive hemodynamic measurements are obtained via a closed catheter system **Figure 14-1**. A bag of crystalloid fluid is placed within a pressure bag that assists the flow of fluid through the system's catheter as it passes through a stopcock and a transducer, ultimately ending up in the desired location in the body. Once the catheter is in place, a cable is connected to the catheter's transducer and to the monitor, which reads the respective waveform and displays the appropriate measurement.

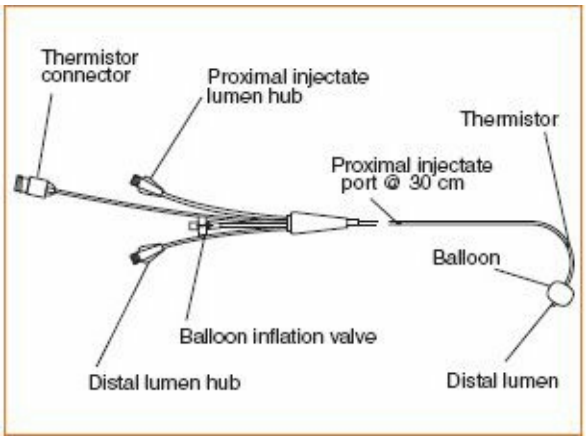


Figure 14-1 A closed catheter system. Courtesy of Edwards Lifesciences. Used with permission.

Invasive hemodynamic monitoring is not a panacea for patient assessment because the catheters are associated with line infection, sepsis, thrombophlebitis, thromboembolism, and associated tissue injury. Measurements may also be misread from inadvertent patient repositioning; moving the patient will produce incorrect measurements unless the equipment is also repositioned appropriately. Physicians typically place these invasive catheters in patients with a diagnosis of shock, pulmonary edema, complicated myocardial infarction, sepsis, pulmonary embolism, diagnostic monitoring, or major cardiac surgery. Catheter placement is usually outside the scope of CCTPs.

Asepsis

Asepsis is an element of care for patients with central lines and hemodynamic monitoring equipment that cannot be overemphasized. The incidence of catheter-related bloodstream infections is a prime focus of critical care practitioners worldwide. Because central venous catheters disrupt the integrity of the skin, they provide a pathway for bacteria and fungi to enter the bloodstream. The mortality of bloodstream infections in critically ill patients approaches 20%, and most of these infections are attributed to the use of central venous catheters. There are five key practices that have been shown to decrease the incidence of catheter-related bloodstream infections: hand hygiene, maximal barrier precautions during insertion, chlorhexidine skin antisepsis, optimal catheter site selection, and prompt removal of unnecessary lines.

The CCTP should be certain to carry and frequently use an alcohol-based hand gel during transport and to carefully disinfect access sites used on patients. For line placement, skin should be prepared with antiseptic-detergent 2% chlorhexidine in 70% isopropyl alcohol using a back-and-forth friction scrub of at least 30 seconds. The antiseptic solution should not be wiped off or blotted from the skin and must be allowed to dry completely (for approximately 2 minutes) before puncturing the site. Older antiseptic solutions, such as povidone-iodine solution, are no longer considered effective skin antisepsis.

Noninvasive Hemodynamic Monitoring

Understanding hemodynamic monitoring begins with noninvasive and less invasive techniques of measurement. In fact, as technology evolves, less invasive devices will most likely replace many of the invasive monitoring devices that CCTPs have become accustomed to using. Physical assessment techniques, such as capillary refill, skin color, skin turgor, skin temperature, lung sounds, heart sounds, and mental status, should coincide with high-tech invasive hemodynamic measurements. Although invasive monitoring devices are designed and used to provide data not available from physical assessment alone, CCTPs should never be so caught up in the technology and complicated measurements that they lose sight of basic physical and cardiovascular assessments.

Blood pressure is one of the most often used and perhaps least understood assessments. Direct monitoring of blood pressure is accomplished by the use of an arterial line. Indirect monitoring of blood pressure is accomplished with a device that senses pulsatile flow, most commonly using a sphygmomanometer (that is, blood pressure cuff) to measure the systolic and diastolic blood pressures. **Systolic blood pressure (SBP)** is the measure of pressure on the arterial walls during systole, whereas **diastolic blood pressure (DBP)** is the measure of the pressure on the walls of the arteries during diastole. The formula for blood pressure is cardiac output (CO) multiplied by systemic vascular resistance (SVR) or BP = CO × SVR. Other methods that indirectly measure blood pressure include assessment of pulses, electronic blood pressure cuffs (also called noninvasive blood pressure [NIBP] units), and Doppler ultrasound machines. It is extremely important to note that these indirect measurements assess flow, whereas direct monitoring devices (that is, arterial lines) measure pressure. Because pressure and flow are entirely different parameters, readings obtained by indirect and direct methods cannot be expected to correlate exactly.

When using indirect monitoring devices, CCTPs should be careful to avoid errors in measurement. Blood pressure cuff size and position of the extremity during measurement are the two most common factors contributing to inaccuracies. The cuff bladder size should be matched to the size of the patient's extremity where blood pressure is measured. Determining the bladder size is often difficult in the critical care setting; many practitioners instead choose a cuff that covers two thirds of the extremity being measured. A cuff selected for an upper arm, for example, would be properly sized if it covered two thirds of the distance from the elbow to the shoulder. Using a cuff that is too large will result in readings that are lower than actual; a cuff that is too small will yield readings higher than actual. The significant potential

for error mandates that the transport unit carry various sizes of BP cuffs appropriate for the ages and sizes of the patient population most commonly transported.

Positioning of the extremity in which BP is measured also affects accuracy. Proper assessment requires that the extremity be at mid-heart level when BP is measured. Elevation of an arm (or leg, if that is where the cuff is placed) above mid-heart level will yield falsely low readings, whereas placing the extremity below mid-heart level will yield false high readings. This issue can be particularly problematic when measuring BPs in side-lying patients.

Often, NIBP units are incorporated into transport monitors and used to obtain indirect BP measurement at predetermined intervals automatically. The NIBP units measure heart rate and mean arterial pressure (MAP) only. The NIBP computer software then calculates the patient SBP and DBP values by using the measured parameters to determine the time spent in systole and diastole computed against the MAP. The CCTP should evaluate the heart rate obtained in every NIBP reading against the actual patient pulse, the heart rate displayed on a pulse oximeter, or an electrocardiographic (ECG) heart rate display. If the NIBP-evaluated heart rate differs significantly from the actual patient heart rate, the calculated SBP and DPB should be considered unreliable. Because MAP is actually measured by the NIBP unit, this value is often used in making patient care and treatment decisions.

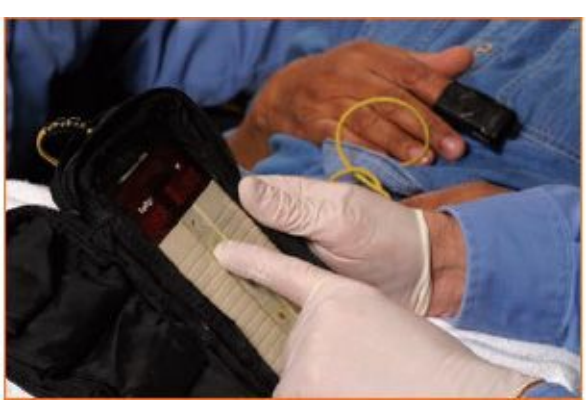


Figure 14-2 Assessment of the patient's heart rate using a pulse oximeter.

Some textbooks suggest that pulses correlate to SBP. The presence of a carotid pulse suggests an SBP of greater than 60 mm Hg, a femoral pulse suggests an SBP of greater than 70 mm Hg, and a radial pulse suggests an SBP of greater than 80 mm Hg. More recent studies failed to find any reliable correlation between BP and the location of pulses. There are data to suggest that skilled critical care clinicians can become fairly accurate at estimating the SBP from palpation of a femoral pulse.

Heart rate is a key sign of hemodynamic instability in emergency and critical care patients. Assessment of the pulse can be performed by arterial palpation; however, in the high noise environment of an ICU or a CCTP vehicle, the heart rate obtained from a pulse oximeter is more reliable than manual assessment **Figure 14-2**. Correlation with an ECG display or pulse oximetry pleth (waveform) provides additional reassurance.

The **Allen test** can be used to assess extremity perfusion and ulnar artery function. With this technique, the patient's hand is initially held above his or her head while the fist is clenched and the radial and ulnar arteries are compressed **Figure 14-3A**. The hand is then lowered and the fist is opened **Figure 14-3B**. Pressure is kept on the radial artery, whereas ulnar pressure is released. After ulnar pressure is released, the color should return to the hand within 6 seconds.

Invasive Hemodynamic Monitoring

Monitoring of hemodynamic status in the critical care transport environment often involves placing

catheters in the venous and/or arterial vasculature. The catheters are connected by using rigid tubing to a pressure transducer (sensor) that will measure the pressure conducted from the patient. As mentioned, placing these invasive catheters requires considerable skill and practice and is usually outside the scope of CCTPs. Although CCTPs may not place the catheters directly, they will provide care to patients with pressure monitoring catheters. Therefore, CCTPs must be familiar with the methods for using each type of catheter and have the knowledge to interpret the data provided. They must also be familiar with common complications involved with these invasive catheters, including what must be done if the catheter is prematurely removed.

Central Venous Lines

Central venous lines are invasive catheters placed inside the central venous system that provide access to the core vessels of the body. The right internal jugular vein **Figure 14-4** is the easiest site to access and insert a central venous line because of desirable anatomy and a relatively low rate of complications. Subclavian sites are more preferable because they best minimize infection risks. Femoral and brachial veins are also common sites, although femoral lines are strongly discouraged in adults because they have the highest risk for infection of any central venous line location.

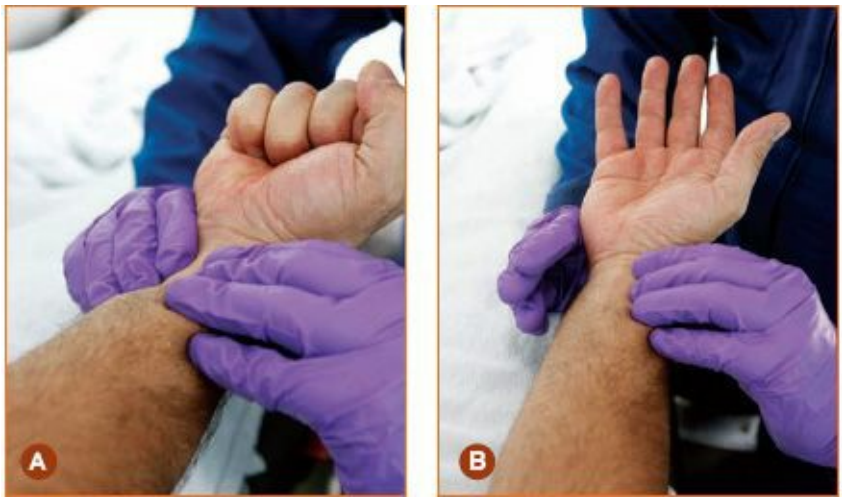


Figure 14-3 The Allen test. **A.** The patient raises his or her hand above his or her head with the fist clenched. The CCTP compresses the radial and ulnar arteries. **B.** The patient lowers his or her hand with the fist open and the CCTP releases ulnar pressure while keeping pressure on the radial artery.

The catheter is inserted until the tip resides just outside the right atrium (in the superior vena cava). This position allows for central venous pressure (CVP) readings (also known as right atrial [RA] readings). Monitoring of the CVP is used to determine right ventricular preload and intravascular volume status and to assess right-sided heart function. The normal range for the CVP is 2 to 6 mm Hg. A CVP reading can be misinterpreted because of thoracic pressure fluctuations during spontaneous and assisted respiration. For this reason, CCTPs should obtain a CVP measurement during end expiration to ensure its accuracy. Most patient care monitors designed for critical care measure the CVP with a presumption that the patient is being mechanically ventilated.

Indications

In addition to pressure monitoring, central lines often include outlet ports for obtaining blood specimens and for the delivery of IV fluids or medications. Central venous lines are frequently used when providers are unable to establish adequate peripheral vascular access or require access to central circulation for medications that cannot safely be given through peripheral sites. Central venous lines provide a means for

the following:

- Rapid fluid replacement
- Medication administration
- Rapid access to central circulation
- Invasive monitoring

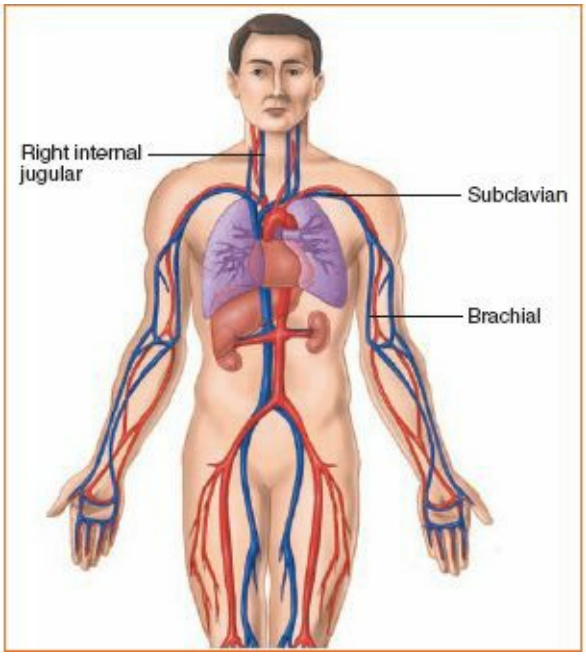


Figure 14-4 The right internal jugular vein is the easiest site to access and insert a central venous line. The subclavian, brachial, and femoral veins are also common sites.

Contraindications

There are few contraindications for central venous access. Relative contraindications include the following:

- Significant coagulopathy
- Local trauma to site of insertion
- Infection at site of insertion

Complications

Insertion of a central line is associated with some risk to the patient, including infection or bleeding at the insertion site. The most common complication is pneumothorax or hemothorax from an unsuccessful attempt at subclavian insertion. Additional complications include the following:

- Air embolism
- Infection (bloodstream)
- Thrombosis
- Phlebitis
- Limb ischemia
- Arterial puncture
- Improper placement (within the body)
- Myocardial perforation (resulting in pericardial tamponade)

- Thoracic duct injury (with left internal jugular or subclavian approaches)
- Nerve injury
- Catheter occlusion
- Sluggish infusion
- Catheter damage with resultant air embolism or infection
- Blood withdrawal problems (inability to obtain specimens)

Types of Central Venous Catheters

The most commonly used central line in critical care has three lumens and is simply referred to as a **triple-lumen catheter** Figure 14-5. All three lumens can be used for phlebotomy or fluid and drug administration; as an alternative, two lumens can be used for fluid administration and the other for CVP monitoring. In some cases, one port of a triple-lumen catheter may be reserved for total parenteral nutrition and should not be used during transport. There are additional types of central venous catheters—some that have one or two access ports and some that are installed under the skin (tunneled) and are designed for long-term multiple use for IV medication administration, dialysis, and parenteral nutrition. Tunneled central line devices are generally designed for long-term use. Emergency use of these catheters should be guided by direction from the receiving facility and local protocols.

Figure 14-5 HICKMAN® tri-fusion catheter. © C. R. Bard, Inc.

The HICKMAN® catheter Figure 14-6 is a semipermanent central venous catheter used for venous access and for drawing blood. Placement of this line can be into the cephalic, subclavian, external, or internal jugular vein with the distal tip advanced to just above the right atrium. The proximal end of the catheter exits from the lower portion of the anterior chest wall via a subcutaneous tunnel. Daily flushing of this line is necessary to prevent it from clotting and is also required every time the line is used to draw blood.

Complications associated with the HICKMAN® catheter include clotting of the line, catheter breakage, and infection of the catheter or insertion site. If the line becomes clotted, it should not be force flushed because the catheter could rupture or, more important, a thrombus could be released into the circulatory system. If catheter breakage should occur, a clamp should be placed between the break and the patient's skin until repair of the catheter can be accomplished. Infection at the insertion site is evidenced by erythema, edema, or discharge. Catheter infection is often evidenced as bacteremia, with fever and chills progressing into septic shock if not recognized and promptly treated. All complications should be reported to the receiving facility as soon as possible.

The GROSHONG® catheter Figure 14-7 is thinner, is more flexible, can have up to three lumens, and is equipped with a subcutaneous cuff. The catheter exits the skin near the nipple. There is a pressure-sensitive, two-way valve on the adjacent lateral wall that allows for the tip of the catheter to be closed. Because venous pressure maintains the valve in the closed position, there is no backflow of blood into the

catheter. The advantages of this design are that clamping and frequent heparin flushing are not necessary, although a saline flush is needed after each use or once daily. This type of catheter can also last between 6 weeks and 6 months or longer and allows the patient the freedom to do most activities.

Figure 14-6 A HICKMAN® catheter. © C. R. Bard, Inc.

Figure 14-7 GROSHONG® catheter. © C. R. Bard, Inc.

The port-a-cath is a titanium chamber with a catheter that threads under the skin into the subclavian vein to the right atrium. The titanium chamber is 1″ × 1″ × 0.5″ and has a selfsealing rubber top, which is implanted under the skin of the anterior part of the chest in the pectoral region. The catheter can be used for the infusion of fluid or medications and for blood draws and is accessed using a Huber needle, which is inserted through the skin and into the rubber top of the portal. A Huber needle is a specially designed noncoring needle that minimizes damage to the port and bends at a 90° angle to allow it to be secured to the chest wall once inserted into the port. The advantages of this catheter are that it may remain in place for years with proper care, it allows the patient to be active, and it does not require daily care. This catheter must be flushed with heparin once every 30 days if not in use. The risks of the port-a-cath are similar to those of external catheters and include kinks, ruptures, and, rarely, infections.

The peripherally inserted central catheter (PICC) **Figure 14-8** is used when medium-duration, long-term access is needed (usually limited to several months). This catheter can be used in ways similar to other centrally placed lines. This catheter is inserted via the brachial or other arm vein and is advanced to the superior vena cava, with the tip of the line just outside the atrium. The PICC requires regular flushing with heparin or saline solution to prevent clotting. Low-dose warfarin may be added to the patient's daily medications to augment line patency if clotting problems have occurred in the past. The disadvantages of this catheter include limited arm mobility and limitations on aggressive exertion (eg, swimming), in addition to other risks associated with placement of centrally placed catheters.

Figure 14-8 A PowerGroshong® peripherally inserted central catheter. © C. R. Bard, Inc.

Central Venous Catheter Placement

Although central venous catheters are rarely placed by CCTPs, for the student's background knowledge, this section explains how they are placed.

Equipment

The following equipment is needed to insert a central venous line:

- Catheters (various sizes and lengths)
- 5- and 10-mL syringes
- Saline flush solution
- Chlorhexidine-isopropyl alcohol solution
- J-tipped guide wire
- IV tubing
- IV fluids Suture materials
- Occlusive dressing
- Sterile drape
- Tape
- Local anesthetic

Steps

The most common technique for line insertion is the **Seldinger technique**.

Skill Drill 14-1 outlines the steps for inserting a central venous line using this technique:

1. Use universal precautions.
2. Prepare all equipment and flush all lumen ports.
3. Locate landmarks **Step 1**.
4. Palpate for entry location **Step 2**.
5. Clean and prepare the site using a sterile technique.
6. Administer a local anesthetic.
7. Insert the needle with connected syringe at the site, aspirating until blood is drawn into the syringe **Step 3**.

8. Remove the syringe and insert the guide wire into the needle. Feed the needle into the vein, never losing contact with the guide wire **Step 4**.
9. Remove the needle while holding the guide wire in place **Step 5**.
10. Pass the dilator over the guide wire to the skin. Use a scalpel to make a small (0.5- to 1-cm) incision in the skin to facilitate passing of the dilator **Step 6**.
11. Remove the dilator, maintaining contact with the guide wire **Step 7**.
12. Insert the catheter over the guide wire and into the vessel, removing the guide wire as the catheter advances **Step 8**.
13. Aspirate blood, and flush all ports to ensure patency **Step 9**.
14. Secure the line to the patient's skin using sutures or staples **Step 10.**
15. Dress the site, and follow up with a radiograph to ensure proper line placement. It is imperative that line placement be radiographically confirmed before departing with the patient.

The following lists outline specific procedures for insertion of central lines in the femoral, internal jugular, and subclavian veins.

Steps for femoral vein insertion include the following:

1. Prepare the equipment.
2. Clip hair from the area, and, if necessary, drape the area.
3. Cleanse the area with chlorhexidine–isopropyl alcohol solution.
4. Locate the femoral artery.
5. Numb the area using a local anesthetic.
6. Puncture the area two finger breadths below the inguinal ligament, medial to the artery, directing the needle cephalad at a 45° angle with the skin until the needle can no longer be advanced **Figure 14-9**.
7. Maintain suction on the syringe and pull the needle back slowly until blood appears in the syringe.
8. Lower the needle so that it is more parallel to the frontal plane. Remove the syringe and insert the catheter.

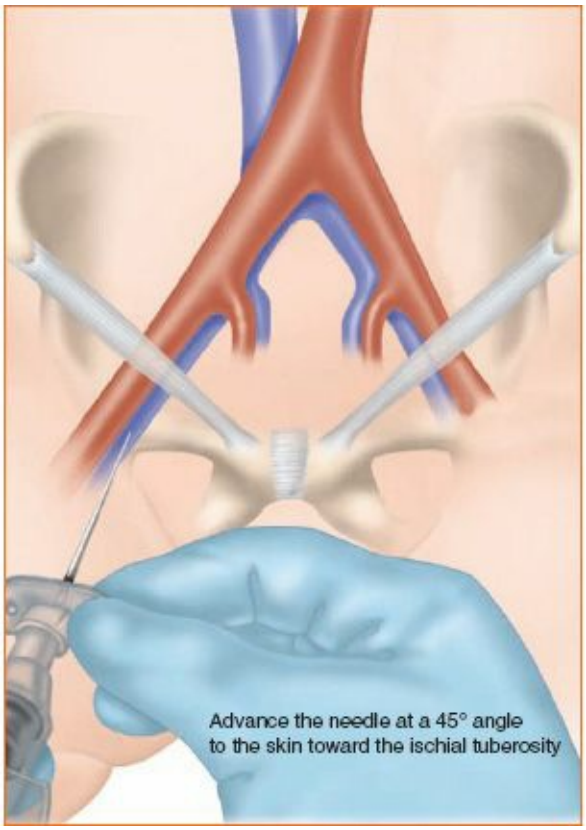


Figure 14-9 Femoral vein insertion. Adapted from: Henretig FM, King CC, eds. *Textbook of Pediatric Emergency Procedures*. Baltimore, MD: Williams and Wilkins; 1997.

Steps for internal jugular vein insertion include the following:

1. Prepare the equipment.
2. Determine the depth of catheter placement by measuring from the point of insertion to the following surface markers on the chest wall **Figure 14-10**:
   - **a**. Sternoclavicular joint to the subclavian vein
   - **b**. Midmanubrial area to the brachycephalic vein
   - **c**. Manubrial-sternal junction to the superior vena cava
   - **d**. 5 cm below the manubrial-sternal junction (the right atrium of the heart)
3. Place the tip of the catheter above the right atrium for administration of fluids.

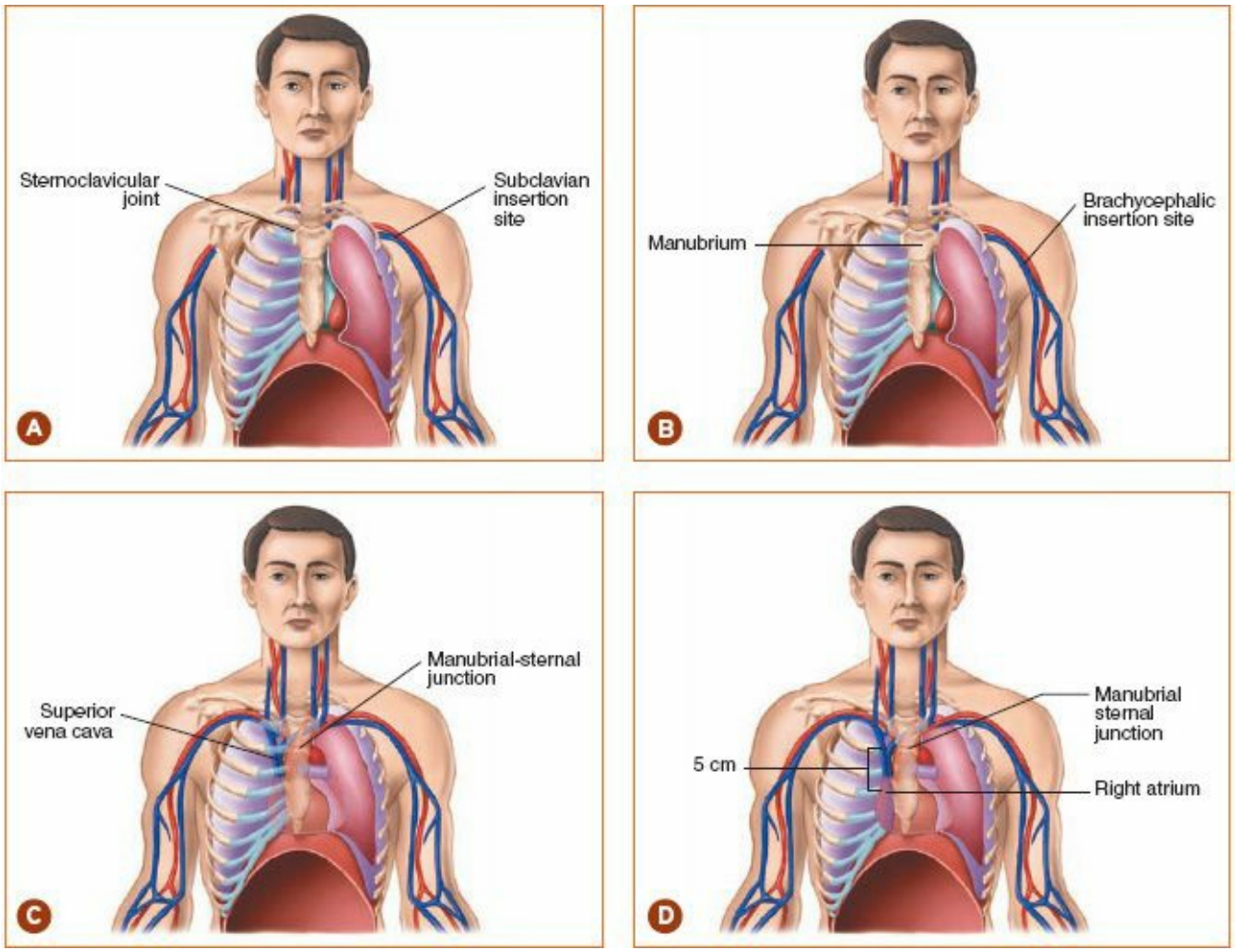


Figure 14-10 Determine the depth of catheter placement by measuring from the point of insertion to the following surface markers on the chest wall. **A.** Sternoclavicular joint to the subclavian vein. **B.** Midmanubrial area to the brachycephalic vein. **C.** Manubrial-sternal junction to the superior vena cava. **D.** Five centimeters below the manubrial-sternal junction (right atrium of the heart).

4. Place the patient in a supine, head-down position of 15°. Extend the patient's head and turn it away from the side of venipuncture.
5. Cleanse the area with chlorhexidine–isopropyl alcohol and drape the area. Maintain sterility.
6. Administer a local anesthetic.
7. Insert the needle at a 45° to 60° angle directed caudally **Figure 14-11**. Advance to a depth of 3 to 5 cm, depending on patient size.
8. If the vein is not entered, redirect the needle tip slightly more medially and repeat; do not direct the needle across the midline because the carotid artery may be punctured.
9. After entering the vein, advance the guide wire through the needle—minimal to no resistance is met. Monitor the ECG during insertion for atrial ectopy.
10. With the guide wire in place, withdraw the needle and advance the catheter to the predetermined depth.

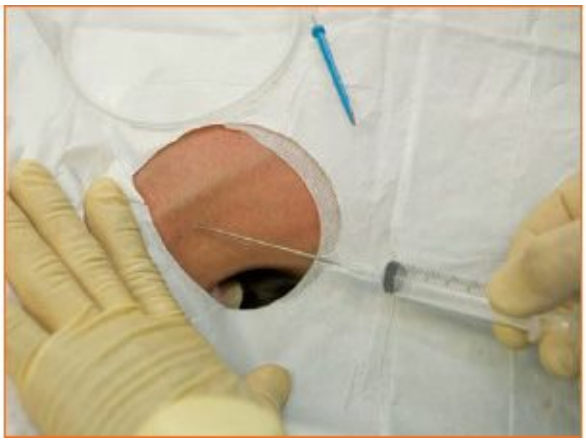
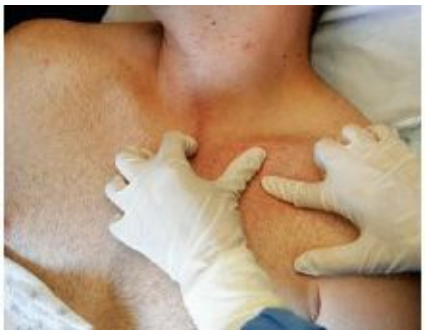


Figure 14-11 Internal jugular vein insertion.

Skill Drill 14-1

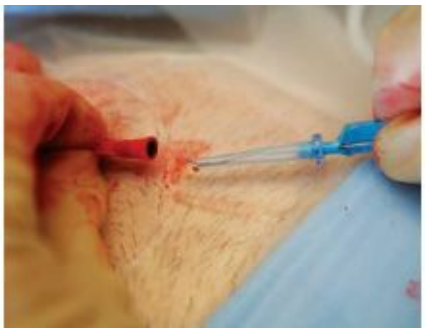
Inserting a Central Venous Line



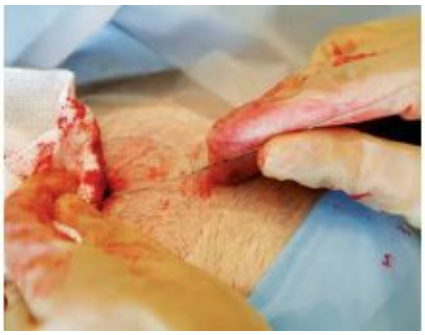
1 Locate landmarks.

2 Palpate for entry location.

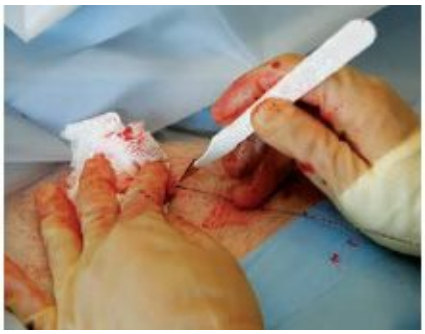
3 Insert the needle with connected syringe at the site, aspirating until blood is drawn into the syringe.



4 Remove the syringe, insert the guide wire into the needle, and feed the guide wire into the vein.



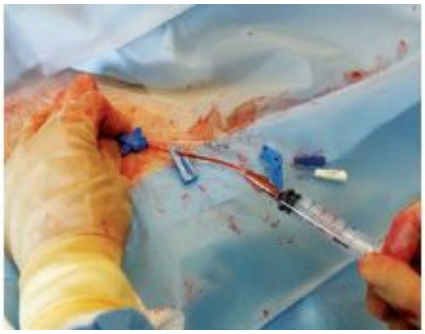
5 Remove the needle while holding the guide wire in place.



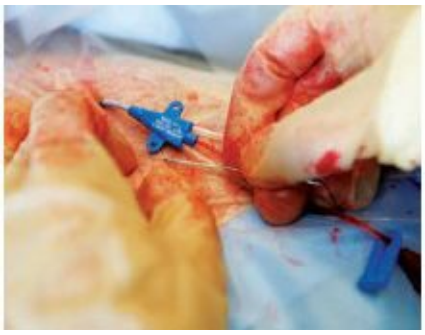
6 Pass the dilator over the guide wire to the skin. Use a scalpel to make a small (0.5- to 1-cm) incision in the skin.

7 Remove the dilator, maintaining contact with the guide wire.

8 Insert the catheter over the guide wire and into the vessel, removing the guide wire as the catheter advances.



9 Aspirate blood and flush all ports to ensure patency.



10 Secure the line to the patient's skin using sutures or staples.

Steps for subclavian vein insertion include the following:

1. Position the patient in a 15° head-down position.
2. Stand at the side of the bed.
3. Turn the patient's head away from the side to be cannulated.
4. Puncture the skin at the junction of the medial (middle of the clavicle) and middle third of the clavicle **Figure 14-12**.
5. Advance the needle beneath the clavicle toward the sternal notch to a depth of 3 to 5 cm.
6. Once the lumen of the vein has been entered, rotate the needle cephalad and clockwise 90° and proceed to insert the guide wire and catheter.

Troubleshooting

The most common problems that occur when inserting a central venous line are listed in **Table 14-1**. The table lists potential solutions to the problems.

TABLE 14-1 Common Problems Encountered When Inserting a Central Venous Line

Problem	Possible Actions to Resolve the Problem
Sluggish infusion	Check for kinks in the catheter. Reposition the catheter. Remove the injection cap and check for clots. Flush vigorously with 20 to 30 mL of normal saline.
Inability to withdraw blood	Check for kinks in the catheter. Make sure the catheter clamp is open. Flush vigorously with 20 to 30 mL of normal saline. Use repositioning maneuvers. Remove the injection cap and attach the syringe. Flush with saline.
Catheter damage	Clamp the catheter between the damaged area and the skin. Cover the damaged area with a sterile dressing. Do not attempt to repair.

Arterial Lines

Arterial lines (sometimes called A-lines) are inserted into the patient's arterial vascular system. The transducer converts the pressure waveform into an electrical signal and displays the waveform and its corresponding values on the transport (or bedside) monitor. In the United States, pressure waveforms are usually measured in millimeters of mercury or centimeters of water pressure. Arterial lines may be inserted in the radial, brachial, femoral, axillary, and dorsalis pedis arteries **Figure 14-13**. The radial artery is the preferred site because of its easy access and because the patient's hand can receive collateral circulation from the ulnar artery. It is best to use the patient's nondominant hand. In addition to the benefit of a continuously displayed BP reading while titrating vasoactive agents, the arterial line is an alternative trigger source for an intra-aortic balloon pump and enables access for arterial blood samples for arterial blood gas analysis.

Indications

Direct BP measurement using an arterial line is indicated for patients who require constant BP measurement. Direct BP measurement is also more accurate than indirect measurements (cuff pressures) in patients with low-flow states or high SVR conditions (ie, any patient in shock). The current indication for an arterial line is any patient in shock who is not rapidly responsive to therapy, including not only patients who are hypotensive, but also patients who are hypertensive and require powerful vasodilator therapy to reduce their vascular resistance. Arterial lines are also used routinely in patients receiving vasoactive or antihypertensive infusions.

Contraindications

There are several contraindications to arterial lines, including the following:

- Ischemia of the extremity
- Infection at the puncture site
- Raynaud disease
- Prior vascular surgery in the area of the insertion site

Complications

Complications from arterial line insertion include the following:

- Arterial line thrombosis
- Embolization
- Hematoma
- Insertion site infection
- Median nerve neuropathy (radial artery insertion)
- Pseudoaneurysm of the artery
- Ischemic necrosis
- Digit, hand, leg, or foot ischemia
- Hemorrhage
- Arterial air embolism
- Arteriovenous fistula
- Arterial aneurysm

Arterial Line Placement

Although some CCTPs may be trained and credentialed to insert central venous catheters, they generally do not place arterial lines. They may, however, need to assist in the procedure. For the reader's background knowledge, this section explains how they are placed.

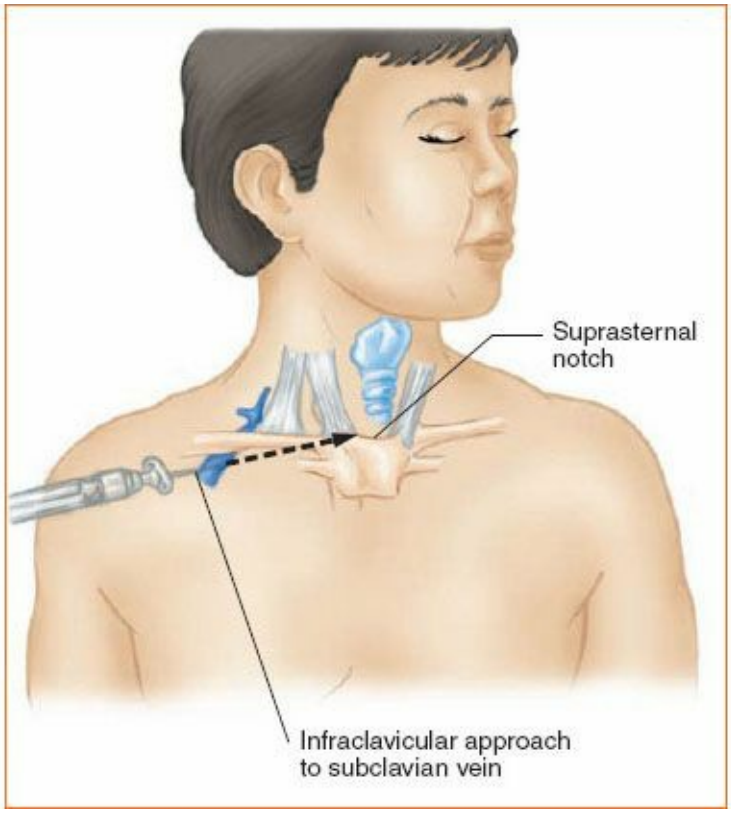


Figure 14-12 Subclavian vein insertion.

Equipment

The following equipment is needed to insert an arterial line:

- 20-gauge radial over-the-needle catheterization kit with guide wire
- 18- to 20-gauge 6″ femoral artery catheterization kit with guide wire
- Tape
- Sterile 2″ × 2″ gauze sponges; 4″ × 4″ gauze sponges
- Local anesthetics
- Sedation medications
- Sterile gloves, gown, mask, and drapes
- Chlorhexidine–isopropyl alcohol solution Pressure transducer Tubing
- Pressure monitor
- Suture material or commercial suture kit

Steps

Skill Drill 14-2 outlines the steps for inserting an arterial line using the modified Seldinger technique:

1. Use universal precautions.
2. Using a sterile technique, prepare all equipment.
3. Position the patient by placing rolled gauze under the wrist area, hyperextending the wrist **Step 1**.

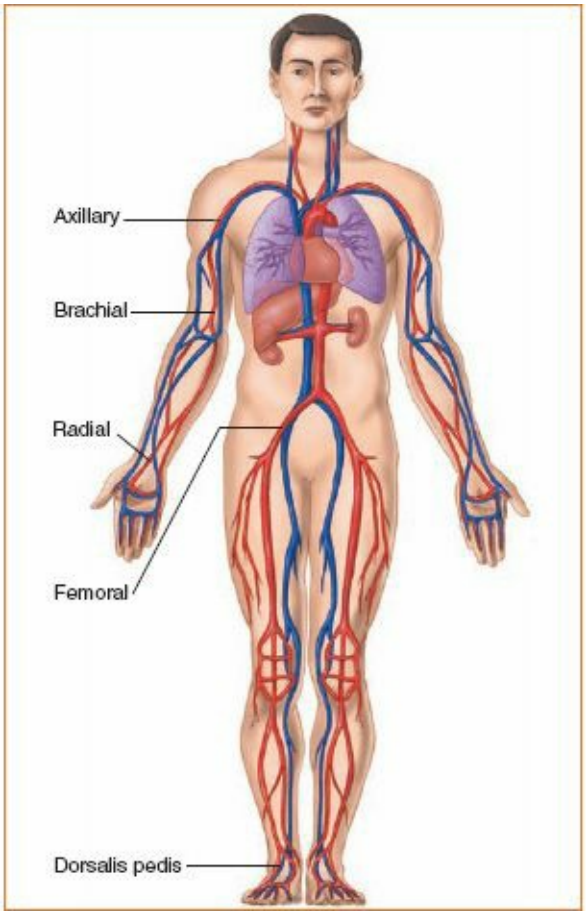
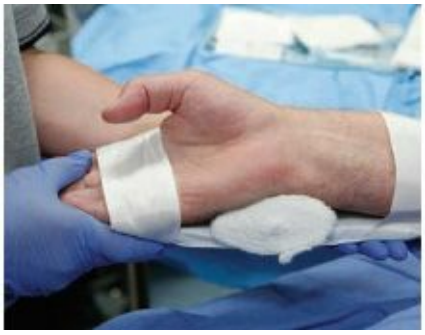


Figure 14-13 The radial artery is the preferred site for an arterial line. Other possible sites are the brachial, femoral, axillary, and dorsalis pedis arteries.

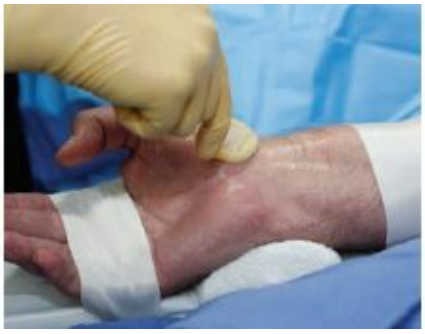
4. Select and clean the insertion site; ensure IV access. Use sterile drapes if available to reduce infection risk **Step 2**.
5. Palpate the artery at the distal radius **Step 3**.
6. Anesthetize the area **Step 4**.
7. Insert the needle over the radial artery approximately 1 cm distal to the wrist joint **Step 5**.
8. Advance the needle at an approximately 20° to 45° angle. Entry into the artery will be indicated by pulsating arterial blood.
9. Immobilize the needle with your free hand.
10. Advance the guide wire, and then remove the needle, leaving only the guide wire in the artery **Step 6**.

Skill Drill 14-2

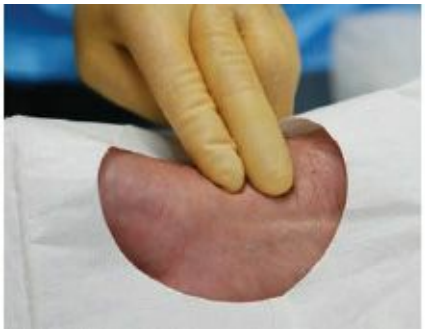
Inserting an Arterial Line



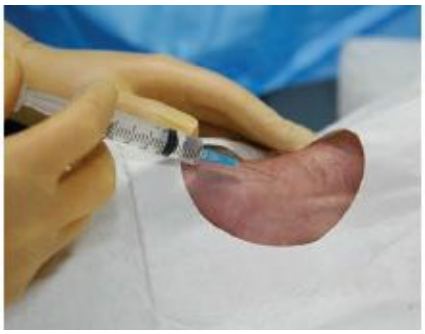
1 Place rolled gauze under the wrist area, hyperextending the wrist.



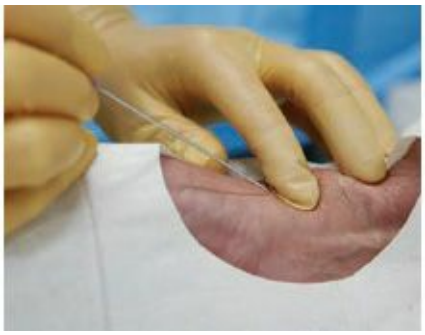
2 Select and clean the insertion site; ensure IV access. Use sterile drapes if available to reduce infection risk.



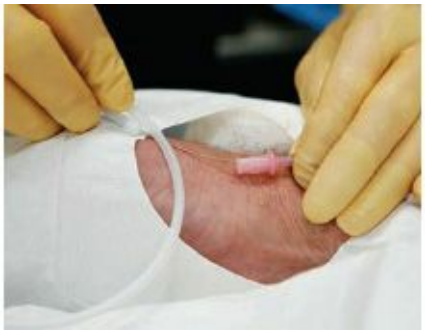
3 Palpate the artery at the distal radius.



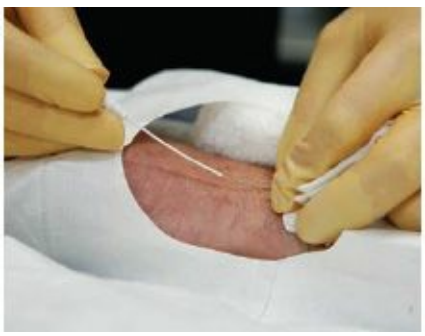
4 Anesthetize the area.



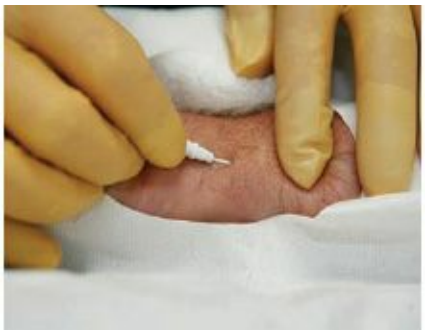
5 Insert the needle over the radial artery approximately 1 cm distal to the wrist joint. Advance the needle at an approximately 20° to 45° angle, watching for entry into the artery, indicated by pulsating arterial blood.



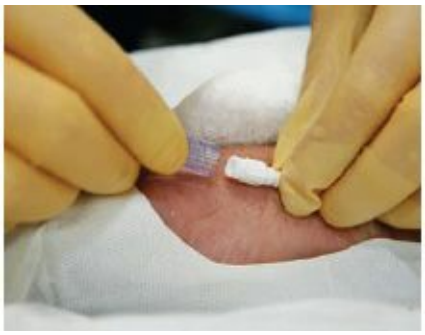
6 Immobilize the needle with your free hand. Advance the guide wire, and then remove the needle, leaving only the guide wire in the artery.



7 Place the arterial catheter over the guide wire.



8 Remove the guide wire, leaving only the cannula in place.



9 Connect the tubing to the pressure transducer. Secure the catheter to the skin with sutures and apply a sterile dressing to the insertion site.

11. Place the arterial catheter over the guide wire **Step 7**.
12. Remove the guide wire, leaving only the cannula in place **Step 8**.
13. Connect the tubing to the pressure transducer **Step 9**.
14. Secure the catheter to the skin with sutures and apply a sterile dressing to the insertion site.

The steps for femoral artery cannulation are also listed here for reference:

1. Prepare the equipment.
2. Use a complete aseptic technique.
3. Ensure IV access.
4. Palpate the femoral artery below the inguinal ligament.
5. Anesthetize the area.
6. Enter the skin over the femoral artery approximately 1 to 2 cm below the inguinal ligament **Figure 14-14**.
7. Advance the needle at an approximately 45° angle. Entry into the artery will be indicated by pulsating arterial blood.
8. Immobilize the needle with your free hand.
9. Advance the guide wire through the needle. Remove the needle, leaving the guide wire in place.
10. Pass the arterial catheter over the guide wire; remove the guide wire.
11. Connect the tubing to the pressure transducer.

12. Secure the catheter with sutures and apply a sterile dressing.

Pulmonary Artery Catheters

Like a central line, a **pulmonary artery catheter (PAC)** is inserted into the venous system of a patient. The PAC is a flow-directed catheter with a balloon at the tip that allows blood flow to carry the catheter through the heart into the pulmonary artery, where it terminates. The PAC is also known as a Swan-Ganz catheter (or simply a "swan") after Harold Jeremy Swan and William Ganz, who introduced this catheter in 1970.

The Pulmonary Artery Catheter Education Project (http://www.pacep.org) is an online, state-of-the-art educational program on how to use the PAC in the clinical environment. Supported by multiple critical care organizations and associations, the Pulmonary Artery Catheter Education Project Web site is an excellent resource for CCTPs to develop and enhance their knowledge base.

A PAC uses the same access routes as a central line. The PAC is fed into the vena cava and then through the right atrium **Figure 14-15**. Once past the lumen of the introducer sheath, the catheter tip balloon is inflated with 1.5 mL of air to help facilitate further placement. With the balloon inflated, the catheter is "floated" through the right atrium into the right ventricle and ultimately "wedges" into a proximal branch of the pulmonary artery. Once in place, the balloon is deflated to allow blood flow around the catheter.

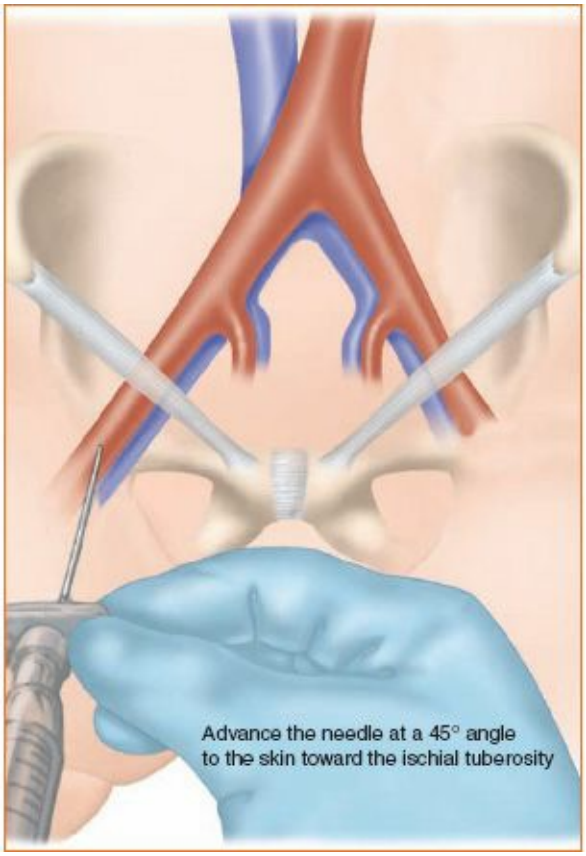


Figure 14-14 Femoral artery cannulation toward ischial tuberosity. Adapted from: Henretig FM, King CC, eds. *Textbook of Pediatric Emergency Procedures*. Baltimore, MD: Williams and Wilkins; 1997.

A PAC is 110 cm long and contains multiple lumens that terminate at various points along the length of the catheter, corresponding to different locations in the heart. The PACs are marked at 10-cm intervals beginning at the distal tip of the catheter, with one thin black line representing 10 cm, two thin black lines representing 20 cm, and three thin black lines representing 30 cm, up to one thick black line representing 50 cm, one thick black line accompanied by a thin black line representing 60 cm, one thick black line and

two thin lines representing 70 cm, and so on. During initial assessment, CCTPs should note the centimeter measurement of the PAC at the introducer. This information will help to determine if the catheter moves into or out of the patient during transport.

Two of the lumens are connected to pressure transducers that continuously display measurements. The first pressure reading is the RA pressure, and the second pressure reading is the pulmonary artery pressure. The RA pressures are displayed as mean pressures, whereas pulmonary artery pressures are displayed as systolic, diastolic, and mean values. Once the PAC is properly placed in the pulmonary artery, inflation of the PAC balloon results in blockage of blood flow from the right ventricle behind the balloon tip and the appearance of a pulmonary capillary wedge pressure (PCWP) waveform on the monitor. The PCWP waveform, like the RA pressure, is measured as a mean value.

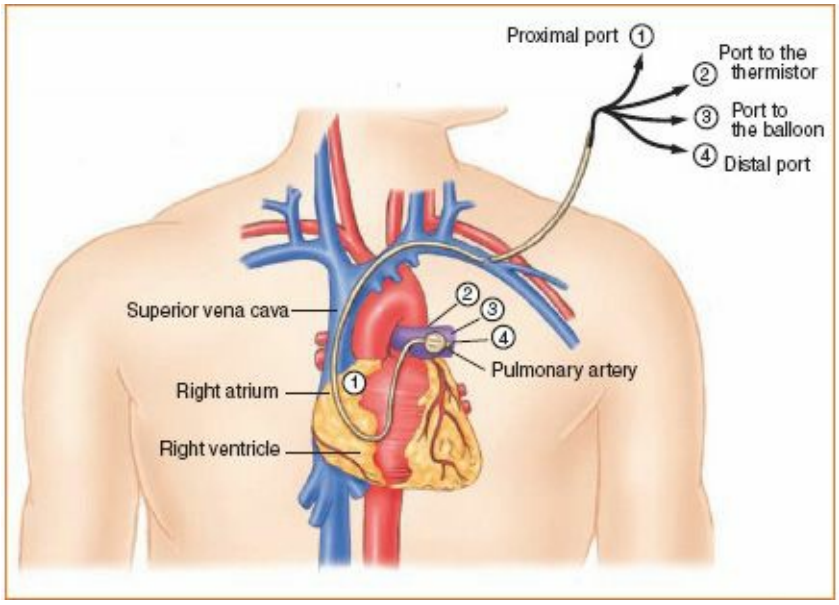


Figure 14-15 Pulmonary artery catheter placement. Once in place, the tip is deflated to allow blood flow around the catheter.

Complications of PAC insertion are nearly the same as complications of central line insertion, with the addition of pulmonary artery perforation or rupture, pulmonary infarction, arrhythmias, tricuspid and pulmonic valve injury, tamponade, or knotting of the catheter. The greatest potential complication of PAC use is probably misinterpretation of the data by clinicians, resulting in inappropriate therapy and potentially worsening outcomes.

A **thermistor** used for thermodilution CO measurement is connected to the distal end of the PAC. This thermistor provides continuous reading of pulmonary artery temperature, considered the gold standard for core body temperature measurement. Some PACs also have a fiberoptic oximetric catheter at the distal tip for continuous monitoring of mixed venous oxygen saturation **Figure 14-16**. Although variations exist, manufacturers have standardized coloring of PAC lumens. All PACs have a blue RA (proximal) port, a yellow pulmonary artery (distal) port, a red balloon inflation port, and a yellow temperature thermistor port. Some PACs have additional RA and/or right ventricular ports that can be used for infusions or pressure monitoring. There are also specially designed PACs with integrated pacing wires that can be used for transvenous cardiac pacing and PACs with an integrated thermal filament that allows for continuous CO measurement using pulsed electrical signals.

Indications

Commonly accepted indications for pulmonary artery catherization have little or no evidence basis and are largely generated by clinician experience. Broadly speaking, a PAC is thought to be warranted when

clinicians have a specific question about a patient's hemodynamic status that has not or cannot be answered readily by noninvasive assessment. Despite common use in cardiac, vascular, and other high-risk surgery and frequent insertion for management of end-stage heart failure, there have yet to be any outcome studies demonstrating improved outcomes with PAC use in critically ill patients. Conversely, there are also no clear data that there should be a moratorium on use of PACs. Instead, clinicians are advised to carefully consider the risks and benefits in critically ill patients on a case-by-case basis.

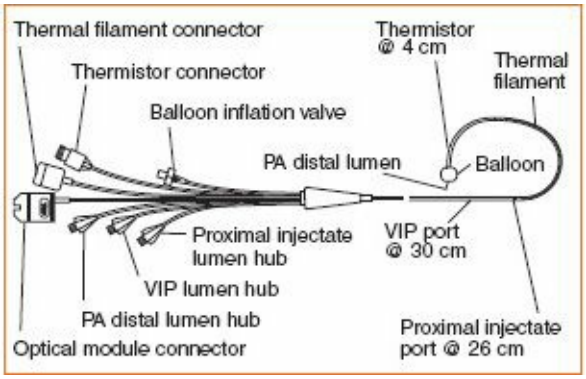


Figure 14-16 A pulmonary artery catheter with a thermistor. Courtesy of Edwards Lifesciences. Used with permission.

Some situations in which the use of a PAC might be considered include the following:

- Differentiation among causes of shock
- Determining mechanisms responsible for pulmonary edema
- Diagnosis or detection of pericardial tamponade
- Evaluation or diagnosis of intracardiac shunt(s)
- Evaluation of pulmonary hypertension
- Perioperative and postoperative management of patients with unstable cardiac status
- Management of complex myocardial infarction
- Management of patients undergoing cardiac and major vascular surgery
- Guidance for titration of inotropic, vasopressor, or vasodilator therapy
- Management of severe preeclampsia
- Fluid volume status management
- Assessment of cardiac performance

Contraindications

Like central venous catheterization, there are few absolute contraindications for pulmonary artery catheterization, although the risks of PAC use are greater than those associated with the use of central venous catheters alone. Relative contraindications to the use of PACs include the following:

- Significant coagulopathy
- Local trauma to the site of insertion
- Infection at the site of insertion
- Inability to float the PAC into the pulmonary artery

Equipment

The following equipment is needed to insert a PAC:

- The PAC
- A PAC introducer kit (must be 0.5F to 1F larger than the PAC)
- Sterile gowns and drapes for complete draping of patient and bedside area
- ECG and pulse oximetry monitoring
- Transducer set up
- Patient monitor and cables to connect to transducers
- Thermodilution CO set **Figure 14-17**
- Appropriate antiseptic solution (usually chlorhexidine)
- 2% Lidocaine and atropine (to treat arrhythmias during the procedure)

Steps

The steps for inserting a PAC are as follows. This skill would not be performed by CCTPs, but the steps are useful to understand as background knowledge:

1. Assemble and connect the transducers to the monitor.
2. Zero the transducers (described later in the section on "Leveling and Zeroing the Pressure Transducer").
3. Assemble the equipment.
4. Position the patient in reverse Trendelenburg (head-down) position, arms by his or her side, with his or her head turned in the opposite direction of the intended cannulation site.
5. Ensure that cardiac and pulse oximetry monitoring devices are in place and functioning.
6. Establish a sterile field. Open the introducer supplies.
7. Prepare the insertion site.
8. Administer a local anesthetic agent.
9. Prepare the introducer equipment while awaiting anesthetic effects.
10. Cannulate the vein at the selected insertion site using a needle that is 18 gauge or larger and attached to an empty 10-mL syringe.

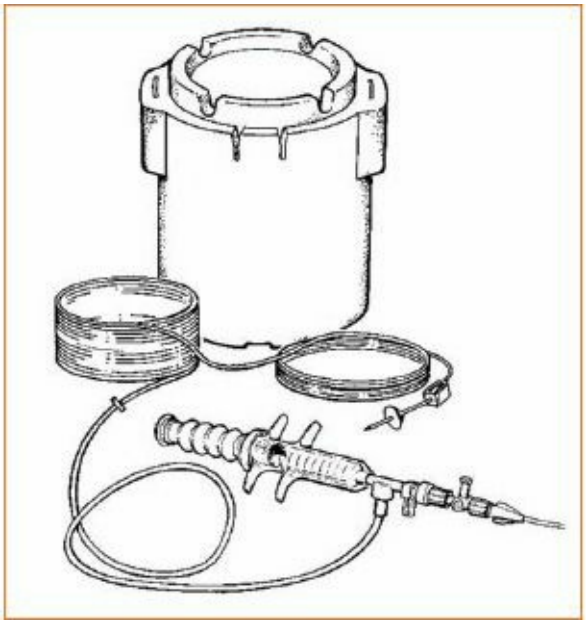


Figure 14-17 A thermodilution cardiac output set. Courtesy of Edwards Lifesciences. Used with permission.

11. Once the vein has been entered and the IV position of the needle has been confirmed, pass a Seldinger wire (included in introducer insertion supplies) through the needle.
12. Remove the needle.
13. Widen the skin opening using a scalpel.
14. Using a dilator, pass the PAC introducer over the wire into the vein. Remove and discard the wire.
15. Aspirate and flush the side port of the introducer with sterile saline.
16. Secure the introducer to the skin with suture(s).
17. Prepare the PAC by flushing the ports. Connect to the transducer(s) and slide the sterile sleeve over the catheter.
18. Test the PAC balloon and distal port to ensure that the waveform appears on the monitor.
19. Advance the PAC through the introducer.
20. Inflate the balloon once the PAC is past the distal end of the introducer (usually 15 cm).
21. Advance the PAC to the wedge position by observing waveforms on the monitor through the right atrium, right ventricle, and pulmonary artery.
22. If arrhythmias occur while the PAC is in the right ventricle, quickly advance the PAC into the pulmonary artery.
23. Once the pulmonary artery wedge tracing is observed, deflate the balloon to check for return of the pulmonary artery pressure waveform.
24. With the balloon deflated, pull back the catheter 1 to 2 cm and recheck wedge function.
25. Wedging the catheter should require 1 to 1.5 mL of air. If the catheter wedges with less than 1 mL of air, it has been inserted too far and will need to be pulled back slightly.
26. Secure the sterile sheath to the introducer and attach the PAC to the patient to prevent accidental dislodgement.
27. Obtain an anterior-posterior chest radiograph to check for pneumothorax and proper catheter position.

Invasive Pressure Measurements

A later section in this chapter will discuss how to take invasive hemodynamic measurements. Before moving on to the practical aspects of taking measurements, we will discuss the various measurements and their clinical significance.

By placing invasive catheters into the vasculature, hemodynamic parameters are continually monitored, and more advanced pressures can be assessed. Pressure for hemodynamic monitoring is measured in millimeters of mercury or, outside of the United States, in torr and kilopascals.

Common hemodynamic parameters are given in the following list. The line from which the measurement is obtained is shown in parentheses after each item, and the values that are calculated by a provider rather than obtained from the device are also noted.

- Central venous pressure (central line or PAC)
- Systemic blood pressure (arterial line)
- DBP (arterial line)
- MAP (arterial line, calculated or computed)
- Pulse pressure (arterial line, calculated)

- Pulmonary artery pressure (systolic, diastolic, and mean) (PAC)
- PCWP (PAC)
- CO (PAC)
- Pulmonary vascular resistance (PAC, calculated)
- SVR (PAC, calculated)
- Stroke volume (PAC, calculated)
- Mixed venous oxygen saturation (PAC)
- Total body surface area
- Cardiac index (PAC, calculated)

Normal ranges and values for invasive and noninvasive measurements are given in **Table 14-2**.

Cardiovascular Physiology

Cardiac Output

Critical care generally focuses on improving perfusion (the ability to deliver oxygen and nutrients to tissues). Hemodynamic monitoring assesses the effects of perfusion, and PACs look specifically at CO and its constituents: preload, afterload, and contractility of the heart. The CO is the product of stroke volume (SV) and heart rate (CO = SV × Heart Rate). The SV, or the amount of blood ejected with each ventricular contraction, is determined by preload, afterload, and contractility of the heart. Because SV results from three combined influences, heart rate has a far greater contribution and more immediate effect on CO than any one component of SV. When confronted with a low CO, a CCTP should investigate heart rate before evaluating constituents of SV.

TABLE 14-2 Normal Ranges and Values for Hemodynamic Measurements

Assessment Parameter	Normal Range/Value
CVP, mm Hg	2 to 6
SBP, mm Hg	100 to 120
DBP, mm Hg	60 to 80
MAP, mm Hg	70 to 105
Pulse pressure, mm Hg	40 to 60
Heart rate, mm Hg	60 to 100
Pulmonary artery pressure, systemic	15 to 30
Pulmonary artery pressure, diastolic	8 to 15
PCWP	4 to 12
CO, L/min	4 to 8
Pulmonary vascular resistance, dyne-sec/cm^{-5}	100 to 250
SVR, dyne-sec/cm^{-5}	800 to 1,200
SV, mL/beat	60 to 100

Sv_{O_2}, %	60 to 80
Scv_{O_2}, %	≥ 70
Cardiac index, L/min/m^2	2.5 to 4.2
Stroke volume index, mL/beat/m^2	30 to 50

Abbreviations: CO, cardiac output; CVP, central venous pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; Scv_{O_2}, mixed central venous oxygen saturation; SV, stroke volume; Sv_{O_2}, mixed venous oxygen saturation; SVR, systemic vascular resistance.

To account for differences in body size, some hemodynamic values are indexed. Indexing divides a hemodynamic parameter by the patient's total body surface area (BSA), which is stated in terms of meters squared. The BSA can be derived from a paper-and-pencil BSA chart but is most commonly calculated by hemodynamic monitoring software using the patient height and weight values entered by the clinician. For example, a **cardiac index** (average 2.5 to 4.2 L/min/m^2) is the patient's CO divided by his or her total BSA:

$$\text{Cardiac Index} = \frac{\text{CO}}{\text{BSA}}$$

A CO of 4.5 L/min may be perfectly adequate for a 17-year-old girl involved in a multivehicle crash, but not for a 325-lb professional athlete who crashed his motorcycle. The indexing of various hemodynamic parameters is often applied to ventricular work values, SVs, and calculations of oxygen consumption.

Preload

Preload is the degree of myocardial fiber stretch induced by volume in the ventricle at the end of diastole. Preload is synonymous with end-diastolic volume and end-diastolic pressure. Preload is determined by total blood volume, blood volume distribution, and atrial kick. States of hypovolemia result in decreased preload and may result from hemorrhage, diaphoresis, protracted vomiting or diarrhea, dehydration, burns, and gastric suction, among other causes. Hypervolemia, which typically results from fluid overload as a result of overzealous or runaway IV fluid administration or renal failure, is reflected in increased preload. Third-space losses create a relative hypovolemia reflected in decreased preload.

The distribution of blood volume also influences preload. Venodilation, as might result from drugs, fever, exercise, or distributive shock states such as neurogenic shock or sepsis, reflects decreased preload. Vasoconstrictive states induced by pressors, hypothermia, or cardiogenic shock states reflect increased preload. Intrathoracic pressure changes that decrease venous return, such as mechanical ventilation, positive end-expiratory pressure (PEEP), or pneumothorax, cause decreased preload. Intrapericardial pressure changes resulting from tamponade, pericarditis, or effusions affect the ability of the heart to fill and, hence, decrease preload.

Atrial kick, or atrial-ventricular synchrony, reported variously to be responsible for 10% to 20% of CO, is a preload-enhancing cardiac function. The small injection of blood from the atria into the ventricles just before ventricular systole boosts CO. Loss of atrial-ventricular synchrony decreases preload.

Preload is measured in several ways. Right ventricular preload is measured by CVP, which is closely synonymous with RA pressure. Left ventricular preload is measured by PCWP or, in some patients, by pulmonary artery diastolic pressure. During cardiac surgery, a left atrial pressure monitoring

catheter is sometimes placed to continually monitor left ventricular preload.

Afterload

Afterload is the resistance the ventricles must overcome to eject blood. Three factors typically determine afterload: outflow obstructions, vascular tone, and blood viscosity. Outflow obstructions, such as aortic valvular stenosis, hypertension, and atherosclerosis, produce greater resistance and, hence, increase afterload. Aortic aneurysms, in which blood flows through a false lumen rather than the inner lumen of the aorta, represent decreased afterload. As discussed with preload, vasoconstriction and vasodilation also affect afterload: vasodilation reduces afterload, and vasoconstriction increases afterload. Blood viscosity directly affects afterload. Increased afterload results from polycythemia because very viscous blood does not flow as readily as less viscous or very thin blood. This concept is important and explains why lowered hemoglobin levels are often tolerated and, in fact, sometimes considered beneficial in critically ill patients. Presuming that there is adequate hemoglobin to carry oxygen, values below those considered normal may well improve CO by decreasing afterload.

Left ventricular afterload is measured by SVR, and right ventricular afterload is measured by PVR. In clinical practice, left ventricular afterload is more often evaluated when addressing issues with CO because manipulations of SVR affect systemic circulation. Two instances when clinicians are interested in right ventricular afterload are when assessing a correlation between pulmonary artery diastolic pressure and PCWP, and when evaluating a patient for heart transplantation to determine if significant PVR is reversible. Patients with end-stage heart failure with elevated PVR that is not reversible will require heart and lung transplantation because a transplanted heart would fail when faced with significant right-sided resistance.

Contractility

Contractility is the inotropic state of the heart or the force with which the heart muscle contracts. Contractility is not directly measurable using conventional hemodynamic monitoring technology but can be assessed with the newer Doppler and ultrasound devices increasingly being used for evaluation of critically ill patients. Six factors influence contractility: myocardial muscle health, autonomic nervous system, metabolic states, ion environment, pharmacologic agents, and heart rate.

Myocardial muscle injury or ischemia can decrease contractility temporarily or permanently, although conditioning seems able to improve contractility following injury. The parasympathetic and sympathetic arms of the autonomic nervous system responsible for increasing and decreasing heart rate simultaneously affect contractility. Metabolic states such as acidosis, hypoxemia, and sepsis decrease contractility. Three ions have significant influence on contractility: calcium, sodium, and potassium. Hypocalcemia, hyponatremia, and hyperkalemia all decrease contractility, although opposite changes in these ion levels do not seem to boost contractility above normal.

Multiple pharmacologic agents influence myocardial contractility. Agents that increase contractility are referred to as positive inotropic agents and include digoxin, dopamine, dobutamine, milrinone, calcium, epinephrine, and glucagon. Negative inotropic agents, or those that decrease contractility, include beta-blockers, quinidine, procainamide, and calcium-channel blockers. Finally, heart rate can influence contractility by its effect on diastolic filling time and coronary artery perfusion. Slower heart rates have a greater diastolic time than faster heart rates, allowing more time for coronary artery filling compared with very rapid heart rates. Less diastolic time seen with rapid heart rates potentially diminishes coronary artery perfusion and may lower myocardial perfusion below that needed for adequate contractility.

Contractility is inferred from the values for **stroke volume index, left ventricular stroke work index**, and **right ventricular stroke work index**.

Measurements From a Central Venous Line

Central Venous Pressure

Central venous pressure (CVP) is a measure of the vena caval and RA pressures. It measures the RA filling pressures and is, therefore, indicative of the patient's fluid volume status and right-sided heart performance. The CVP can be measured by using a central line or the proximal port of a PAC. Changes in the CVP generally correlate with changes in left ventricular filling pressure in healthy people. This correlation may not exist in patients with hypovolemic shock or significant pulmonary disease.

Even though a CVP waveform tracing has systolic and diastolic values, only the mean value is used in clinical practice because the numbers are extremely low **Figure 14-18**.

The normal range for the CVP is 2 to 6 mm Hg. As fluid is administered to a patient, a rise in the CVP is expected. Elevated CVP readings can result from positive pressure ventilation, increased PVR, hypervolemia, right-sided heart failure, cardiac tamponade, tricuspid insufficiency, significant vasoconstriction, pulmonary embolus, or obstructive pulmonary disease; they can also be a late sign of left ventricular failure. Lower CVP values can indicate hypovolemia or significant vasodilation. Typically, a value between 5 and 8 mm Hg is considered the cutoff between low and normal preload. Fluids are often administered to patients with CVP values below this range to increase their preload.

Measurements From an Arterial Line

Arterial Blood Pressure

Direct blood pressure measurement using an arterial line provides continuous assessment of arterial (or systemic) pressure. Because hemodynamic status can change by the minute, continual blood pressure monitoring can help CCTPs more closely observe patients during transport. An arterial line connected to the transport monitor provides constantly updated SBP, DBP, and MAP values and an arterial waveform **Figure 14-19**. Components of the arterial waveform include the **dicrotic notch**, mean pressure, systole, diastole, systolic pulse pressure, and diastolic pulse pressure. The dicrotic notch represents the brief increase in aortic pressure reflected in a notching of the wave. It is caused by the sudden closure and spring back of the aortic valve leaflets and signals the start of diastole.

Blood pressure readings from an invasive arterial catheter, when properly obtained, have less potential for inaccuracies in measurement that are often associated with BP cuff measurements, such as operator error, inappropriate size or placement, and effects of low flow or high SVR (shock) states.

Mean Arterial Pressure

An important calculation, sometimes done by CCTPs and sometimes by the machine, is **mean arterial pressure (MAP)**, a function of the SBP and DBP. The MAP represents the average (ie, constant) pressure the arterial vasculature feels from the pulsatile nature of the heartbeat. The MAP is calculated by the transport monitor software measuring the mean area under the arterial pulse pressure waveform. A reverse of this calculation is used by electronic blood pressure (NIBP) software to derive the SBP and DBP for display. If precisely one third of the cardiac cycle was spent in systole and two thirds was spent in diastole, the MAP could be calculated by using the following formula:

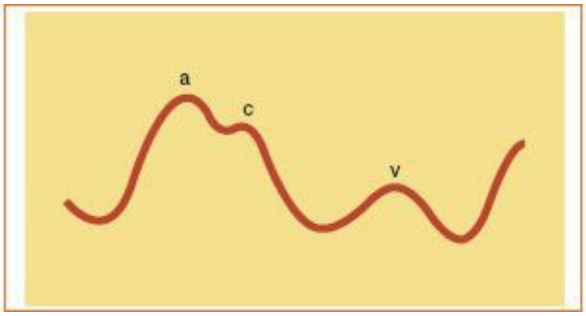


Figure 14-18 A central venous pressure waveform.

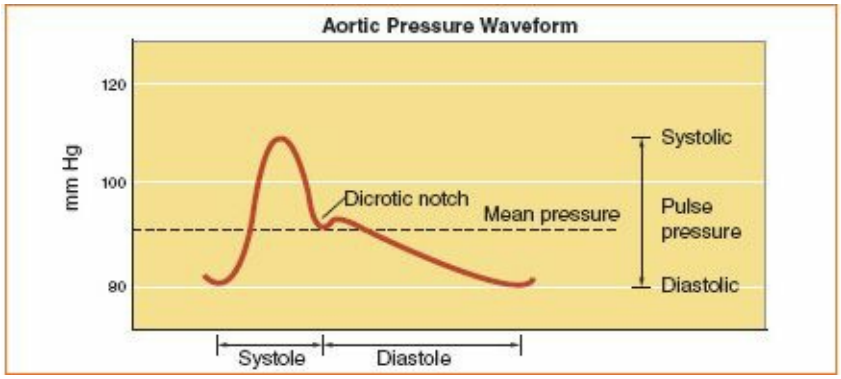


Figure 14-19 A blood pressure waveform from an arterial line.

$$MAP = DBP + \frac{1}{3}(SBP - DBP)$$

Because a heart rate of 70 beats/min is typically required, the formula is relatively useless in practice. The MAP measured by a transport monitor connected to an arterial line or by an NIPB is, however, the most precise of the pressure measurements available to CCTPs. Placement of an arterial line significantly affects the SBP. The further out from the heart that an arterial line is placed, the more pressure will be needed to flow blood and, therefore, the higher the SBP will be. The MAP, however, is unaffected by location of line placement and is the best parameter to use when making treatment decisions. Usually, a person needs an MAP between 70 and 105 mm Hg to satisfactorily perfuse the tissues. The most sensitive organs to hypotension are the kidneys, which typically require an MAP of 60 mm Hg or above and are likely to sustain irreversible injury when faced with an MAP below 60 mm Hg for more than 20 minutes. For that reason, most medical orders for blood pressure parameters require titration of drugs or administration of fluids to maintain an MAP of 70 mm Hg or greater.

Pulse Pressure

Pulse pressure is another value calculated by using the SBP and DBP. It is the mathematical difference between the SBP and DBP. Normal values of 40 to 60 mm Hg are seen in healthy people but may be altered in critically ill patients. The SV and vascular compliance affect the pulse pressure. An elevated pulse pressure can be indicative of high SV states (such as hypovolemia) or low vascular compliance (such as arteriosclerosis). In these states, there is a rise in SBP. If there is not a concomitant rise in DBP, elevated pulse pressure develops. Bradycardia can also cause an elevated pulse pressure. In this instance, lower DBP develops because of an extended diastole, which can lead to diastolic runoff. Low SV states (such as hypovolemia) and high vascular compliance (such as shock) will cause a decrease in SBP and, therefore, a decrease in pulse pressure (barring a proportional decrease in DBP). Tachycardia may cause a decreased pulse pressure by the reverse of the bradycardia mechanism.

Measurements From a Pulmonary Artery Catheter

Right Ventricular Pressure

Although not routinely monitored or measured, the **right ventricular pressure** is important during insertion and use of a PAC. The right ventricular pressure has three components **Figure 14-20**:

1. Systolic pressure, usually 20 to 30 mm Hg
2. Diastolic pressure, usually 0 to 5 mm Hg

3. Mean pressure, usually 10 to 20 mm Hg

Ventricular ectopy is common when the PAC is in the right ventricle; hence, it is important for CCTPs to be able to recognize a right ventricular waveform and its characteristic low diastolic pressure, which distinguishes it from a pulmonary artery pressure waveform.

Pulmonary Artery Pressure

Like the arterial BP waveform, the **pulmonary artery pressure** has three components **Figure 14-21**:

1. Systolic pressure, usually 15 to 30 mm Hg
2. Diastolic pressure, usually 8 to 15 mm Hg
3. Mean pulmonary artery pressure, usually 10 to 20 mm Hg

The respective pulmonary artery pulse pressure can also be calculated.

Pulmonary artery pressures are a function of right ventricular health and the vascular resistance of the pulmonary circuit. Although patients with significant heart failure or pulmonary obstructive disease can have chronically elevated pulmonary artery pressures or pulmonary hypertension, acute elevations in pulmonary artery pressures are unlikely to result from causes other than hypoxemia or hypercarbia. An astute CCTP who observes a sudden elevation in pulmonary artery pressures will immediately assess and correct any issues with oxygen saturation or ventilation (as reflected by end-tidal carbon dioxide readings).

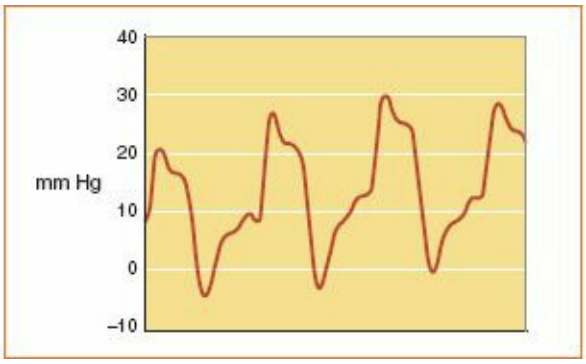


Figure 14-20 A right ventricular pressure waveform has a systolic pressure comparable to the pulmonary artery systolic pressure.

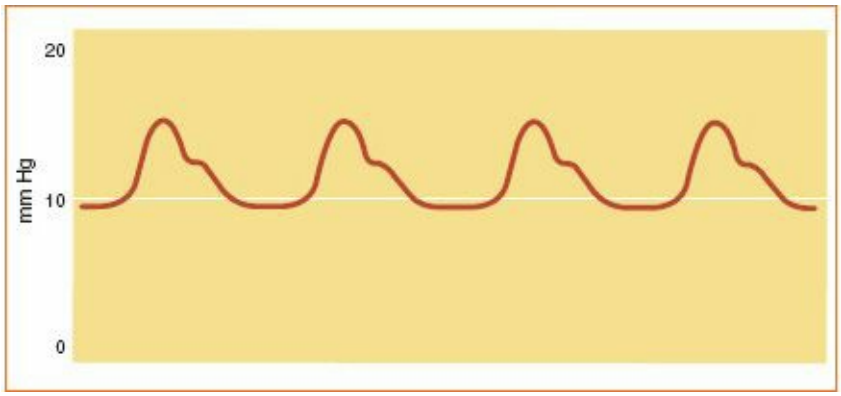


Figure 14-21 A pulmonary artery pressure waveform, which is similar to the arterial pressure waveform (ie, systolic, dicrotic notch, and diastolic components).

Elevations in the pulmonary artery pressure can result from conditions that increase pulmonary blood flow, such as atrial and ventricular septal defects or hypervolemia. Elevations in the pulmonary artery

diastolic pressure can result from tachycardia (usually >120 beats/min in adults) or pulmonary emboli. Decreased pulmonary artery diastolic pressure is seen with hypovolemia.

If pulmonary artery diastolic pressures suddenly drop, the catheter position should be reevaluated to determine if the PAC has migrated into the right ventricle (see Right Ventricular Pressure earlier in the chapter).

Pulmonary Capillary Wedge Pressure

The **pulmonary capillary wedge pressure (PCWP)** reflects preload to the left ventricle and is considered the gold standard for preload assessment. This measurement is also called pulmonary artery occlusion pressure and pulmonary artery wedge pressure. Also a function of a PAC, PCWP is an intermittent assessment performed by inflating a small balloon on the distal tip of the PAC, which causes the PAC to shift forward to "wedge" against the opening of the proximal branch of the pulmonary artery. This occludes the pulmonary artery from any antegrade blood flow. With the tip inflated, the transducer reads a pressure that is equivalent to the left atrial pressure. The left atrial pressure is closely related to and often parallels the **left ventricular end-diastolic pressure**, which is indicative of left ventricular health. The normal range for the PCWP is 4 to 12 mm Hg. A properly obtained PCWP will normally be slightly less than the pulmonary artery diastolic pressure (≤4 mm Hg). A PCWP of 10 to 12 mm Hg is normally used to differentiate hypovolemia from euvolemia in clinical practice. Patients with PCWP values below this level are often given fluids to optimize their preload.

The CCTP needs to understand how to read the PCWP waveform **Figure 14-22**. Hemodynamic waveforms often show variations in baseline as a result of respiratory-induced intrathoracic pressure fluctuations. Atmospheric and pleural pressures are most closely approximated at end expiration. To eliminate the effects of intrathoracic pressure on PCWP values, they are measured at end expiration.

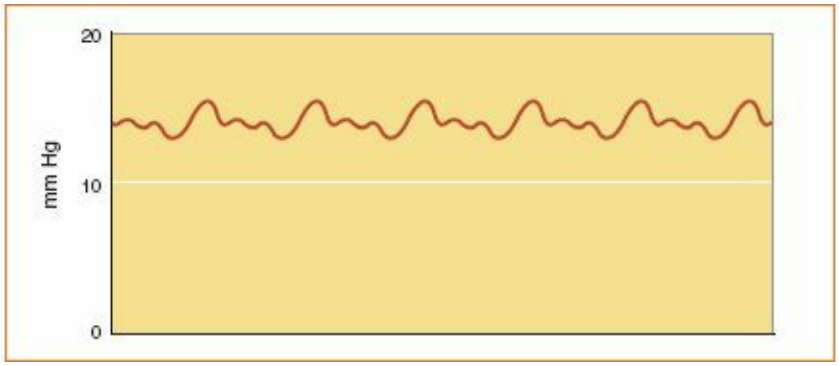


Figure 14-22 The waveform of a normal pulmonary capillary wedge pressure.

Depending on whether the patient is mechanically ventilated or spontaneously breathing, end expiration will be observed in different locations on the PCWP baseline. In mechanically ventilated patients, positive pressure inspiration elevates intrathoracic pressure and results in a positive fluctuation in the PCWP tracing baseline. Expiration is passive; end expiration is found at the lowest point in the waveform baseline. In spontaneously breathing patients, inspiration is induced by negative pressure. Expiration produces an increase in the waveform baseline; end expiration can be found at the highest point.

When measuring PCWP, a helpful tool to recall where to locate end expiration on the waveform baseline is "patient—peak; ventilator—valley." End expiration in spontaneously breathing patients is found at the peak of the waveform baseline. In mechanically ventilated patients, end expiration is found in the valley of the waveform baseline.

Once the proper position on the waveform is located, measurement of the PCWP waveform is done by locating two sequential positive deflections on the waveform immediately before the point where the

baseline changes. In spontaneously breathing patients, this would be immediately before the baseline begins to fall; in mechanically ventilated patients, this would be immediately before the baseline starts to rise.

The two sequential deflections are averaged to determine the mean pressure, as shown in **Figure 14-23**.

The PAC balloon typically holds up to 2 mL of air. Syringes included with the PAC normally allow the operator to inject only 1.5 mL of air to inflate the balloon. Regardless of the balloon's ability to hold up to 2 mL of air, it is recommended to only inflate the balloon with 1.5 mL, as overinflation may result in rupture of the pulmonary artery or damage to the balloon. This injection should be done slowly, over 2 to 3 seconds. Once a PCWP waveform is observed, the tracing can be "frozen" on the monitor screen, and the balloon should be allowed to passively deflate. Maximum inflation times should not exceed 10 to 15 seconds. If less than 1 mL of air is needed to produce a PCWP tracing, the PAC is likely inserted too far into the patient and is at risk for spontaneously wedging. Prolonged inflation or spontaneous wedging can result in pulmonary infarction. Manual flushing of a wedged catheter may cause pulmonary artery perforation.

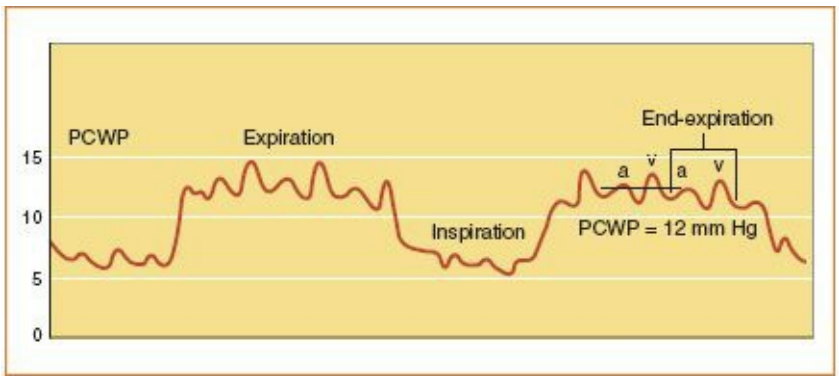


Figure 14-23 Respiratory variation in the pulmonary capillary wedge pressure waveform.

Causes of elevated PCWP are numerous and difficult to resolve definitively. Often, elevated PCWP results from the left ventricle not being able to clear the antegrade blood flow. If the left ventricle cannot clear increased flow from the right side of the heart (as in hypervolemia), an elevated PCWP will result. Left-sided heart dysfunction (as in left ventricular failure and mitral valve disease) will cause retrograde blood accumulation within the pulmonary vasculature and thereby show an increased PCWP. A PCWP level greater than 18 mm Hg often identifies a potential for pulmonary edema, especially with a normal colloidal osmotic pressure (ie, normal serum albumin level). High levels of PEEP used with mechanical ventilation can also cause an elevated PCWP. When PEEP levels above 10 cm H_2O are used, correction of measured PCWP may be requested. This is done by subtracting 1.5 mm Hg from the measured PCWP for every 5 cm H_2O of PEEP. For example, if the PEEP is 15 cm H_2O and the measured PCWP is 22 mm Hg, the corrected PCWP is 17.5 mm Hg.

If a patient has pulmonary edema, CCTPs may use the PCWP to aid in differentiating the cause. If pulmonary edema is the result of left-sided heart failure (cardiogenic), the PCWP will often be elevated. In cases in which pulmonary edema is the result of increased pulmonary capillary permeability (as in pneumonia or acute respiratory distress syndrome), the PCWP most likely will not be elevated. Care must be exercised with patients diagnosed as having acute respiratory distress syndrome because they often have elevated mean airway pressure levels and elevated PEEP, so the PCWP reading may be elevated as well.

Between the transducer on the tip of the PAC and the left ventricle lie three major anatomic components that may interfere with an accurate PCWP reading: the mitral valve, the left atrium, and the

pulmonary venous vasculature. Furthermore, the PCWP is a pressure trying to indicate a volume. This relationship generally holds unless the distensibility of the left ventricle has been affected (such as in a left ventricular myocardial infarction). In this case, the relationship fails.

The Frank-Starling Law

The **Frank-Starling law** states that there is a relationship between the distention of the ventricular myocardium and the force of the contraction. As the ventricular preload increases, the force of contraction of the myocardium also increases. This is the result of the increased stretch of the myocardial fibers. This relationship of increasing contraction with increases in the stretch of the myocardium immediately before systole continues until a point is reached and the force of contraction actually begins to fall off with subsequent detrimental increases in preload. The PCWP reflects preload in the left ventricle; the CVP reflects preload in the right ventricle. The Frank-Starling law describes how judicious administration of fluids results in improved CO up to a certain point, which varies between individual patients. Additional fluids given beyond the preload at which optimal CO is achieved result in worsened CO and the patient is said to have "fallen off the end of the Starling curve."

Cardiac Output

Cardiac output (CO) measurement rounds out the basic assessments performed by a PAC. Like the PCWP, CO is an intermittent assessment that involves some action by CCTPs. Typical CO assessment using a PAC uses the thermodilution method. This method involves injecting 5% dextrose in water (D_5W) at a known temperature (iced or room temperature) into the proximal port (usually the CVP port) of the PAC. Note that D_5W is the solution that is supposed to be injected; other solutions should not be used (other solutions, such as normal saline, introduce a low error level of approximately 2%). As the cold injectant moves past the thermistor on the distal end of the catheter, the temperature is recorded by the monitoring system and a curve is plotted comparing the temperature change over time between the injectant sensor and the distal end of the catheter. The monitor then calculates the CO. This procedure is repeated several times (three to six depending on local protocol), and the mean of these results is used to interpret the patient's CO. Curves that suggest erroneous injection technique or faulty temperature measurement are not included in the averaging strategy. Most transport monitors will not have the capability of performing thermodilution CO measurements during transport.

Normal CO ranges between 4 and 8 L/min. Investigation of low or higher than normal CO requires a stepwise approach considering each of the elements that compose the CO formula (CO = Heart Rate × SV).

The Fick Principle

The CO can also be calculated or estimated by using the **Fick principle**. First described by Adolph Fick in 1870, this method divides oxygen consumption by the difference between oxygen content of arterial and venous blood. The formula uses assumed values for oxygen consumption derived from basal metabolic studies on healthy subjects, which may or may not be valid in critically ill patients. Nevertheless, the Fick principle is commonly used in cardiac catheterization laboratories and critical care units. The formula is as follows:

$$CO = \frac{\text{Oxygen Consumption}}{\text{Arteriovenous Oxygen Content Difference}}$$

Data needed for calculating CO with this equation include the following:

1. BSA

2. Hemoglobin level
3. Arterial oxygen saturation (from a blood gas measurement or oxygen saturation as measured by pulse oximetry [Spo_2] from a pulse oximeter reading)
4. Mixed venous oxygen saturation (Spo_2) (from a blood gas sample drawn from the pulmonary artery port of a PAC or Svo_2 display reading from a fiberoptic PAC)

The BSA is expressed in square meters, and oxygen saturations are expressed as a decimal (eg, 98% would be expressed as 0.98).

Calculation of the estimated Fick CO is done as follows:

1. Calculate the oxygen consumption based on presumed 125 mL of oxygen consumed per square meter of BSA (ie, 125 × BSA).
2. Calculate the arteriovenous oxygen content difference. Each gram of hemoglobin can carry 1.36 mL of oxygen. Hence, the difference is expressed as follows: 1.36 × Hemoglobin × (Arterial Saturation — Venous Saturation) × 10.
3. Divide the calculated oxygen consumption by the calculated arteriovenous oxygen content difference to produce the estimated CO in liters per minute.

Assuming the following values, here is an example of the calculation:

- BSA = 1.33 m^2
- Hemoglobin = 12 g/dL
- Spo_2 (pulse oximeter) = 0.98 (98%)
- Svo_2 (saturation of blood gas sample drawn from pulmonary artery port) = 0.70 (70%)

$$CO = \frac{125 \times BSA\ 1.33}{Hgb\ 12 \times 1.36 \times (0.98 - 0.70) \times 10}$$

$$CO = \frac{166.25}{16.32 \times (0.28) \times 10}$$

$$CO = \frac{166.25}{45.696} = 3.64\ Liters/min$$

A simplified version of the formula can be used. This version calculates the constants (here, 125 has already been divided by 1.36 and 10), so that there are fewer calculations needed overall:

$$CO = \frac{9.19 \times BSA\ 1.33}{Hgb\ 12 \times (0.98 - 0.70)}$$

Pulmonary Vascular Resistance

Pulmonary vascular resistance (PVR) is calculated with the following equation:

$$PVR = \frac{(Mean\ Pulmonary\ Artery\ Pressure - PCWP)}{CO} \times 80$$

The PVR represents the resistance of the pulmonary vascular circuit that opposes flow out of the right ventricle and pulmonary artery, commonly referred to as right-sided heart afterload. The normal value is 100 to 250 dyne-sec/cm^{-5}. The CCTP may see an elevated PVR in patients with pulmonary hypertension or mitral valve stenosis.

Systemic Vascular Resistance

If a patient's MAP, CVP, and CO are known, the CO computer will calculate the **systemic vascular resistance (SVR)** by using the following equation:

$$SVR = \frac{(MAP - CVP)}{CO} \times 80$$

In a manner similar to PVR, a patient's SVR represents the total impedance to blood flow felt by the left ventricle, commonly referred to as afterload. Calculation of the SVR—with a normal range of 800 to 1,200 dyne-sec/cm^{-5}—is extremely useful in helping to differentiate the cause of shock. If the SVR is elevated, the patient has vasoconstriction, suggesting hypovolemia or pump failure. If the SVR is low, a distributive shock state such as neurogenic or septic shock is likely. Obviously the effects of vasodilating or vasoconstricting agents can affect SVR values. Also, SVR is a calculated, not a measured, value. As such, changes or errors in measuring any of the components included in the calculation could alter the results and lead to decisions based on inaccurate information.

The ideal SVR is thought to be just below 1,000 dyne-sec/cm^{-5}, and when a PAC is used to titrate vasodilator therapy (usually angiotensin converting enzyme inhibitor agents) in patients with heart failure, the goal is to achieve an SVR of 900 to 1,000 dyne-sec/cm^{-5}.

Stroke Volume

The amount of blood ejected in each stroke of a ventricle is measured in milliliters and recorded as the **stroke volume (SV)**. A normal SV is 60 to 100 mL/beat. The familiar equation that relates SV to CO and heart rate is as follows:

$$SV = \frac{CO}{\text{Heart Rate}}$$

As explained previously, ventricular performance is affected by the contractility (health) of the heart muscle and the preload and afterload to the respective ventricle. Any pathophysiologic factor that affects one of the performance determinants will augment or depress the SV.

The stroke volume index is calculated by dividing the SV by the BSA:

$$\text{Stroke Volume Index} = \frac{SV}{BSA}$$

The normal stroke volume index is between 33 and 47 mL/beat/m^2.

An estimation of contractility can be made by calculating left and right ventricular stroke work indexes as follows:

$$\text{Left Ventricular Stroke Work Index} = \text{Stroke Volume Index } (MAP - PCWP) \times 0.0136$$
$$\text{Right Ventricular Stroke Work Index} = \text{Stroke Volume Index } (\text{Mean Pulmonary Artery Pressure} - CVP) \times 0.0136$$

The normal left ventricular stroke work index is 40 to 75 gm-m/m^2/beat. (Note: gm-m represents grams per meter.) The normal right ventricular stroke work index is 5 to 10 gm-m/m^2/beat.

Mixed Venous Oxygen Saturation

If a fiberoptic oxygen saturation probe is attached to the distal end of the PAC, a **mixed venous oxygen saturation (SvO_2)** measurement can be recorded. This measurement is constant and requires no action by the clinician once the catheter is placed. In PACs without fiberoptic oximetry capability, a blood gas specimen can be obtained from the distal (pulmonary artery) port of the catheter to determine the mixed venous oxygen saturation. In effect, SvO_2 reflects the global balance between oxygen delivery and

consumption. The measurement is performed at the distal end of the PAC because the pulmonary artery is the most appropriate place to sample a mixture of all blood returning from the body. There are four factors that affect Svo_2; three pertain to oxygen delivery and one to oxygen consumption in the body. Oxygen delivery is affected by total hemoglobin levels, arterial oxygen saturation, and CO. Increases or decreases in any of these can affect the Svo_2. Changes in oxygen consumption (also called oxygen demand) can also affect the Svo_2. Oxygen consumption is increased with conditions that increase metabolic rate or muscle activity, such as fever, seizures, shivering, and increased work of breathing. Oxygen consumption may decrease when there is maldistribution of blood, which most commonly occurs in sepsis as a result of microcapillary obstruction leading to arterial blood being shunted past the capillaries into the venous blood. Normally, when oxygen demand increases and threatens to exceed oxygen supply, the body compensates by increasing CO and/or increasing oxygen extraction. When evaluating an Svo_2 value outside the normal range (0.60 to 0.80 [60% to 80%]), CCTPs should consider each of the four components that contribute to this parameter: total hemoglobin level, arterial oxygen saturation, CO, and oxygen consumption.

A measurement similar to mixed venous oxygen saturation is the **mixed central venous oxygen saturation ($Scvo_2$)**. Like mixed venous oxygen saturation, the $Scvo_2$ reflects the balance between oxygen supply and delivery. Measurements of $Scvo_2$ are obtained from a central venous catheter by drawing a blood gas specimen or from a fiberoptic oximetric tip connected to a specially designed monitor that displays the oxygen saturation. Because the location of the central venous line is more proximal than the pulmonary artery, blood sampled from a central venous line reflects extraction by the brain and upper body rather than global extraction; therefore, the $Scvo_2$ values are approximately 8% to 10% higher than the Svo_2 values. A normal $Scvo_2$ value is considered to be above 0.70 (70%).

Obtaining Invasive Hemodynamic Measurements

Once the central venous line, arterial line, or PAC is placed, it is connected to a pressure-monitoring transducer by using rigid pressure-monitoring tubing. The tubing is filled with an isotonic solution, usually normal saline. In the past, heparinized saline was often used, but lack of demonstrated benefit and the potential for harm to patients have all but eliminated its use as a flush solution. The difference between pressure-monitoring tubing and IV extension tubing is important because use of the latter will absorb much of the pressure waveform generated by the patient before it reaches the transducer. Transducers are biomedical devices designed to convert a physical or pressure waveform signal into an electrical signal. The electrical output of the transducer is connected by a proprietary electrical cable to the transport monitor.

To maintain patency of the pressure-monitoring line and fluid-filled system, a continuous flush solution system is used. Most often, this is done by placing the flush solution used to fill the monitoring system into a disposable pressure bag. Once the monitoring system is connected to the patient, free of bubbles, and functioning properly, the pressure bag is inflated to 300 mm Hg, or above the patient's BP. Whenever the pressure in the monitoring system exceeds the patient's BP, each transducer will flow 3 mL/h of flush solution into the patient. This continuous flush system maintains catheter patency and prevents blood backflow into the catheter and monitoring system. In cases in which less than 3 mL/h of flow through each transducer is necessary (such as in a neonate), the flush solution can be delivered to the transducer(s) by using an infusion pump.

If the transfer will involve air medical transport, all of the air left in the fluid bag, placed inside of the pressure bag, should be expelled to prevent expansion during flight. As mentioned earlier, the

pressure bag itself must remain at 300 mm Hg and may have a pressure indicator that will display the color green when inflated above 300 mm Hg. After the pressure bag is properly inflated, air should be turned off to prevent deflation **Figure 14-24**. The pressure bag should be periodically checked during transport to verify that it remains inflated.

Before moving a patient with hemodynamic monitoring equipment already in place, CCTPs should examine the equipment, note the values currently being displayed, and trace the tubing from each transducer to the pressure-monitoring catheter it is connected to. The catheter site should be examined and documented to be visibly clean, dry, and covered by an occlusive dressing. CCTPs should be familiar with operation of the stopcocks and catheters in use or should ask the clinician at the bedside for a review of its proper use.

Figure 14-24 An inflated pressure bag with proper inflation.

A CCTP may elect to transfer a patient to the transport stretcher before connecting transducers to the transport monitor or may make the connections to the transport monitor before moving the patient. In either case, one pressure cable is moved at a time to the transport monitor, ensuring that waveforms and values similar to those displayed on the hospital monitor appear on the transport equipment before transferring additional lines and pressure channels. Proceeding in a deliberate, stepwise manner avoids periods in which the patient is not connected to any monitoring equipment when changes may not be readily detected.

Extra caution is needed when moving a patient with hemodynamic monitoring equipment in place because catheters can easily become dislodged or pulled out when they are not adequately secured. Agency or service policy for securing monitoring lines and catheters during transport should be followed. The most reliable hemodynamic waveforms and pressure readings will be obtained from tubing less than 4′ (48″) from transducer to invasive catheter, with no more than one stopcock between the transducer and invasive catheter.

The best protection against accidental dislodgement or dislocation of invasive monitoring catheters is carefully set monitoring alarms. The transport environment is no exception to the need for monitoring alarm parameters designed to alert CCTPs if hemodynamic parameters exceed reasonable thresholds. Disconnected catheters will invariably register less than physiologic levels that can be readily detected when alarms are properly set by CCTPs.

Leveling and Zeroing the Pressure Transducer

The most common cause of error in obtaining direct hemodynamic pressure measurements is incorrect leveling of the transducer. Fluid used to fill the rigid pressure tubing that connects the monitoring catheter between the patient and the transducer will interfere with pressure readings if the pressure transducer is lower or higher than mid-heart level, commonly located at the patient's right atrium. The location of this

reference point is referred to as the **phlebostatic axis** and is located at the fourth intercostal space, mid-chest position **Figure 14-25**. **Referencing** is the process of ensuring that the hemodynamic pressure transducer is at the level of the left atrium. The transducer must be releveled with every position change of the patient; therefore, this point is often marked on the patient's skin. The simplest way to ensure proper placement during transport is to secure the transducer at the phlebostatic axis, although this is not always practical in the critical care setting or transport environment.

The phlebostatic axis moves with the patient and can be adjusted to obtain accurate readings in virtually any sitting or lying position. Transducers must be releveled with any change in patient position. For each inch that a transducer is leveled above the phlebostatic axis, readings will be 1.86 mm Hg less than actual pressures in the patient. For each inch that a transducer is leveled lower than the phlebostatic axis, values obtained will be 1.86 mm Hg greater than actual. Inches of error in leveling can have significant implications if treatment decisions are based on erroneously obtained values. For this reason, all leveling should be performed with a carpenter's level or leveling tool designed for hemodynamic monitoring applications.

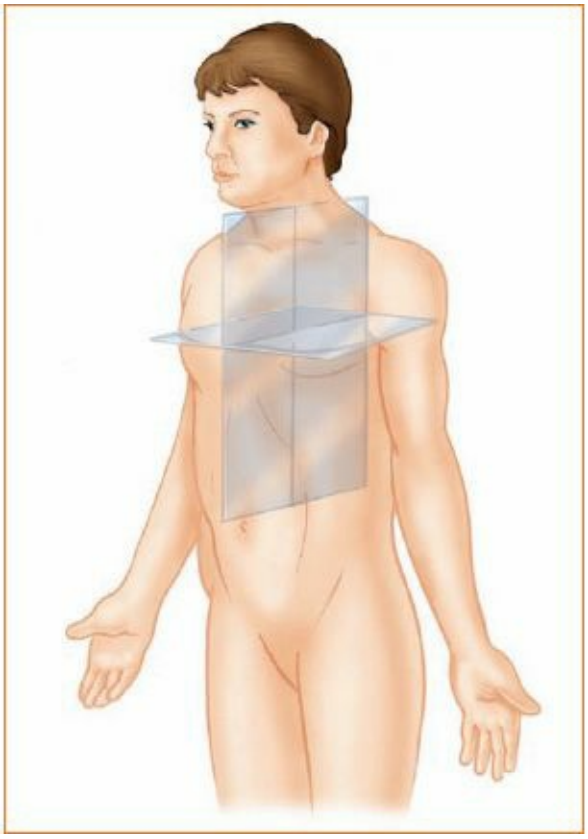


Figure 14-25 The phlebostatic axis.

In addition, any pressure transducer must be "zeroed" to eliminate the effects of atmospheric pressure on the measurements obtained. **Zeroing** is the process in which a transducer connected to the transport monitor is opened to atmospheric air to obtain a baseline reading of pressure in the environment. Once opened to air, the monitor is zeroed to eliminate the effects of atmospheric pressure on the transducer **Figure 14-26**. The procedure is as follows to zero the transducer: ensure that the cable is connected (or reconnected) to the monitor, close the stopcock to the patient, open the stopcock to air, and press the zero button on the monitor. Confirm that the monitor has accepted the zero value and actually indicates zero on the pressure and waveform displays.

Zeroing should be performed:

- Immediately after connecting the transducer to the transport monitor

- With any significant change of elevation greater than 1 atm
- Whenever displayed values are in question

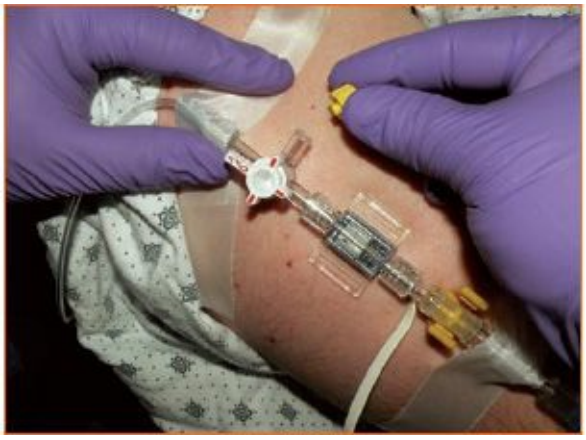


Figure 14-26 Zeroing the transducer.

Depending on flight patterns and altitudes, zeroing is often conducted at the initial connection of the transducer to the transport monitor, at cruising altitude, and again on arrival at the destination.

Respiratory Variation

Changes in intrathoracic pressure result in variable direct pressure measurements. To accurately obtain invasive pressure measurements, it is important to take readings at end expiration. Only at end expiration are the pressures inside the thorax equalized to atmospheric pressure outside the body. Respiratory variations produce a rise and fall of the baseline observed on hemodynamic pressure waveforms. In mechanically ventilated patients (positive-pressure ventilation), end expiration occurs in the lower portions, or valleys, of the rise-and-fall pattern. End expiration in spontaneously breathing patients (negative-pressure ventilation) correlates to the peaks of the rise-and-fall pattern. Making certain to read CVP and PCWP in the proper location based on whether the patient is mechanically ventilated (valley) or spontaneously breathing (peak) will ensure that the values are obtained during end expiration and not falsely affected by intrathoracic pressure changes.

Transducers and Cables

There are several transducer brands on the market, each with its own fast-flush system. The two most common types are the squeezable style and the pull style. The squeezable-style device has two pieces of plastic on either side of the transducer that, when squeezed, will fast flush the closed fluid-filled system **Figure 14-27A**. The pull-style (or pigtail) device has a small piece of rubber that resembles a pigtail **Figure 14-27B**. To flush the system, the CCTP pulls the tail straight out (away from the transducer).

Fast flushing overrides the normal delivery of 3 mL/h of flush solution through the transducer into the patient and allows free flow of pressurized flush solution through the system for as long as the fast flush assembly is activated. Fast flushing is useful as an initial technique to troubleshoot a **dampened waveform** or absent waveform in a system that had previously been functioning normally.

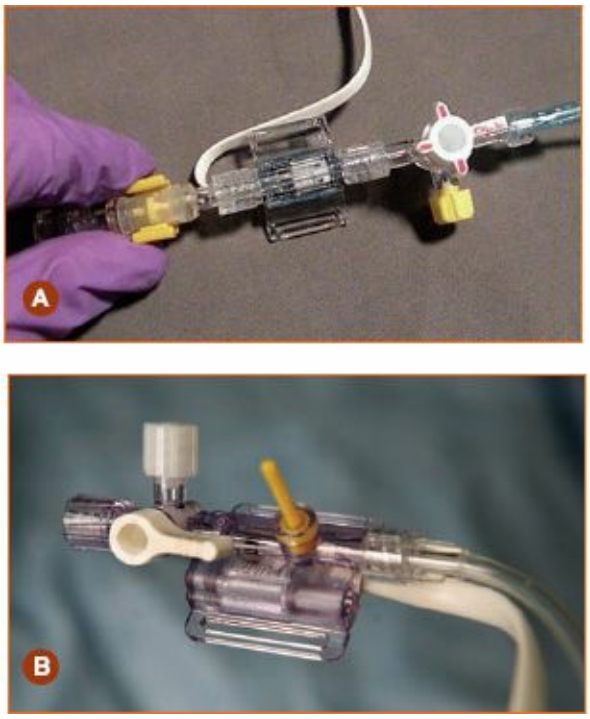


Figure 14-27 The two most common transducer flush system devices. **A.** Squeezable. **B.** Pull style.

CCTPs should become familiar with transport monitors and the cables needed to connect to pressure-monitoring equipment and transducers. Transport monitors are often limited to monitoring two pressure channels; it may be necessary to decide which pressures will be observed during transport. Any patient with a PAC must have the distal port (pulmonary artery) continuously monitored in case the PAC spontaneously wedges. Unmonitored or undetected spontaneous wedging can result in pulmonary infarction.

The cables that connect patient transducers to the monitor that an agency uses must be compatible with the equipment in use at the referring and receiving hospitals. There are two main cable types on the market. The most common has an end that attaches to the transducer that resembles a phone line connection; the other is best described as a half-moon connection **Figure 14-28**. It is best to carry at least two sets of each cable type to ensure accommodation of various transport monitors and transducers.

Potential Complications During Transport

Many complications can occur during transport of patients with central lines, arterial lines, or PACs. Probably the most common is accidental dislodgement or removal of the line or catheter. Meticulous attention to securing catheters, lines, and attached tubing before moving a patient to the transport stretcher with repeated checks for security and stability of these devices will help avoid loss of these important monitoring or access devices. Nevertheless, sudden patient movements or snags during movement happen, and lines sometimes become dislodged. Should an invasive line come completely out of a patient, the CCTP should immediately apply direct pressure to the insertion site and hold until bleeding stops. Central line and PAC insertion sites will usually stop bleeding with 3 to 5 minutes of direct pressure. Arterial lines can take considerably longer, depending on the size of the catheter and the clotting status of the patient. When holding pressure on an arterial line insertion site, CCTPs need to be certain that they are actually compressing the artery. Failure to apply pressure directly over the artery will allow continued bleeding into the tissue around the insertion site and can result in significant hematomas. Once bleeding is controlled, an occlusive dressing is applied to the site and the site is inspected frequently for continued bleeding.

Figure 14-28 Two types of cables that connect the transducer to the monitor are a phone line–type connection and a half-moon connection. A half-moon connection is shown here.

No attempt should be made to replace a catheter that dislodges but does not come completely out of the patient. Patency should be assessed by attempting to aspirate blood from each lumen to determine if each remains in a blood vessel. Options for continued use should be discussed with medical control. A catheter that needs to be removed is gently withdrawn from the patient and steps are followed as outlined previously to control bleeding at the insertion site.

Central venous access can have many of the same complications as peripheral venous access. Some of the complications include blood clotting from inadequate flow, breaking off of an embolism, catheter extravasation from improper placement, and catheter migration, which can cause inadvertent cannulation and pericardial tamponade. Other common problems encountered during invasive hemodynamic monitoring and actions to resolve them are listed in **Table 14-3**.

TABLE 14-3 Common Problems Encountered During Hemodynamic Monitoring

Problem	Actions to Resolve the Problem
Absence of a waveform tracing	Activate the fast-flush feature of the transducer for 1 to 2 seconds. Check the connections of the monitor cable. Ensure that the fluid-filled system is patent. Check that the stopcock is in the correct position and that the pressure bag is properly inflated. Aspirate blood from the stopcock. Notify the sending physician.
Damping of a waveform	Ensure that the flush solution container is not empty. Ensure that the pressure bag is properly inflated. Check the tubing for air bubbles or clots. Assess the catheter and insertion site for kinks.
Abnormally high or low readings	Verify that the transducer is properly leveled at the phlebostatic axis. Ensure that the pressure bag is properly inflated. Check that the fluid-filled system is patent.

	Rezero the transducer. Consider the possibility of actual hypertension or hypotension. Assess pressure with an alternative means to confirm values.
Air bubbles within the fluid-filled system	Ensure that the circuit is filled slowly to avoid air bubbles. Verify that the fluid chamber is not upside down and that the stopcocks are closed to air. Aspirate blood to the stopcock. Slowly flush fluid from the pressure bag to clear the line. Avoid pressurizing the system until it is connected to the patient and fully functional.
Confusing the arterial with the venous line	Clearly label arterial line tubing as "ARTERIAL" to avoid mistaking as the line for venous access.
Hemorrhage from an accidental disconnection	Set monitor alarms. Check tubing and connectors for tightness.
Unable to wedge	If the balloon inflates but cannot be withdrawn, suspect rupture: label the balloon port as follows: "Ruptured—DO NOT WEDGE." If the balloon functions normally, the catheter may need to be advanced: notify the receiving facility on arrival.
Continuous PCWP waveform	Verify that the balloon tip has not been left inflated. Have the patient cough or change position to free the catheter tip. Fast flush the PAC to free the catheter tip from the wall of the pulmonary artery. Notify the sending physician.
Blood in the line	Confirm that the flush solution container is not empty. Check that the stopcocks are in the correct position and that the tubing is securely connected. Verify that the patient's blood pressure has not exceeded that in the pressure bag (the pressure bag is properly inflated).
Pulmonary artery waveform now appears to be a right ventricular waveform	Observe for cardiac irritability. If ectopy is compromising hemodynamic status, pull back the catheter into the right atrium (15- to 20-cm marking at the introducer). Inflate the balloon to float the tip of the PAC back into the pulmonary artery. Notify the sending physician.
Loss of circulation distal to the arterial line site	Notify medical control. May need to remove the arterial line.

Clotted	Do not attempt to forcefully flush a clotted central venous or arterial line. Cap the line. Label the line as "clotted."

Abbreviations: PAC, pulmonary artery catheter; PCWP, pulmonary capillary wedge pressure.

TABLE 14-4 Relationship Between Shock Type and Invasive Hemodynamic Measurements

	Shock Type		
Measurement	Hypovolemic	Cardiogenic	Distributive
Preload (CVP, PCWP)	Decreased	Increased	Decreased
Afterload (SVR, PVR)	Increased	Increased	Decreased
Cardiac output	Decreased	Decreased	Increased
Svo_2 or $Scvo_2$	Decreased	Decreased	Increased
Blood pressure (MAP)	Decreased	Increased	Decreased

Abbreviations: CVP, central venous pressure; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; $Scvo_2$, mixed central venous oxygen saturation; Svo_2, mixed venous oxygen saturation; SVR, systemic vascular resistance.

Recognition of Shock

Shock is a common cause of morbidity in the critical care setting. Hemodynamic monitoring can help CCTPs to differentiate between various types of shock and assess the effectiveness of treatment during transport. There are three recognized shock states: hypovolemic, cardiogenic, and distributive. Hypovolemic shock includes volume loss from acute or chronic hemorrhage, excessive diuresis, third-space losses, and any other condition that depletes intravascular volume. Cardiogenic shock causes are diverse and include myocardial depression from ischemia and/or infarcts, cardiomyopathies, stunning following cardiac surgery, arrhythmias that decrease flow, mechanical abnormalities leading to pump failure, and obstructive conditions such as massive pulmonary embolism, tension pneumothorax, and pericardial tamponade. Distributive shock states include sepsis, systemic inflammatory response syndrome, toxic shock syndrome, anaphylaxis, poisonings, neurogenic conditions such as spinal cord injury, and other vasodilatory etiologies. Each has classic hemodynamic findings that help distinguish among them. Because shock is characterized by reduced systemic tissue perfusion, the end result is decreased tissue perfusion. Prompt recognition and reversal of shock avoids a rapidly irreversible sequence of cell death, end-organ damage, lactic acidosis resulting from anaerobic metabolism, multisystem organ failure, and death.

Two hemodynamic parameters distinguish the different types of shock: CO and SVR. In hypovolemic shock, preload is decreased as a consequence of depleted intravascular volume. Decreased preload leads to a decline in SV, which results in decreased CO. To maintain end-organ perfusion, SVR increases to compensate for the declining CO. Cardiogenic shock involves a complex cascade of events resulting from pump failure leading to decreased CO. The SVR typically increases in an effort to maintain CO, which further exacerbates pump failure and leads to increased preload and a relative total hypervolemic condition. In distributive shock, vasodilation is often the primary culprit and is observed by a significantly decreased SVR. When afterload declines dramatically, CO increases. **Table 14-4** summarizes the findings expected with each of the three shock states.

Flight Considerations

Concerns pertaining to flight physiology include the hypoxic effects of altitude and gas expansion. Even in pressurized cabins, oxygen saturation in healthy people declines by a mean of 5.5%, and the saturation often approaches 0.90 (90%). Patients with reduced oxygen saturation on the ground can be expected to experience further declines. Close monitoring of oxygen saturation will enable CCTPs to respond to changes in a patient's condition and maintain sufficient levels of inspired oxygen to ensure adequate saturation.

Physiologic changes that may be evident to CCTPs in response to lowered oxygen levels at high altitude include a chemoreceptor-induced increase in tidal volume and, if respiratory compensation is inadequate, an increased CO achieved mainly through an increased heart rate. Hypoxia and increased sympathetic nervous system tone predispose patients to arrhythmias.

Gas expansion in flight occurs in accordance with Boyle's law (the volume to which a given quantity of gas is compressed is inversely proportional to the surrounding atmospheric pressure). Pneumothoraces may become tension pneumothoraces with significant hemodynamic compromise. Air should be removed from IV bags because it will expand, potentially increasing infusion rates. Pumps should be used for medication infusions. Zeroing of transducers is recommended when cruising altitude is reached and with changes of altitude of 1,000′ or more.

Summary

Hemodynamic monitoring provides CCTPs with multiple measures of the cardiovascular status of their patients. Proper use of these clues to guide patient management requires technical skill in the correct use of the equipment and an understanding of the meaning of these data. Experienced CCTPs will use multiple methods (such as physical assessment findings and lab test results) to confirm that the hemodynamic measurements obtained reflect perfusion. Hemodynamic monitoring provides one piece of the clinical picture needed to form an overall impression and diagnosis. The most important tool for hemodynamic monitoring will always be the CCTP's own knowledge and experience.

Case Study

A 68-YEAR-OLD MAN WITH PULMONARY EDEMA was brought to the emergency department of a community hospital by EMS. Paramedics found him with acute shortness of breath and intubated him in the field when he failed to respond to nitroglycerin, furosemide, and morphine. He is presently in the community hospital ICU awaiting your arrival for ground transport to a heart failure program at a tertiary center 2 hours away. The referring hospital personnel advise that an arterial line and PAC have been placed and that they are attempting to lower the patient's BP while they await your arrival. There is no evidence of an acute myocardial infarction. A chest radiograph revealed diffuse interstitial edema consistent with pulmonary edema. The results of lab work, including cardiac markers, electrolytes, and renal function tests, were normal.

On arrival, you confirm the information previously reported. You note the patient ventilating well on a fraction of inspired oxygen of 0.4, a tidal volume of 7 mL/kg, a respiratory rate of 14 breaths/min in the synchronized intermittent mandatory ventilation (SIMV) mode, and a PEEP of 5 cm H_2O. Continuous infusions of propofol, fentanyl, and nitroglycerin are running. The nitroglycerin is infusing at 200 μg/min. The registered nurse reports that escalating doses of furosemide have not resulted in any increased urine output. The following hemodynamic readings were taken 5 minutes before your arrival at the bedside:

- Pulse rate: 100 beats/min

- Blood pressure (SBP/DBP/mean): 220/110/135 mm Hg
- Pulmonary artery pressure (S/D/M [systolic/diastolic/mean]): 57/34/40 mm Hg
- PCWP: 28 mm Hg
- CVP: 20 mm Hg
- CO: 3.9 L/min
- Cardiac index: 1.7 L/min/m^2
- SVR: 2,359 dyne/s/cm^{-5}

1. What are the implications of these hemodynamic values?
2. What interventions would help stabilize this patient's condition for transport?
3. Are there other hemodynamic data that would be helpful?

Analysis

Familiarity with hemodynamic findings associated with various forms of shock would immediately point to cardiogenic shock in this patient. An initial decline in myocardial pumping efficiency from a yet undetermined cause triggered a cascade of events beginning with vasoconstriction and progressing to hypervolemia that ultimately resulted in pulmonary edema. Vasoconstriction is seen in the elevated afterload (SVR) and hypertension; hypervolemia is evidenced by elevated preload values (CVP and PCWP). The CO is likely low because the heart is unable to pump against the incredibly high afterload. Urine output is likely compromised by renal retention of sodium and water in response to the low CO. The values seen in this case example are, in fact, classic values seen in patients with cardiogenic shock. As their condition deteriorates, they will ultimately become hypotensive and acidotic and will die if untreated.

The first intervention suggested by the values obtained with the PAC is use of an agent to reduce the high afterload state. Staff reported attempting to lower the patient's blood pressure with nitroglycerin, but current dosing is generally considered maximal. Nitroglycerin is primarily a venodilating agent with minimal arterial dilating properties except at extremely high doses. Given the patient's degree of hypertension, which persists even with the nitroglycerin infusion, significant afterload reduction will require an agent with more potent arterial vasodilating properties. The transport crew consulted with providers caring for the patient and decided to initiate an infusion of sodium nitroprusside. Nitroprusside has venous and arterial dilating properties and is a potent antihypertensive agent. Following initiation of a nitroprusside drip, gradually titrated up to 3 μg/kg/min, the patient's MAP declined to 95 mm Hg, CVP to 14 mm Hg, and PCWP to 24 mm Hg. The CO doubled to 7.8 L/min. The calculated SVR was then 831 dyne/s/cm^{-5}. With the improved CO, the patient's urine output immediately increased. Had CO remained low once afterload reduction was achieved, the transport crew would likely have sought to initiate an inotropic infusion. Dobutamine is the inotropic agent of choice for first-line treatment of low CO associated with heart failure.

Not all hemodynamic parameters discussed in this chapter were used in evaluating this patient. Patient problems and potential treatment options under consideration guide the hemodynamic measurements and calculations used. With the data used by the CCTPs receiving this patient, a clear picture of pump failure with low CO emerges. One question not apparent from these data is whether the perfusion needs of the patient are being met by the present CO. If oxygen demands exceed supply, the patient's condition will deteriorate and most likely will do so with the additional stress of transport.

Clearly, the patient's CO is low. Mixed venous oxygen saturation would indicate whether the present CO is meeting the perfusion needs of the patient. As described, some PACs have a fiberoptic oximetric

tip that continuously displays the mixed venous oxygen saturation. Because this PAC did not, the transport crew requested that the hospital staff send a blood specimen from the pulmonary artery port of the PAC for blood gas analysis. The results showed a mixed venous oxygen saturation of 0.49 (49%), well below the normal range of 0.60 to 0.80 (60% to 80%). The result for a repeated specimen drawn after hemodynamics appeared more stable following initiation of the nitroprusside infusion was 0.64 (64%), a dramatic improvement reflecting adequate tissue perfusion.

Prep Kit

Ready for Review

- Invasive hemodynamic monitoring is used to assess the heart, vascular network, and fluid volume status. It is used in conjunction with, not as a replacement for, physical assessment techniques such as capillary refill, skin color, skin turgor, body temperature, heart and lung sounds, and mental status.
- Invasive hemodynamic measurements are obtained via a closed catheter system, which is typically placed in the patient by a physician. Catheter placement is usually outside the scope of CCTPs. Measurements are read on a monitor that is connected to a transducer on the catheter.
- Asepsis is critical in the care of patients with central lines and hemodynamic monitoring equipment. Five key practices decrease the incidence of catheter-related bloodstream infections: hand hygiene, maximal barrier precautions during insertion, chlorhexidine skin antisepsis, optimal catheter site selection, and prompt removal of unnecessary lines.
- Blood pressure is measured directly through the use of an arterial line or indirectly through the use of a device that senses pulsatile flow such as a sphygmomanometer, which measures systolic and diastolic pressures. Indirect measurements assess flow, and direct monitoring devices measure pressure. Pressure and flow are different parameters, so readings obtained using indirect and direct methods cannot be expected to correlate exactly.
- Systolic blood pressure is the measure of pressure on the arterial walls during systole; diastolic blood pressure is the measure of pressure on the walls of the arteries during diastole. The formula for blood pressure is cardiac output multiplied by systemic vascular resistance (BP = CO × SVR).
- To avoid errors when using indirect monitoring devices to measure blood pressure, CCTPs should be careful to use the correct blood pressure cuff size for the patient and to position the patient's extremity in which blood pressure is measured at mid-heart level.
- Transport monitors often include noninvasive blood pressure (NIBP) units, which automatically measure indirect blood pressure at predetermined intervals. The NIBP units measure heart rate and mean arterial pressure (MAP) only. Computer software then calculates systolic and diastolic blood pressures.
- CCTPs should evaluate the heart rate obtained from the NIBP unit against the actual patient pulse, the heart rate displayed on a pulse oximeter, or an ECG heart rate display to determine the accuracy of the NIBP measurements. If the NIBP heart rate differs significantly from the actual patient heart rate, the calculated systolic and diastolic blood pressures should be considered unreliable.
- Because the MAP is actually measured by the NIBP unit, this value is often used in making patient care and treatment decisions.
- Heart rate is a key sign of hemodynamic instability in the emergency or critical care patient. A heart rate obtained from a pulse oximeter is more reliable than manual assessment by the CCTP, particularly

in the noisy environment of an ICU or a CCTP vehicle.

- The Allen test is used to assess extremity perfusion and test ulnar artery function.
- Central venous lines provide access to the core vessels of the body. The right internal jugular vein is the easiest site to access. Other common insertion sites are the subclavian, brachial, and femoral veins. Subclavian sites have the lowest infection risk; femoral sites have the highest risk for infection, and their use is strongly discouraged in adults.
- Central venous pressure (CVP) monitoring is used to determine right ventricular preload and intravascular volume status and to assess right-sided heart function. The normal range for the CVP is 2 to 6 mm Hg. To ensure accuracy, CVP measurement should be obtained during end expiration.
- Central venous lines often include outlet ports, which are used for rapid fluid replacement, medication administration, rapid access to central circulation, and invasive monitoring.
- Contraindications for central venous access are few. They include significant coagulopathy, local trauma to the site of insertion, and infection at the site of insertion.
- The most common complication associated with the insertion of a central line is pneumothorax or hemothorax from an unsuccessful attempt at subclavian line insertion. Other complications range from bleeding, to infection, and to blood withdrawal problems.
- The triple-lumen catheter is the most commonly used central line. The lumens can be used for fluid and drug administration and for CVP monitoring. For some patients, one port may be reserved for total parenteral nutrition; that port should not be used during transport.
- Some central venous catheters have one or two access ports, and some are installed under the skin (tunneled). Consult the receiving facility and local protocols for direction in using tunneled central lines.
- CCTPs should be familiar with placement; use; maintenance required for different types of catheters, including the HICKMAN® catheter, the GROSHONG® catheter, the port-a-cath, and the peripherally inserted central catheter (PICC); and the possible complications and advantages and disadvantages of each catheter. All complications should be reported to the receiving facility as soon as possible.
- Although central venous lines are rarely placed by CCTPs, knowledge of how they are placed will help CCTPs care for patients who have a central venous line.
- The Seldinger technique is the most common technique used for line insertion. Specific procedures are used for femoral, internal jugular, and subclavian vein insertion.
- Common problems that occur when inserting a central venous line include sluggish infusion, inability to withdraw blood, and catheter damage.
- Direct blood pressure measurement using an arterial line is indicated for patients who require constant blood pressure measurement, particularly patients who are in shock and are not responding to therapy. Arterial lines are also used for patients receiving vasoactive or antihypertensive infusions.
- Contraindications for the use of an arterial line include ischemia of the extremity, infection at the puncture site, Raynaud disease, and prior vascular surgery in the area of the insertion site.
- CCTPs generally do not place arterial lines, but they may need to assist in the procedure; therefore, they should be familiar with the insertion procedure.
- A pulmonary artery catheter (PAC), also known as a Swan-Ganz catheter or “swan,” is inserted into the venous system of a patient. Although CCTPs do not insert PACs, knowledge of the procedure is useful in providing care.

- PACs have multiple lumens that terminate at different points along the catheter and correspond to different locations in the heart. PACs are marked at 10-cm intervals beginning at the distal tip of the catheter. CCTPs should note the centimeter measurement of the PAC at the introducer when initially assessing a patient. This information will help to determine if the catheter moves into or out of the patient during transport.
- The PAC measures right atrial (RA) and pulmonary artery pressure. The RA pressures are displayed as mean pressures; pulmonary artery pressures are displayed as systolic, diastolic, and mean values. Inflation of the PAC balloon blocks blood flow from the right ventricle behind the balloon tip and results in the appearance of a pulmonary capillary wedge pressure (PCWP) waveform on the monitor. The PCWP waveform is measured as a mean value.
- Complications of PAC insertion are nearly the same as those of central line insertion. Probably the greatest potential complication of PAC use is misinterpretation of the data by clinicians, resulting in inappropriate therapy and potentially worsening outcomes.
- A thermistor is connected to the distal end of the PAC. The thermistor measures thermodilution cardiac output and provides a continuous reading of the pulmonary artery temperature, which is considered the gold standard for core body temperature measurement.
- Indications for the use of PACs are based on clinician experience, not outcome studies. Clinicians are advised to consider the risks and benefits for critically ill patients on an individual basis.
- Contraindications to the use of a PAC include significant coagulopathy, local trauma to the site of insertion, infection at the site of insertion, and inability to float the PAC into the pulmonary artery.
- CCTPs should be familiar with normal hemodynamic ranges and values and the lines from which the measurements are obtained.
- Cardiac output (CO) is the product of stroke volume (SV) and heart rate: (CO = SV × Heart Rate). The SV is determined by preload, afterload, and contractility of the heart. Because the SV is determined by three components, heart rate contributes more to and has a more immediate effect on CO than any one component of SV. When confronted with low CO, CCTPs should investigate heart rate before evaluating the components of SV.
- Some hemodynamic values are indexed to account for differences in body size; for example, a cardiac index is the patient's cardiac output divided by his or her total body surface area.
- Preload is the degree of myocardial fiber stretch induced by volume in the ventricle at the end of diastole. Hypovolemia, venodilation, intrathoracic pressure changes, and intrapericardial pressure changes result in decreased preload. Hypervolemia and vasoconstriction result in increased preload.
- Right ventricular preload is measured by central venous pressure. Left ventricular preload is measured by pulmonary capillary wedge pressure or pulmonary artery diastolic pressure.
- Afterload is the resistance the ventricles must overcome to eject blood. Three factors typically determine afterload: outflow obstructions, vascular tone, and blood viscosity. Outflow obstructions, vasoconstriction, and polycythemia increase afterload. Vasodilation reduces afterload. Lowered hemoglobin levels are often tolerated in critically ill patients because cardiac output may be improved by decreasing afterload.
- Contractility is the inotropic state of the heart or the force with which the heart muscle contracts. Contractility is assessed with newer Doppler and ultrasound devices. Six factors influence contractility: myocardial muscle health, autonomic nervous system, metabolic states, ion environment, pharmacologic agents, and heart rate.

- Central venous pressure (CVP) is a measure of the vena caval and right atrial pressures. It is measured from a central venous line or pulmonary artery catheter. A CVP waveform tracing has systolic and diastolic values, but only the mean is used in clinical practice because the numbers are extremely low.
- The normal range for CVP is 2 to 6 mm Hg. Fluids are often administered to patients with CVP values below 5 to 8 mm Hg to increase their preload.
- Continuous blood pressure monitoring through an arterial line provides CCTPs with constantly updated systolic and diastolic blood pressure and mean arterial pressure values and an arterial waveform. Components of the arterial waveform include the dicrotic notch, mean pressure, systole, diastole, systolic pulse pressure, and diastolic pulse pressure.
- Mean arterial pressure (MAP) is a function of the systolic blood pressure and diastolic blood pressure. It is calculated by the transport monitor software or the CCTP. An MAP between 70 and 105 mm Hg is needed to satisfactorily perfuse the body's tissues; most medical orders require titration of drugs or administration of fluids to maintain an MAP of 70 mm Hg or greater.
- Pulse pressure is another value calculated using systolic and diastolic blood pressures. It is the mathematical difference between these values. Normal values are 40 to 60 mm Hg.
- Pulmonary artery catheter measurements include right ventricular pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output. CCTPs should know the normal values for each of these measurements and how to evaluate each of the measurements.
- CCTPs should be able to recognize a right ventricular waveform and its characteristic low diastolic pressure, which distinguishes it from a pulmonary artery pressure waveform.
- A CCTP who observes a sudden elevation in pulmonary artery pressures should immediately assess and correct any issues with oxygen saturation or ventilation. If pulmonary artery diastolic pressures suddenly drop, the catheter position should be reevaluated to determine if the PAC has migrated into the right ventricle.
- CCTPs should also know how to read a pulmonary capillary wedge pressure (PCWP) waveform. When a PCWP waveform is observed, the tracing can be frozen on the monitor screen, and the balloon should be allowed to passively deflate.
- The Frank-Starling law states that there is a relationship between the distention of the ventricular myocardium and the force of the contraction. As the ventricular preload increases, the force of contraction of the myocardium also increases due to the increased stretch of the myocardial fibers. The law describes how administration of fluids can improve cardiac output to a certain point, which varies among individual patients. Additional fluids given beyond that point worsen cardiac output.
- The thermodilution method is typically used to assess cardiac output with a pulmonary artery catheter. Most transport monitors are not capable of performing thermodilution cardiac output measurements during transport.
- Cardiac output can be calculated or estimated by using the Fick principle. The formula used is as follows:

$$CO = \frac{\text{Oxygen Consumption}}{\text{Arteriovenous Oxygen Content Difference}}$$

- Pulmonary vascular resistance (PVR) is the resistance of the pulmonary vascular circuit that opposes flow out of the right ventricle and pulmonary artery. It is commonly referred to as right-sided heart afterload. The PVR may be elevated in patients with pulmonary hypertension or mitral valve stenosis.
- Systemic vascular resistance (SVR) is the total impedance to blood flow felt by the left ventricle, commonly referred to as afterload. Calculation of the SVR is helpful in determining the cause of shock.

- The amount of blood ejected in each stroke of a ventricle is measured in milliliters and recorded as the stroke volume (SV). A normal SV is 60 to 100 mL/beat.
- Mixed venous oxygen saturation (SvO_2) reflects the global balance between oxygen delivery and consumption. It is measured at the distal end of the PAC because the pulmonary artery is the most appropriate place to sample a mixture of all the blood returning from the body. Four components contribute to SvO_2: total hemoglobin level, arterial oxygen saturation, cardiac output, and oxygen consumption.
- Mixed central venous oxygen saturation ($ScvO_2$) reflects the balance between oxygen supply and delivery in the brain and upper body.
- Before moving a patient with hemodynamic monitoring equipment in place, CCTPs should examine the equipment in use, note the values displayed, and trace the tubing from each transducer to the pressure-monitoring catheter. The catheter site should be examined to ensure that it is visibly clean, dry, and covered by an occlusive dressing and its condition documented. A CCTP who is unfamiliar with the operation of the stopcocks and catheters in use should ask the clinician at the bedside to explain their operation.
- The patient can be transferred to a stretcher before or after transducers are connected to the transport monitor. A deliberate, orderly manner is needed when moving connections from the hospital monitor to the transport monitor to avoid periods when the patient is not connected to any monitor. Agency or service policy should be followed for securing monitoring lines and catheters during transport. Monitoring alarms need to be set to protect against accidental dislodgement or dislocation of invasive monitoring catheters.
- The most common cause of error in obtaining direct hemodynamic pressure measurements is incorrect leveling of the transducer. To ensure accurate pressure readings, the transducer must be at mid-heart level. The transducer must be releveled with each position change of the patient. Because errors in leveling can significantly impact treatment decisions, all leveling should be performed using a carpenter's level or a leveling tool designed for hemodynamic monitoring applications.
- Pressure transducers must be "zeroed" to eliminate the effects of atmospheric pressure on the measurements obtained. Depending on flight patterns and altitudes, transducers may be zeroed at initial connection to the transport monitor, at cruising altitude, and at the destination.
- Direct pressure measurements are affected by changes in intrathoracic pressure. To obtain accurate measurements, readings should be taken at end expiration (the lower portions, or valleys, of the rise-and-fall pattern for mechanically ventilated patients; and the peaks of the rise-and-fall pattern for spontaneously breathing patients).
- CCTPs should become familiar with transport monitors, cables, and transducers. Cables must be compatible with the equipment used at the referring and receiving hospitals.
- CCTPs should be familiar with common problems encountered during hemodynamic monitoring and actions to resolve them. The most common complication during transport of a patient with a central line, an arterial line, or a PAC is accidental dislodgement or removal of the line or catheter. If an invasive line is completely removed from a patient, direct pressure is immediately applied to the insertion site and held until the bleeding stops. If a catheter dislodges but does not come completely out of the patient, replacement should not be attempted. An attempt should be made to aspirate blood from each lumen to determine if each one remains in a blood vessel. Options should be discussed with medical control. A catheter that must be removed is gently withdrawn from the patient and bleeding is controlled at the insertion site.

- Prompt recognition and reversal of shock avoid a rapidly irreversible sequence that can result in the patient's death. Understanding the relationship between hemodynamic measurements and the different types of shock can help CCTPs recognize the type of shock and assess the effectiveness of treatment during transport.
- Oxygen saturation and gas expansion are concerns during flight. CCTPs should closely monitor the patient's oxygen saturation level to respond to changes in the patient's condition and maintain sufficient levels of inspired oxygen. Gas expansion should be addressed by removing air from IV bags, using pumps for medication infusions, and zeroing transducers.

Vital Vocabulary

afterload The tension or stress that develops in the ventricles during systole; measured by pulmonary and systemic vascular resistance.

Allen test A technique in which the patient's hand is initially held above the head while the fist is clenched and the radial and ulnar arteries are compressed; the hand is then lowered and the fist is opened, ulnar pressure is released, and radial pressure is maintained; after ulnar pressure is released, color should return to the hand within 6 seconds.

arterial lines Catheters inserted into the patient's arterial vascular system for the purpose of producing a waveform with pressure measurements; also called an A-line.

cardiac index A hemodynamic value that adjusts a patient's cardiac output to take into account his or her total body surface area.

cardiac output (CO) The amount of blood pumped out of the heart in 1 minute; the product of the stroke volume (average 70 mL) and the heart rate (average 60 to 100 beats/min).

central venous lines IV access catheters that terminate in central circulation, usually just proximal to the right atrium.

central venous pressure (CVP) The pressure in the superior vena cava (average 2 to 6 cm H_2O); reflects the pressure in the venous system when the blood is returned to the right atrium; indicative of a patient's fluid volume status and right-sided heart performance.

dampened waveform A hemodynamic pressure waveform that appears to have lost crisp deflections.

diastolic blood pressure (DBP) The trough or resting pressure in the arterial system that occurs during ventricular diastole.

dicrotic notch The brief increase in aortic pressure reflected in a notching of the wave; caused by the sudden closure and spring back of the aortic valve leaflets; signals the start of diastole.

Fick principle A method of indirectly determining cardiac output, in which the amount of oxygen uptake of blood as it passes through the lungs is equal to the oxygen concentration difference between mixed venous and arterial blood; the formula uses assumed values for oxygen consumption derived from basal metabolic studies on healthy subjects, which may or may not be valid in critically ill patients.

Frank-Starling law A law of physiology that states that the greater the myocardium is stretched, the greater the force of contraction.

invasive hemodynamic monitoring A term describing methods for assessing the physiologic condition of the three principle components of the cardiovascular system: heart, vascular network, and fluid volume; mainly assesses the capability of a patient's heart to pump the requisite amount of blood to the

body, but also can assess compliance, tone, resistance of the vascular network, and fluid status; includes a variety of pressure values and other measurements.

left ventricular end-diastolic pressure The pressure exerted on the left ventricle at the end of diastole; the normal value is 4 to 12 mm Hg and is measured by using a pulmonary artery catheter.

left ventricular stroke work index A calculation of the contractility of the left ventricle indexed to the patient's body surface area; same as stroke volume index.

mean arterial pressure (MAP) Represents the average (that is, constant) pressure in the arterial vasculature; a function of systolic and diastolic blood pressures.

mixed central venous oxygen saturation ($Scvo_2$) The percentage of oxygen bound to hemoglobin in blood returning to the right side of the heart from the head and upper body, representative of oxygen extraction by the head and upper extremities.

mixed venous oxygen saturation (Svo_2) The percentage of oxygen bound to hemoglobin in blood returning to the right side of the heart, representative of global oxygen extraction.

phlebostatic axis An imaginary point located at the fourth intercostal space, mid-chest level, which serves as an external landmark for the right atrium.

preload The end-diastolic stretch of the muscle fibers of the ventricle; measured by right atrial pressure or central venous pressure and wedge pressure.

pulmonary artery catheter (PAC) A catheter with a balloon near its tip that is passed through a vein into the right side of the heart, through the right ventricle, and into the pulmonary artery; records the pressure transmitted back from the left atrium; also referred to as a Swan-Ganz catheter.

pulmonary artery pressure The pressure measured in the pulmonary artery, usually displayed with a pressure waveform and digital systolic, diastolic, and mean values.

pulmonary capillary wedge pressure (PCWP) The mean pressure measured while occluding the pulmonary artery with a balloon-tipped catheter proximal to the site of measurement; reflects left atrial pressure (also called pulmonary artery wedge pressure and pulmonary artery occlusion pressure).

pulmonary vascular resistance (PVR) The resistance or impedance to ejection of the right ventricle of the heart.

pulse pressure The difference between systolic and diastolic blood pressures.

referencing The process of ensuring that the hemodynamic pressure transducer is at the level of the left atrium.

right ventricular pressure The pressure in the right ventricle that consists of systolic, diastolic, and mean pressures; important during insertion and use of a PAC.

right ventricular stroke work index A calculation of contractility of the right ventricle indexed to the patient's body surface area.

Seldinger technique The most common technique for inserting a central venous line; involves inserting a needle with a syringe, then inserting a guide wire into the needle. Once the guide wire is in place, the needle is removed, an incision is made, and a catheter is inserted over the guide wire; the guide wire is then removed.

stroke volume (SV) The amount of blood ejected by the ventricles during each contraction; varies between 60 and 130 mL/beat, with the average being 70 mL.

stroke volume index A calculation of contractility of the left ventricle indexed to the patient's body

surface area; same as the left ventricular stroke work index.

systemic vascular resistance (SVR) The resistance or impedance to ejection of the left ventricle of the heart.

systolic blood pressure (SBP) Peak pressure in the arterial system that occurs during ventricular ejection or systole.

thermistor The apparatus used for quickly determining very small changes in temperature.

triple-lumen catheter A type of catheter consisting of three distinct continuous tubes that allow for pressure monitoring, blood sampling, and fluid and drug administration.

zeroing A process of calibrating a pressure transducer to eliminate extraneous atmospheric and hydrostatic pressures from the data being measured.

Bibliography

Ahrens T. Continuous mixed venous (SvO_2) monitoring: too expensive or indispensible? *Crit Care Nurs Clin North Am.* 1999;11:33–48.

Ahrens T. Hemodynamic monitoring. *Crit Care Nurs Clin North Am.* 1999;11:19–31.

Brenner M, Safani M, Chan P. *Critical Care Medicine.* Mission Viejo, CA: Current Clinical Strategies; 1997.

Bridges EJ. Monitoring pulmonary artery pressures: just the facts. *Crit Care Nurse.* 2000;20:59–80.

Chang D. *Clinical Application of Mechanical Ventilation.* Albany, NY: Delmar; 1997.

Haupt MT, Kaufman BS, Carlson RW. Fluid resuscitation in patients with increased vascular permeability. *Crit Care Clin.* 1992;8:341–353.

Lanken P. *The Intensive Care Unit Manual.* Philadelphia, PA: WB Saunders Co; 2001.

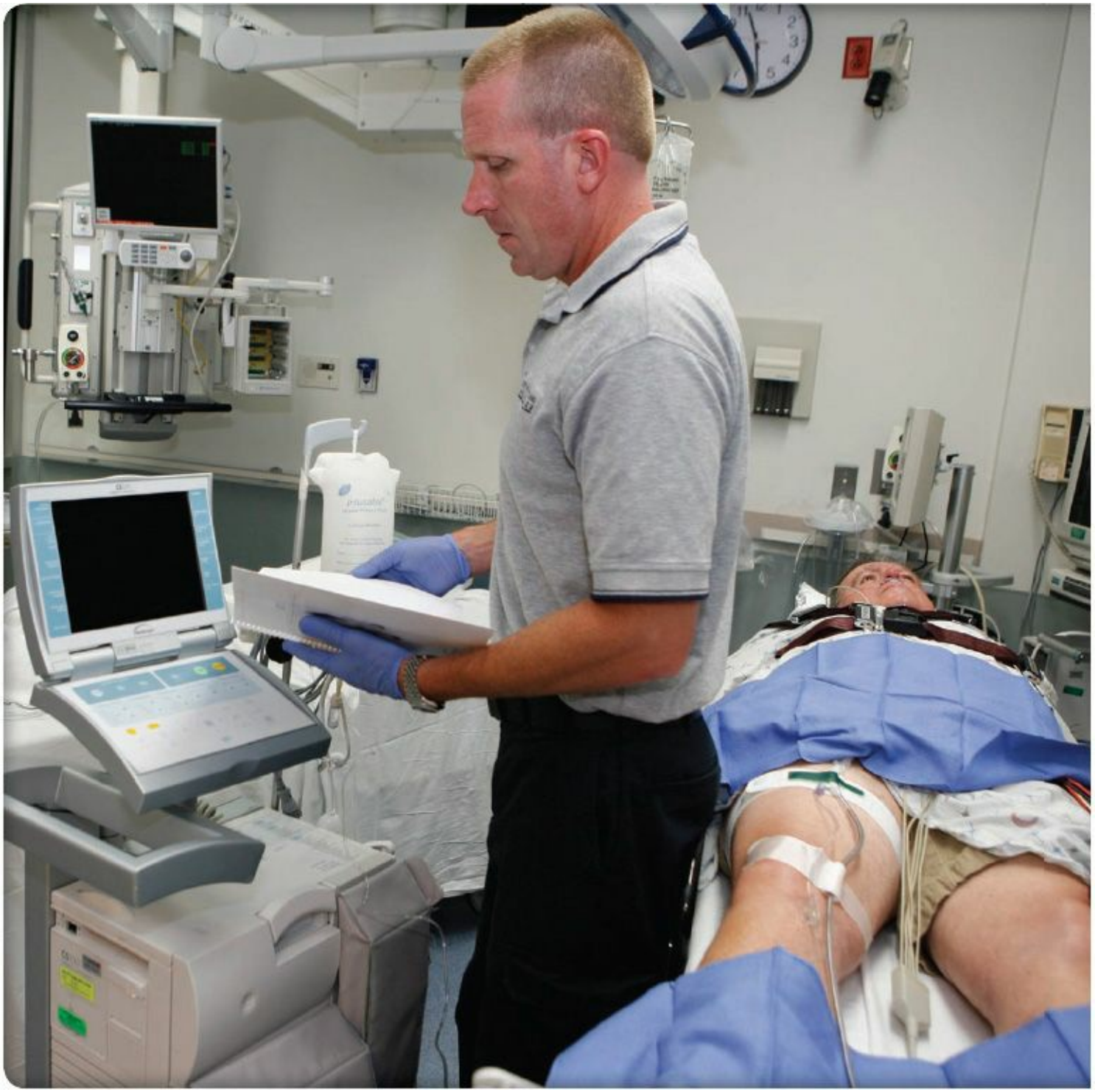
Mermel LA. Prevention of intravascular catheter–related infections. *Ann Intern Med.* 2000;132:391–402.

Parsons P, Wiener-Kronish J. *Critical Care Secrets*, 2nd ed. Philadelphia, PA: Hanley and Belfus; 1998.

Shoemaker W, Velmahos G, Demetriades D. *Procedures and Monitoring for the Critically Ill.* Philadelphia, PA: WB Saunders Co; 2002.

Smeltzer S, Bare B. *Textbook of Medical Surgical Nursing*, 10th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2004.

Soufir L, Timsit JF, Mahe C, Carlet J, Regnier B, Chevret S. Attributable morbidity and mortality of catheter-related septicemia in critically ill patients: a matched, risk-adjusted, cohort study. *Infect Control Hosp Epidemiol.* 1999;20:396–401.



Intra-aortic Balloon Pump Therapy

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Objectives

1. List the five phases of the cardiac cycle and describe the associated events that affect:
   - Blood volume in the heart chambers
   - Coronary perfusion as it relates to the cardiac cycle
   - Myocardial oxygen demand
   - The arterial pressure waveform
   - The physiologic effects of heart failure (p 600–601)
2. Generally describe the setup and operation of an intra-aortic balloon pump (IABP) (p 598).
3. Describe the mechanics of the IABP, including the following:
   - Basic design and functions of the IABP
   - Catheter structure and function
   - Balloon structure and function (p 603)
4. List indications for IABP therapy (p 609).
5. List contraindications to IABP therapy (p 611).
6. Describe methods and locations of insertion (p 613).
7. Discuss complications from IABP insertion (p 617–619).
8. Discuss timing of balloon inflation and deflation (p 608–609).
9. Explain modes of IABP timing (p 605–606).
10. Define these primary physiologic effects achieved by inflation and deflation of the intra-aortic balloon:
    - Diastolic augmentation
    - Systolic afterload reduction
    - Improved coronary flow (p 603–604)
11. Identify improper balloon inflation timing and discuss the hemodynamic effects and corrective action during each of the following stages:
    - Early inflation
    - Early deflation
    - Late inflation
    - Late deflation (p 608–609)
12. Discuss transport considerations and potential complications of IABP therapy.
    - Altitude changes
    - Cardiac arrest during transport Depletion of helium

- Console failure
- Balloon rupture
- Limb ischemia
- Insertion site bleeding (p 612–621)

13. Discuss cardiac-assist devices, including:
    - Extracorporeal membrane oxygenation (ECMO)
    - Implanted left ventricular-assist device (LVAD), right ventricular-assist device (RVAD), and biventricular-assist device (BiVAD) (p 619–621)

Introduction

As described in the preceding chapters, intensive cardiac care requires special technical knowledge and skills, especially for the transport of patients receiving **intra-aortic balloon pump (IABP) therapy**, in which a balloon connected to a pump via a catheter has been inserted into the aorta to provide temporary assistance to a failing heart. Although the transport of the patient during IABP therapy is a daunting task for trained transport professionals, with proper study and education, CCTPs can obtain the knowledge and skill necessary to feel more comfortable in this situation. It is important to note, however, that IABP therapy is a highly advanced skill. This chapter discusses maintaining IABP therapy, recognizing and correcting problems, and performing emergency procedures for IABP failure and is not intended to be all inclusive in regard to IABP therapy.

Previous chapters dealt primarily with the electrical components of the cardiac cycle. This chapter addresses a therapy for compromised mechanical (pumping) function of the heart: the IABP. Electrical and mechanical functions of the heart are interdependent, thus failure of one will quickly lead to failure of the other.

IABP therapy begins with the insertion (by a trained and credentialed physician or surgeon) of a balloon, typically via the femoral artery, into the descending thoracic aorta. The tip of the balloon catheter is located just distal (approximately 2 cm below) the takeoff of the left subclavian artery. Once in place, the balloon is connected to a console to shuttle helium gas, inflating the balloon at the onset of diastole and deflating just before systole (ventricular ejection). Inflation and deflation are timed to the cardiac cycle, resulting in significant augmentation of flow to the coronary and renal arteries during diastole (increased supply) and substantially reduced afterload during systole (decreased demand). The **timing** of inflation and deflation is crucial; if inflation and deflation do not occur at the proper time in the cardiac cycle, the device will not only fail to yield the desired results, but also could harm the patient. One of the CCTP's main roles in transporting a patient undergoing IABP therapy is to ensure that timing occurs at the proper points in the cardiac cycle. Proper timing is ensured by reading the patient's electrocardiographic (ECG) and pressure waveforms and continuously monitoring the patient's condition and all devices. If necessary, the CCTP modifies the timing. Other CCTP responsibilities include securing the patient to prevent movement of the balloon (which can create significant problems or injuries) and ensuring that all equipment has the necessary power supply to continue operating during transport **Figure 15-1**.

Figure 15-1 An important CCTP responsibility when transporting a patient with an intra-aortic balloon is to secure the patient to prevent balloon movement.

To provide proper care to a patient undergoing IABP therapy, it is necessary to have a basic understanding of normal heart anatomy and physiology, the pathophysiology of heart failure, and the benefits that IABP therapy offers. After mastering an understanding of these basic principles, CCTPs may apply them to the use of any model or brand of IABP, with some education specific to each model.

History

Intra-aortic balloon pumping, or counterpulsation, was first used for patients in cardiogenic shock in the late 1960s. Since that time, the number of indications for the intra-aortic balloon (IAB) has steadily increased. Today, IABP counterpulsation is the therapy most frequently used worldwide to assist patients with left ventricular (LV) failure. Original IABP units were crude in design with manual timing of inflation and deflation, requiring great skill and constant monitoring to ensure correct timing of inflation and deflation. With improvements in technology, today's IABP units are computer equipped to determine correct inflation and deflation timing and have benefited from miniaturization so that some IABP models will fit easily into a helicopter for transport. The IAB catheters have also become smaller, with multilumen designs allowing for arterial pressure monitoring and IAB inflation and deflation and, in some models, fiberoptic sensors. With these newer consoles, it is possible to initiate IABP therapy in the community hospital setting and transport the patient in a more stable condition to a larger facility for further diagnosis and treatment.

Cardiovascular Anatomy and Physiology

To understand the mechanics of IABP therapy and the benefits it can offer to a patient with compromised cardiovascular status, it is helpful to review some basic concepts of cardiovascular anatomy and physiology.

The heart lies in the thoracic cavity beneath the sternum; approximately two thirds of it is left of the

midsternal line. The **atrioventricular (AV) valves** (mitral and tricuspid) lie between the atria and the ventricles, and the **semilunar valves** (aortic and pulmonic) are the exits from the ventricles **Figure 15-2**. The AV valves have **chordae tendineae**, which attach the edge of the valve to the **papillary muscles** attached to the endocardium. The chordae tendineae hold the AV valves in place in their closed position and prevent them from prolapsing into the atria during systole, or ventricular contraction. In contrast, the semilunar valves do not have chordae tendineae but are attached to their respective vessels at their outer edges. The valve action depends on the valve leaflets (the thin leaflike cusps or flaps that compose the valves) being pushed together by the force of the blood trying to flow back into the heart, which keeps them in a closed position, much like a parachute opening. The opening and closing of the semilunar valves is related to the pressure changes that occur in the heart, aorta, and pulmonary artery.

The purpose of each of the cardiac valves is to prevent the backward flow of blood in the heart. If a valve is structurally defective, a backward flow of blood through the valve is referred to as valvular regurgitation or insufficiency. A valve scarred so severely that its lumen is reduced while in the open position is referred to as valvular stenosis.

The coronary arteries supply the heart muscle with oxygen. The ostea (openings) to the coronary arteries are located at the base of the aorta, just **caudal** to the attachment points of the aortic valve leaflets **Figure 15-3**. This anatomy is extremely important because their location means that the ostea are obstructed when the valve is in the open position. Therefore, although this is the time that the heart is ejecting blood, virtually none of it can get into the coronary arteries. In addition, because the coronary arteries are embedded in the myocardium, resistance in these vessels increases greatly during systole. As a result of these two factors, *virtually all coronary perfusion occurs during diastole.*

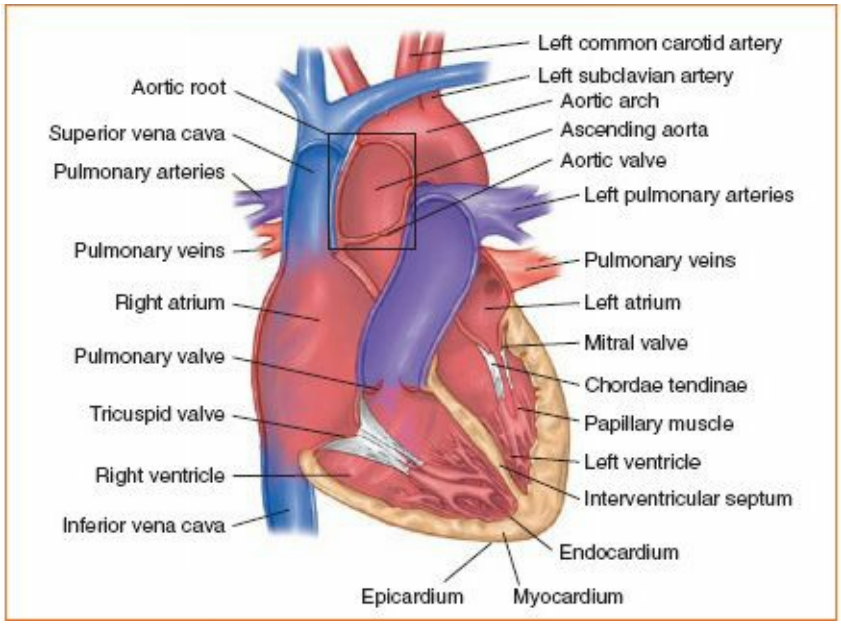


Figure 15-2 Anatomy of the heart.

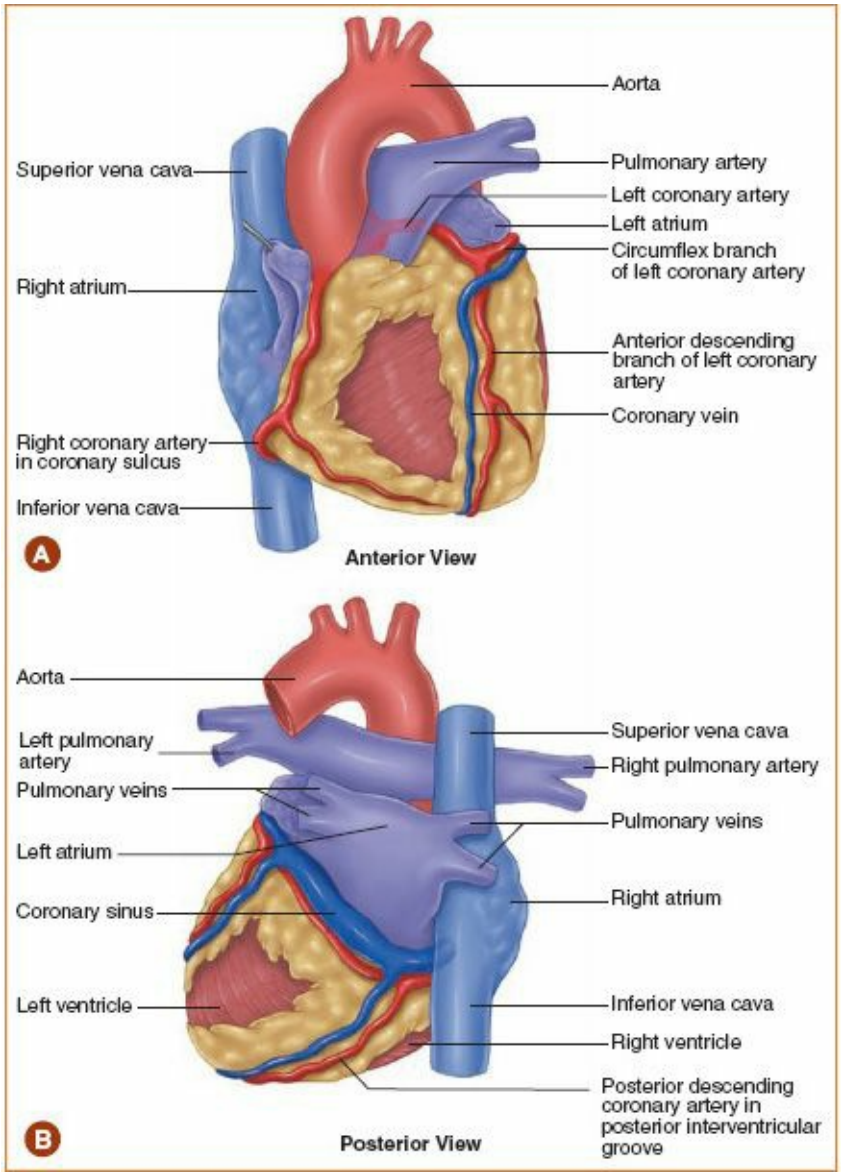


Figure 15-3 Coronary arteries. **A.** Anterior view, showing takeoff point of left and right main coronary arteries from the aorta. **B.** View from below and behind, showing the coronary sinus.

Cardiac Cycle

The cardiac cycle consists of two main stages: **systole**, the period of work or the contraction phase of the heart cycle, and **diastole**, the period of rest or the relaxation phase of the heart cycle. Systole and diastole can be further divided into five phases: atrial systole, isovolumetric contraction, ventricular ejection, isovolumetric relaxation, and ventricular filling **Figure 15-4**.

Atrial Systole

Atrial systole begins in response to an electrical impulse from the sinoatrial (SA) node, indicated as the P wave on an ECG. As the electrical impulse conducts through the atrial myocardium and internodal pathways, the atrium depolarizes and contracts. The mitral and tricuspid valves are open before atrial systole with blood already flowing passively from the atria into the ventricles. The contraction of the atria ejects additional blood into the ventricles and accounts for 20% to 30% of the ventricular filling volume (preload). This “atrial kick” raises ventricular volume and pressure and is important to the effective contraction of the ventricle because of the relationship between the stretching of the myocardium and the force of contraction of the ventricles. This relationship is known as the Frank-Starling curve. Its importance to heart failure will be discussed later.

Isovolumetric Contraction

During **isovolumetric contraction**, the electrical impulse from the atria conducts through the AV node to the ventricular conduction system, causing the ventricles to depolarize and contract. Immediately after ventricular contraction begins, ventricular pressure rises abruptly, closing the AV valves. The volume of blood in the ventricles just before these valves close is the ventricular **preload**. As mentioned, ventricular preload is important to cardiac function because of the relationship between ventricular preload and contractility. This relationship is described as the **Frank-Starling curve**.

During isovolumetric contraction, the pressure in the aorta and pulmonary arteries is higher than the pressure in the ventricles, and, therefore, the semilunar valves remain closed. The pressure in the ventricles continues to rise during ventricular contraction but, because all of the cardiac valves are closed, there is no change in ventricular blood volume. (*Isovolumetric* means "same volume.")

Ventricular Systole

Once the pressure in the pulmonary artery is equaled by the pressure in the right ventricle, the pulmonary valve opens and the right ventricle begins to eject blood into the pulmonary artery. The pulmonary valve opens slightly before the aortic valve because the pressure gradient is less for the pulmonary circulation. As the pressure in the left ventricle exceeds the pressure in the aortic root (the section of the base of the aorta that is attached to the heart, including the leaflets of the aortic valve and the openings where the coronary arteries attach), the valve leaflets open and allow the left ventricle to eject the blood into the aorta. During ventricular ejection, the aortic valve leaflets are forced against the wall of the aorta (their open position). As mentioned earlier, no coronary perfusion occurs at this stage because when the aortic valve leaflets are open, they block the coronary arteries.

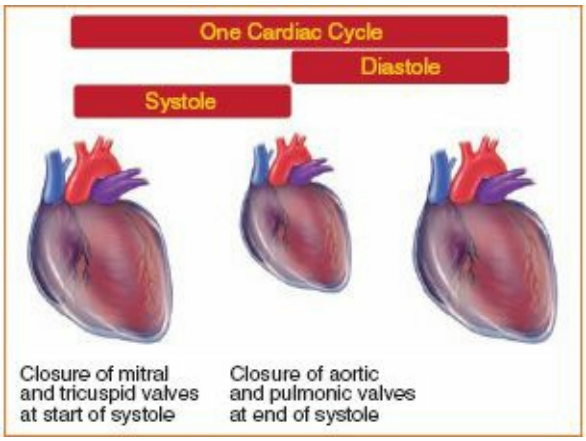


Figure 15-4 Systole and diastole.

Isovolumetric Relaxation

Near the end of ventricular ejection, the ventricles begin to relax as ventricular tissue starts to repolarize. As the ventricles relax, pressure in the ventricles drops below the arterial pressure, and the semilunar (aortic and pulmonary) valves close. The valves close because of the attempt of blood to flow backward into the heart. During this **isovolumetric relaxation** phase, the AV valves have not yet opened. Continued ventricular relaxation leads to the decrease in ventricular pressure that will soon allow the AV valves to open as atrial pressure increases.

Ventricular Filling

When pressure in the ventricles drops below the pressure in the atria, the mitral and tricuspid valves open and ventricular filling begins. Blood flows passively from the atria through the AV valves into the ventricles. When the SA node produces an electrical impulse that initiates atrial systole, the cardiac cycle

begins again.

The five phases of the cardiac cycle are summarized in **Table 15-1**.

Pressure Waveforms

Because an understanding of the pressure changes in the aorta is vitally important to IABP therapy, the following discussion of pressure waveforms focuses on the left side of the heart and the aorta. Pressure changes in the left ventricle during the stages of the cardiac cycle are described as follows:

TABLE 15-1 Summary of the Cardiac Cycle

Phase	Events
Atrial systole	The sinoatrial node gives impulse. Atrium depolarizes. Atria contract, forcing blood into ventricles. Ventricular volume and pressure are increasing.
Isovolumetric contraction	Impulse travels through the atrioventricular node. Ventricles depolarize. Mitral and tricuspid valves close. Ventricular pressure rises abruptly.
Ventricular ejection (systole)	Pressure in the pulmonary artery equals pressure in the right ventricle. Pulmonary valve opens; right ventricle ejects. Pressure in the left ventricle exceeds pressure in the aortic root. Aortic valve opens; left ventricle ejects.
Isovolumetric relaxation	Ventricles relax and repolarize. Pressure in ventricles drops to less than arterial pressure. Aortic and pulmonary valves close owing to blood attempting to flow backward.
Ventricular filling (diastole)	Ventricular pressure drops below pressure in the atria. Mitral and tricuspid valves open. Blood flows through the atria and into the ventricles. Cycle begins again.

1. At the beginning of the cardiac cycle, aortic pressure is at its lowest (ie, diastolic) level. The elastic state and vascular resistance of the arteries determine this pressure. During diastole, the AV valves are open and blood flows passively from the atria into the ventricles. The pressures in the ventricles and atria are nearly equal and significantly lower than the pressure in the aorta.
2. The SA node depolarizes, causing the atria to contract. This contraction causes the pressure in the atria and ventricles to rise. As the impulse is conducted through the ventricles, the pressure rises sharply.
3. When the pressure in the left ventricle exceeds that of the left atrium, the mitral valve snaps closed.

The pressure continues to rise until the pressure in the aorta is exceeded, causing the aortic valve to open. As the aortic valve opens and blood enters the aorta, the pressure will rise to the peak systolic level and then begin to fall as ventricular ejection tapers.

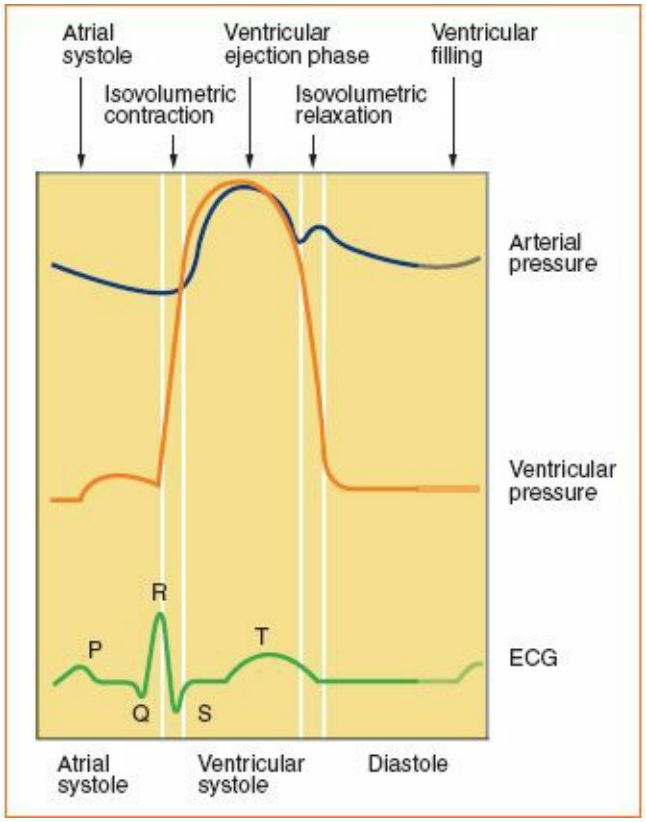


Figure 15-5 Arterial pressure waveform. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

4. The aortic pressure begins to decrease. As the pressure in the left ventricle drops, the pressure in the aorta becomes greater than that in the left ventricle. This change in pressure causes a slight backward flow of blood into the ventricle, and the backward flow causes the valve leaflets to be forced outward and into their closed position, much like that of a parachute opening.
5. As the aortic valve leaflets are forced outward into place, the pressure gradient across the valve increases significantly because the ventricle continues isovolumetric relaxation. The valves "bulge" slightly under the pressure of the blood from the aorta and then spring back slightly. This springing back causes a slight "bump" in the aortic pressure waveform, referred to as the **dicrotic notch**, signaling the onset of diastole. With the valve closed and the ventricles continuing to relax, the ventricular pressure decreases rapidly **Figure 15-5**.
6. When the pressure in the ventricle is less than the pressure in the atrium, the mitral valve opens and blood begins to flow passively into the ventricle from the atrium.

Myocardial Oxygen Supply and Demand

Myocardial oxygen supply must balance myocardial oxygen demand (**myocardial oxygen consumption [MVO_2]**) to meet myocardial metabolic requirements or ischemia will result. Factors that can affect myocardial oxygen supply include coronary artery anatomy, diastolic pressure, diastolic time, and oxygen extraction. Because the majority of blood flow to the coronary arteries occurs during diastole when the ventricle is at rest, diastolic pressure and time spent in diastole (diastolic time) profoundly affect the blood flow to the coronary arteries and, thus, myocardial oxygen supply. Because the time required to

complete systole is fixed, any increases in heart rate (HR) must result from decreased diastolic time and, therefore, less time to perfuse the coronary arteries.

The MVO_2 varies with HR, preload, afterload, and contractility; any increase in these factors increases oxygen demand.

Heart failure is a cyclic phenomenon. Injury to the myocardium, as might occur with an acute myocardial infarction (MI), results in a series of physiologic and hemodynamic changes that can affect the balance between oxygen supply and demand. The injury (from whatever cause) decreases pumping efficiency, cardiac output (CO), and arterial pressure. CO is composed of stroke volume (SV) × HR (CO = SV × HR). In this equation, for CO to remain the same, a decrease in SV or HR must be compensated by an increase in the other. Therefore, if the ability of the heart to pump effectively were impaired (such as might occur with an MI), causing a drop in the SV, the HR must increase for CO to remain the same. As discussed previously, coronary perfusion occurs only during diastole, and any increase in HR results in less diastolic time and, thus, less time for coronary perfusion. Because the HR is increased, the MVO_2 also increases, worsening the mismatch of oxygen supply and demand. The decreased coronary perfusion, coupled with the increased MVO_2, leads to worsened ischemia. Ischemia causes the heart to pump less effectively, leading to a greater decrease in SV. The HR increases to maintain CO, and the cycle of heart failure continues **Figure 15-6**.

The treatment of cardiac failure is aimed at restoring normal cardiac function and the balance between myocardial oxygen supply and oxygen demand. If initial intervention fails to restore the balance, the IABP offers a mechanical means of rebalancing them.

The Intra-aortic Balloon Pump

The IABP is a device that helps increase the blood flow to the heart muscle, thereby decreasing the heart's workload. It contains a small balloon ranging from about 25 to 50 mL of gas capacity for adults (sizes vary by manufacturer). The proper size of IABP catheter is determined according to patient height as indicated in the manufacturer's instructions. Typically, the IABP catheter is threaded into the descending aorta through the femoral artery. Insertion can be accomplished through a sheath (a large-diameter catheter placed into a vessel to allow smaller catheters or devices to be inserted through the catheter lumen into the vessel) or by using a catheter designed for sheathless insertion. When access to the femoral artery has already been obtained using a sheath (such as in a cardiac catheterization laboratory), a practitioner inserting an IABP catheter may decide to insert the device through the existing sheath rather than making a separate puncture. When IABP placement is elective or planned or when significant concerns about leg ischemia exist, sheathless insertion offers the advantage of an overall smaller diameter device in the access vessel. Sheathless insertion, then, may reduce complications of limb ischemia by reducing the size of the obstruction in the femoral artery. As mentioned, IABP therapy is also called counterpulsation because the balloon deflates during systole and inflates during diastole. An ECG machine determines when the balloon should be inflated and deflated.

Mechanics of IABP Function

Balloon Structure and Position

The IAB catheter consists of a long, narrow balloon mounted on a thin catheter. Typical catheters have two lumens: a central lumen and a gas lumen. The **central lumen** is used to guide catheter insertion into the femoral artery and through the arterial system, positioning the IAB tip in the descending thoracic aorta just below the origin of the left subclavian artery **Figure 15-7**. Alternative placement strategies for IAB

catheters include access through the axillary artery and, occasionally, transthoracic placement, which is typically reserved for critically ill patients unable to be weaned from cardiopulmonary bypass following cardiac surgery in whom femoral access is not possible or is contraindicated. Once the catheter is in place, the central lumen also allows monitoring of aortic blood pressure through a setup much like a standard arterial line. The **gas lumen** carries helium gas from the pump console to the IAB to control balloon inflation. Evolving technologies also incorporate a fiberoptic catheter lumen used to measure aortic pressure and (in some models) aortic oximetry.

Counterpulsation Sequence

Following placement of the catheter, it is connected to the IABP console. The IABP console pump inflates and deflates the balloon with helium in conjunction with the mechanical cardiac cycle. Helium is used because of its low molecular weight, which allows it to be pumped rapidly without much turbulence from the flow of the gas. Helium also adds safety because it is less likely to cause an air embolus if the balloon ruptures. Correct IABP timing inflates the balloon during diastole when the left ventricle is relaxed and the coronary arteries are filling with oxygenated blood **Figure 15-8**.

Physiologic Effects of IABP Therapy

For patients in cardiogenic shock, IABP therapy lowers systolic blood pressure as the balloon deflates at the onset of systole and raises diastolic pressure when the balloon inflates during diastole. Mean arterial pressure (MAP) will also rise as a result of the **diastolic augmentation**. Diastolic augmentation may be best thought of as a "second systole" because it occurs *during* diastole, but the rapid inflation of the balloon augments the diastolic pressure and effectively creates a second pressure wave to perfuse the heart and tissues. This sudden increase in pressure improves the flow to the coronary arteries and systemic circulation.

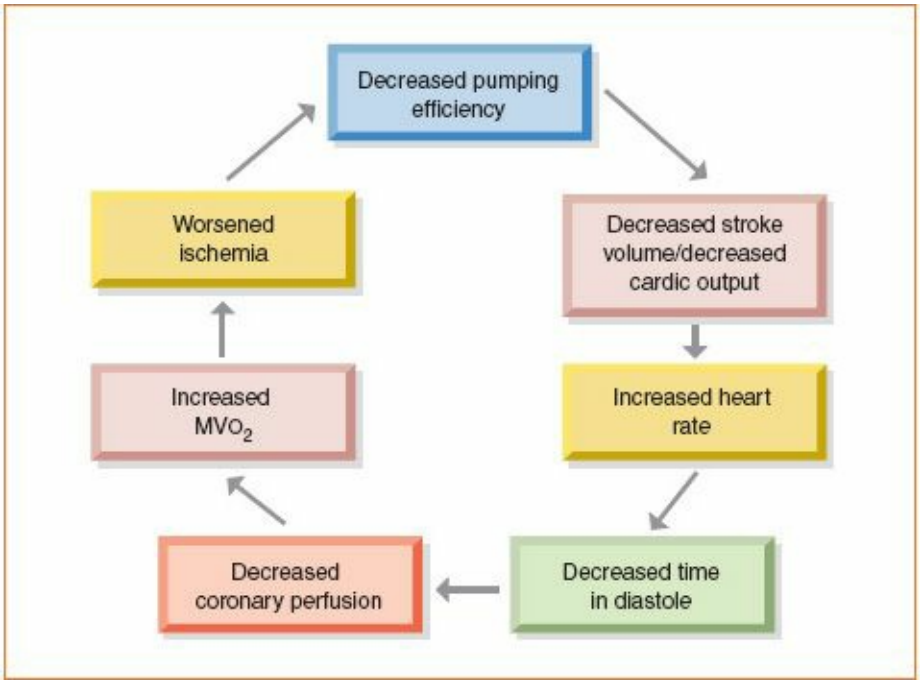


Figure 15-6 The cycle of heart failure.

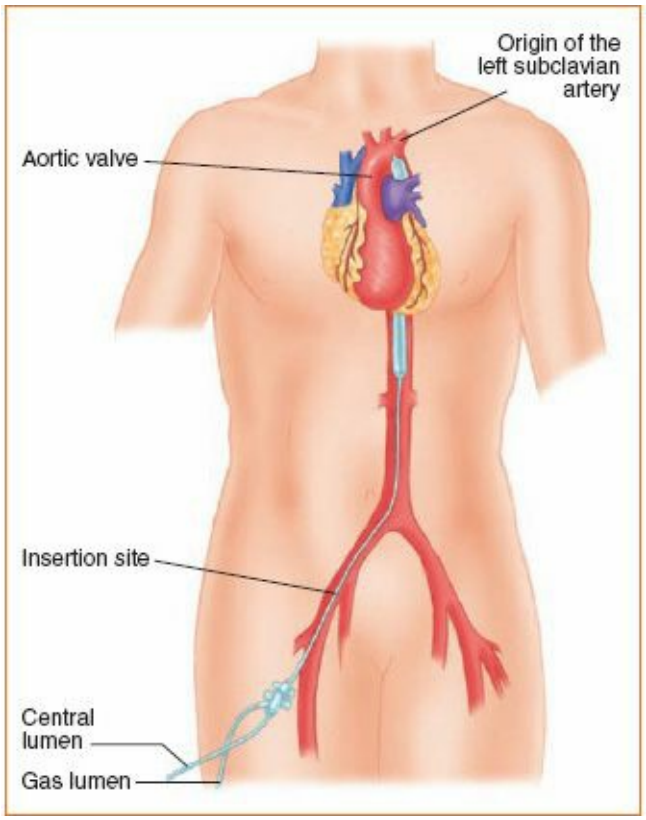


Figure 15-7 Intra-aortic balloon placement. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

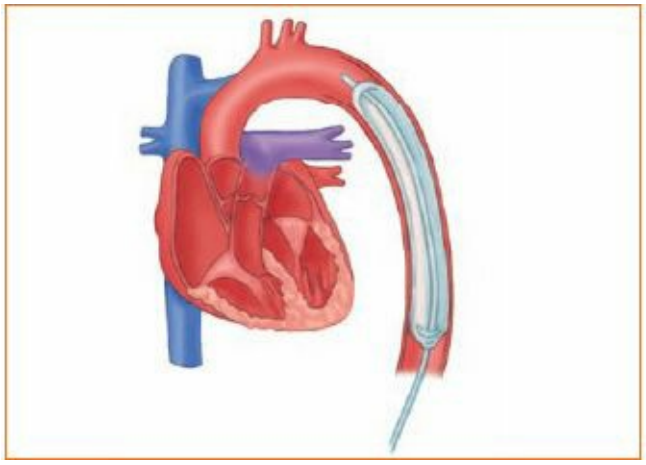


Figure 15-8 Intra-aortic balloon inflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

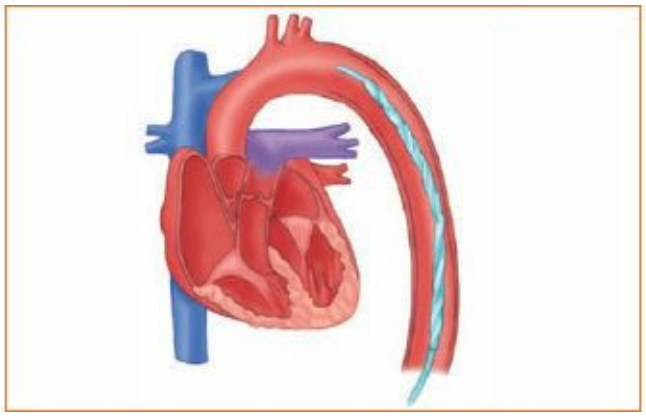


Figure 15-9 Intra-aortic balloon deflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

The balloon deflates at the end of diastole, just before the next ventricular ejection **Figure 15-9**. The sudden deflation of the balloon causes a drop in aortic pressure because the space that was occupied by the balloon is suddenly gone. This dramatic drop in pressure reduces the resistance against which the ventricle has to work to open the aortic valve and eject blood. To review from previous discussion, the ejection from the left ventricle begins when the pressure in the left ventricle is greater than that in the aorta. The sudden deflation of the IAB results in a lower pressure that must be overcome for the valve to open and the ejection of blood to begin. Because the majority of the myocardial oxygen demand is expended in overcoming this pressure, the result is a dramatic decrease in MVO_2. In addition, because less pressure must be overcome, the SV is also maximized. Because CO = SV × HR, an increase in SV allows the HR to decrease while still maintaining CO. A decrease in HR leads to increased diastolic time and, therefore, longer coronary perfusion time. The improvement in CO, with a lower MVO_2, effectively interrupts the cycle of heart failure and allows the heart to "rest" and recover from the insult that started the cycle. The increase in CO results in increased renal and peripheral perfusion and, therefore, may also improve metabolic derangements that may have developed as a result of the shock state during the period of decreased CO.

Balloon Volume or Augmentation

Each IABP console allows the operator to control the volume of gas shuttled back and forth to the balloon with each counterpulsation cycle. This control is variously referred to as augmentation or balloon volume. Typically, when IABP therapy is initiated immediately after the IABP catheter is inserted, software in the console begins shuttling gas at the lowest volume possible, gradually increasing the volume during several inflation-deflation cycles to allow the balloon to unfurl from the compressed condition in which it is packaged. There is little reason during routine use or during transport that the balloon volume or augmentation should be adjusted at less than maximal or full volume. This control is used with nonstandard balloon catheters as might be inserted in pediatric patients or, in some settings, to wean the patient from IABP therapy before removing the balloon. Given that a fully inflated balloon rarely occupies more than half the total diameter of the average aorta, weaning is more efficiently accomplished by reducing the assist ratio or frequency (discussed later in this chapter) rather than the inflation volume.

Waveform Changes and Triggers

As mentioned, proper IABP timing is essential to providing the full benefit of IABP therapy. The console constantly monitors the arterial pressure waveform, the patient's ECG, and the balloon gas pressure waveform during pumping to evaluate the timing and effectiveness of the therapy. The arterial pressure waveform that the IABP console monitors may come from the central lumen of the IAB catheter, from the fiberoptic catheter, or from a peripheral arterial catheter **Figure 15-10**. IAB inflation and deflation can be set to trigger from the ECG, from pressure changes, from a pacemaker, from an internally preprogrammed rate, or from combinations of these. The most reliable is the ECG. Triggering the IABP from the ECG provides the most optimal timing for inflation and deflation while still permitting the IAB to immediately deflate if the console senses an increase in pressure indicating ventricular ejection (such as with a premature ventricular or atrial contraction), a paced beat, or another signal that systole has begun. Most modern IABP console computers are able to track irregular arrhythmias and tachyarrhythmias (such as atrial fibrillation) and adjust timing as needed to protect the patient. The provider caring for the patient chooses the trigger best suited to the patient's condition.

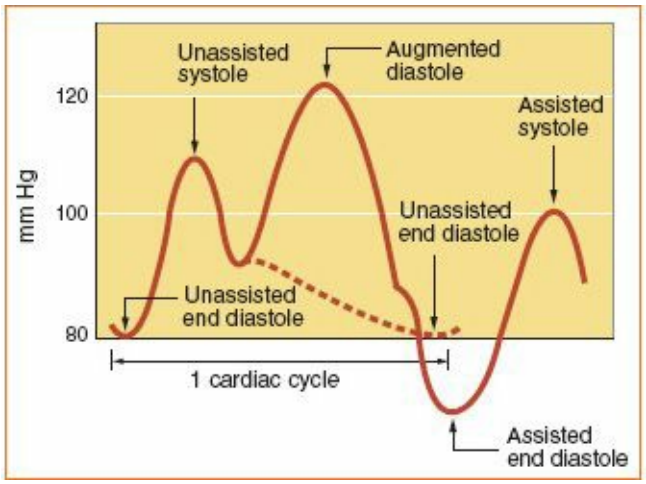


Figure 15-10 Arterial pressure waveform during intra-aortic balloon pump therapy. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

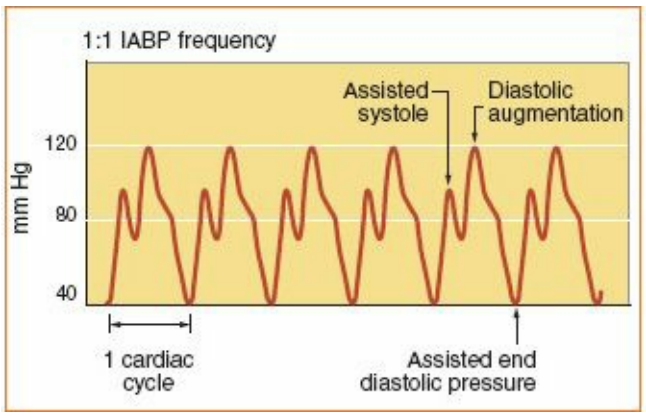


Figure 15-11 Diastolic augmentation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

Proper IABP Timing

The timing of IABP therapy can be selected from a variety of triggers. Early IABP machines had only manual timing, requiring great skill on the part of the operator to adjust the pump for proper timing with every change in the HR of more than 10 beats/min. The newer generation IABP consoles have sophisticated software capable of adjusting balloon inflation and deflation through a variety of changes and arrhythmias. However, computers are not infallible, and the IABP operator must constantly monitor the IABP to ensure correct operation and timing. Incorrect timing can be detrimental to the patient, not only because it does not maximize the therapy, but also because errors in timing, such as late deflation of the balloon, can worsen heart failure. Likewise, early inflation can damage the aortic valve.

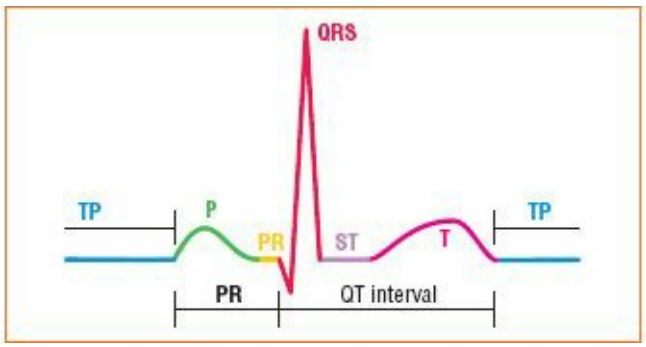


Figure 15-12 The components of an electrocardiographic strip.

Trigger Selection

Trigger signals available to initiate balloon inflation and deflation vary by manufacturer, although all IABP consoles usually default to using the QRS complex from the ECG to locate each cardiac cycle **Figure 15-12**. Early model IABP consoles triggered inflation from the midpoint of the T wave and deflation from the R wave on the ECG. The operator then manually adjusted the inflation and deflation to optimize timing. This so-called conventional timing worked fairly well as long as the R-R intervals remained consistent. Changes in HR, arrhythmias, or ectopic beats, or any other change in diastolic time posed problems for older IABP consoles and required manual correction of timing. Present-generation IABP consoles have sophisticated computer software capable not only of recognizing changes in HR, balloon pressures, patient blood pressures, tubing dead space, and console pneumatic rate of response, but also instantly making changes in timing to inflate and maintain balloon inflation throughout diastole. Modern-day or "real" timing using sophisticated computer software offers greater afterload reduction and improved MAP by extending the inflation period into the early isovolumetric contraction period.

The safest and, usually, the default trigger on most IABP consoles is the ECG, which allows the pump to trigger from the R wave on the patient's ECG for deflation. Pacer spikes are ignored in most consoles when using the ECG as the trigger source. Some manufacturers offer more than one ECG trigger mode, depending on HR and morphologic features of the ECG complexes. During ECG triggering, deflation will also occur if the IABP console senses an increase in systolic blood pressure indicative of the onset of systole, regardless of whether an R wave appears on the ECG.

Pressure can also be selected as a trigger source. The systolic upstroke of the patient's arterial pressure waveform is used as the trigger source. Using pressure is not as desirable as using the ECG as the trigger because it delays deflation until systolic ejection has already begun, reducing the beneficial effects of deflation simultaneous with ventricular ejection. With pressure selected as the trigger, most IABP consoles ignore the ECG signal. Pressure is the preferred trigger mode during CPR because it works synchronously with compressions to improve diastolic perfusion. This mode can be useful when the ECG signal is unavailable, excessive artifact exists, or ECG voltage is too low to produce a recognizable R wave. Excessive movement of ECG wires, very active patients, and procedures such as surgery that involve considerable staff contact with ECG lead areas may make pressure a desirable trigger mode.

A pacemaker trigger is also available on most IABP consoles and is usually further divided into atrial- and ventricular-paced rhythms. The only time that pacemaker triggers are useful is when the patient rhythm is 100% paced and the QRS complex has such poor morphologic features that the IABP console cannot use it in the ECG trigger mode. For patients with an AV-paced rhythm, the ventricular-paced trigger should be selected if pacer triggering is required. Most IABP consoles also offer an internal trigger mode that works completely asynchronously with the patient's ECG and arterial pressure. The internal trigger mode should never be used in any patient with cardiac output, including patients undergoing CPR. Situations when internal triggering may be used are generally limited to cardiac surgical procedures with no available ECG and external circulatory support. Internal triggering can prevent thrombus formation on the IABP catheter in such cases.

Evaluation of an IABP Waveform

The IABP device has the option to pump in several modes called assist ratios or IABP frequency. These modes are as follows:

- 1:1 assist ratio, in which the pump inflates with each heartbeat
- 1:2 assist ratio, in which the pump inflates with every other heartbeat
- 1:3 assist ratio, in which the pump inflates with every third heartbeat

- 1:4 assist ratio, in which the pump inflates with every fourth heartbeat
- 1:8 assist ratio, in which the pump inflates with every eighth heartbeat

CCTPs should assess several parameters of the waveform. The evaluation of the IABP waveform is most easily accomplished by placing the IABP into the 1:2 **assist ratio**. This ratio allows comparison of pressure waves of the unassisted cycle with the assisted one. In this mode, every other contraction is augmented by the IABP. In a strip of 1:2 augmentation waveforms, the peaks created by the inflation of the IAB are easily identified because they should be the highest pressure peaks **Figure 15-13**. Because the IAB inflates during diastole, these pressure waveforms are referred to as the **diastolic augmentation waveforms**. The pressure waveform that follows the diastolic augmentation peak (IAB inflation) is referred to as the **assisted systole** waveform because the deflation of the IAB assists the ventricle in ejecting blood, thereby reducing the ventricular **afterload** (the resistance that must be overcome to eject the blood from the ventricles). The third pressure waveform is referred to as **unassisted systole** because the balloon pump is not moving and the waveform reflects what would normally occur without IABP intervention. To evaluate for correct IABP timing, the pressure peaks, slopes, and inflation and deflation points of each of these pressure waveforms must be evaluated in a systematic manner. The parameters of the IABP waveform that should be evaluated are as follows:

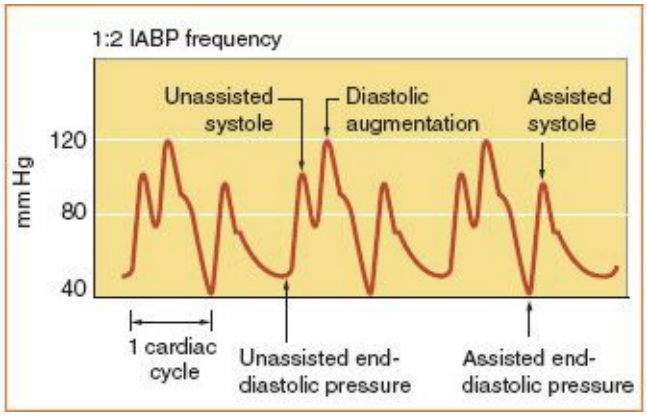


Figure 15-13 Pressure waveform augmented 1:2. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

1. Systole and diastole of each pressure waveform. Although it may seem confusing at first, it is critical to identify systole and diastole in the pressure waveforms. This balloon inflation gives an appearance of systole as the pressure peaks are high; however, it is important to remember that the heart is in diastole. It may help to think of balloon inflation as a second systole that occurs in diastole.
2. Diastolic augmentation, assisted systole, and unassisted systole. When looking at a 1:2 augmentation strip, this can be done easily by identifying the balloon inflation. This pressure waveform will be the diastolic augmentation. Immediately before this waveform is the unassisted systole. The pressure waveform that occurs immediately after the diastolic augmentation is the assisted systole.
3. The point of inflation. Inflation should occur at the dicrotic notch, as this signals the closure of the aortic valve **Figure 15-14A**. To find the dicrotic notch, the IAB inflation point is manually moved to a later time in the cardiac cycle. Once the dicrotic notch is located, the console inflation should be adjusted to an earlier time until the notch created by the inflation of the balloon on the dicrotic notch forms a crisp "V" **Figure 15-14B**. The balloon inflation point should be reflected on the pressure waveform as a sudden increase in pressure during diastole. A crisp V with no evidence of the dicrotic notch reflects inflation of the balloon immediately at the onset of diastole. Note: If the

dicrotic notch is visible, inflation is occurring too late. If inflation occurs too early, the V will open slightly as the slope of the diastolic augmentation decreases slightly (less steep slope).

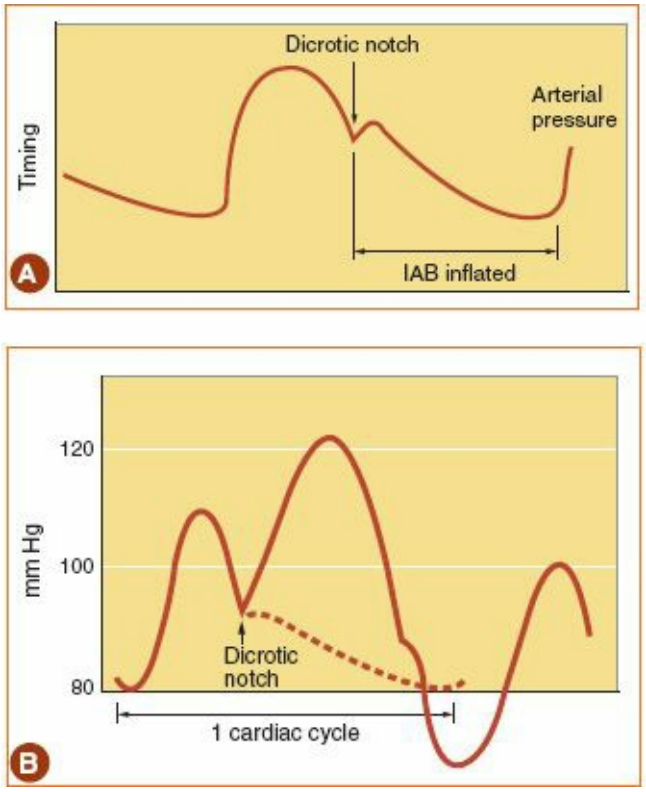


Figure 15-14 Pressure waveform showing the point of inflation. **A.** Dicrotic notch. **B.** A crisp V after adjusting the console for inflation to occur earlier. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

4. End-diastolic dip in pressure created by balloon deflation. The end-diastolic dip should dip below the baseline end-diastolic pressure occurring with unassisted systole. This dip should be followed by a straight-line slope to the next systolic peak. If a diastolic dip is created, followed by a plateau and then the beginning of the systolic peak, deflation is occurring too early. If there is no end-diastolic dip, but rather an end-diastolic pressure that is above the baseline of the unassisted systole and a straight slope up to the next systolic peak, deflation is occurring late. Ideally, CCTPs should optimize deflation by adjusting it to an earlier or a later time to obtain the lowest possible assisted end-diastolic pressure while not reducing diastolic augmentation. It is helpful to be familiar with the expected waveforms, but deflation is best optimized to achieve the most favorable pressures indicative of reduction in after-load (assisted end-diastolic pressure) without impinging on ventricular ejection (reduced diastolic augmentation).

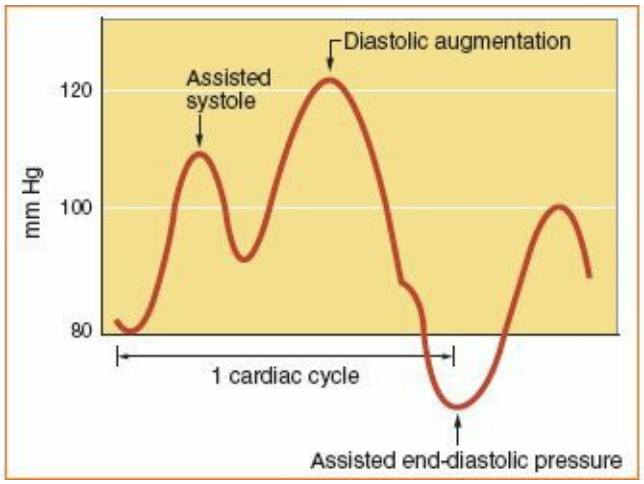


Figure 15-15 Pressure waveform with slopes of diastolic augmentation and assisted systole marked. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

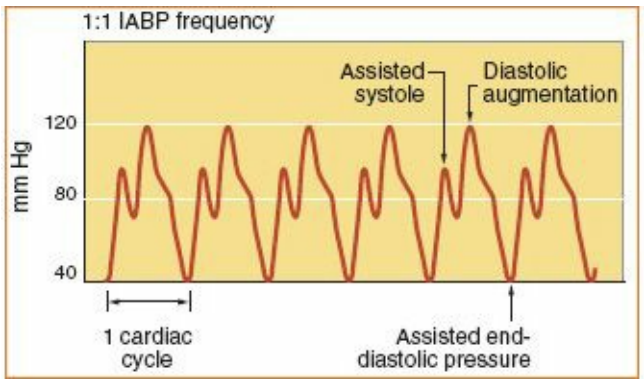


Figure 15-16 Pressure waveform showing peak pressures of assisted systole and unassisted systole. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

5. Slope of diastolic augmentation pressure and slope of assisted systole **Figure 15-15**. The slopes of the diastolic augmentation peak (balloon inflation) and the slope from the end-diastolic dip to the assisted systolic peak should be straight lines and nearly parallel.
6. Diastolic augmentation peak pressure. If effective IABP therapy is being achieved, the diastolic augmentation peak pressure should be higher than (or at least equal to) the systolic pressures.
7. Comparison of the peak pressure of assisted systole with the peak pressure of unassisted systole **Figure 15-16**. If effective IABP therapy is being accomplished, the peak pressure of assisted systole should be lower than the systolic peak of unassisted systole, which reflects that the IABP is reducing the peak pressures and, thus, decreasing the workload of the heart and reducing MVO_2.

TABLE 15-2 IABP Timing Errors

Error	Definition	Waveform Characteristics	Physiologic Effects
Early inflation	Inflation of the IAB before aortic valve closure	IAB inflation before the dicrotic notch; diastolic augmentation encroaches onto systole (may be unable to distinguish the two)	Possible premature aortic valve closure; possible increase in LV, EDV, and EDP, or in pulmonary capillary wedge pressure; increased LV wall stress (afterload); aortic regurgitation; increased MVo_2
Late inflation	IAB inflation markedly after aortic valve closure	IAB inflation after the dicrotic notch; absence of sharp V	Suboptimal coronary artery perfusion
Early deflation	Premature IAB deflation, during the diastolic phase	Sharp drop following diastolic augmentation Suboptimal diastolic augmentation Assisted aortic EDP may be equal to or less than unassisted aortic EDP Assisted systolic pressure may rise	Suboptimal coronary artery perfusion; potential for retrograde coronary and carotid blood flow; suboptimal afterload reduction; increased MVo_2
Late deflation	Balloon deflation late in the cardiac cycle	Assisted aortic EDP may be equal to unassisted aortic EDP; prolonged rate of rise of assisted systole; diastolic augmentation may appear widened	Afterload reduction essentially absent; increased MVo_2 because of prolonged isovolumetric contraction and greater resistance to LV ejection; increased afterload if IAB impinges on LV ejection

Abbreviations: EDP, end-diastolic pressure; EDV, end-diastolic volume; IAB, intra-aortic balloon; LV, left ventricular; MVO_2, myocardial oxygen consumption.

Timing Errors

Timing during IABP therapy is very important. **Table 15-2** summarizes timing errors, waveform characteristics, and physiologic effects, all of which are discussed in the next section. These errors result from incorrect timing by the IABP operator.

Early Inflation

The IAB should inflate at the dicrotic notch, which signals the closure of the aortic valves. Inflation that occurs too early is reflected on the waveform by a shortened normal pressure decrease that follows diastole. Because the aortic valves have not closed, this error in timing can be detrimental to the patient by decreasing pumping efficacy. On the pressure waveform, this is reflected as an "opened V" and with a lessened slope on the diastolic augmentation waveform. The pathophysiologic effect of early inflation is that the ejection of blood from the left ventricle is impaired by the sudden increase in aortic pressure while the aortic valve is open, forcing premature closure of the aortic valves and decreasing the SV **Figure 15-17**. It is also possible for early inflation to result in damage to the aortic valve.

Late Inflation

If the dicrotic notch is visible on the pressure waveform, inflation of the IAB is late. The late inflation of the IAB, although causing little harm, does not maximize the benefits of IABP therapy because the pressure in the aorta has decreased before the IAB inflates, and, therefore, the profound increase in diastolic pressure that maximizes coronary and systemic circulation is not realized **Figure 15-18**.

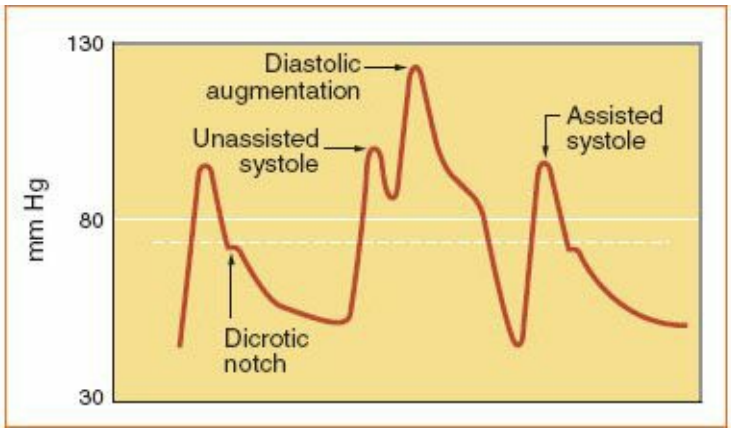


Figure 15-17 Early inflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

Early Deflation

The timing for deflation of the IAB should be the R wave. Using this trigger for deflation is effective for preventing early deflation. This trigger allows for sudden deflation of the IAB, resulting in the end-diastolic dip that decreases the workload of the ventricles to overcome the aortic pressure and eject blood from the ventricles. If deflation occurs too early, the pressure in the aorta will equalize and rise slightly to plateau before systole, meaning that a higher end-diastolic pressure must be overcome for the ventricles to eject blood. The waveform will exhibit a sharp drop from diastolic augmentation with a characteristic prolongation of the assisted aortic end-diastolic period. This lack of inflation through to the end of diastole means not only that the benefits of IABP therapy are not realized by assisting the next systole, but also that the duration of increased pressure during diastole (which benefits the coronary arteries and system circulation) is also decreased **Figure 15-19**.

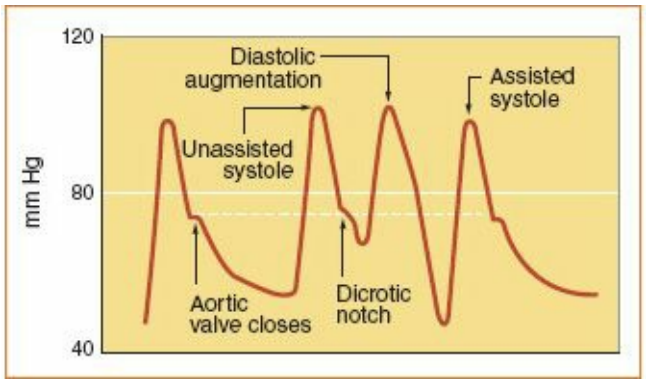


Figure 15-18 Late inflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

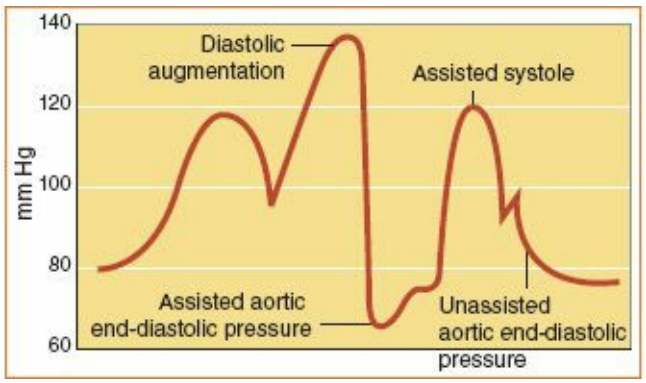


Figure 15-19 Early deflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

Late Deflation

As stated previously, the use of the R wave as the trigger is the most reliable to prevent errors in timing of IAB deflation. However, late IAB deflation can be extremely detrimental to the patient. Late deflation is characterized by a widened appearance of the diastolic augmentation waveform base and a prolonged rate of rise in the assisted systolic waveform **Figure 15-20**. Because the balloon is still inflated while the heart is trying to eject blood from the left ventricle, the pressure in the aorta never drops to the baseline or dips below it. This increased pressure at which the aortic valves can open and eject blood from the ventricles increases the afterload and significantly increases the MVO_2 of the left ventricle because the majority of MVO_2 is used to overcome the pressure in the aorta to eject the blood. Thus, late deflation of the balloon *worsens* myocardial ischemia and needs to be avoided.

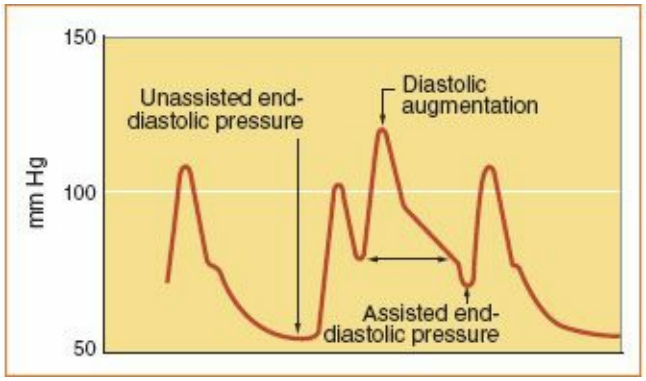


Figure 15-20 Late deflation. Illustration from source material by Datascope Corp. and Maquet Cardiovascular. Used with permission.

IAB Insertion

Indications

IABP therapy can be used effectively to treat a variety of conditions resulting in heart failure. Conditions or situations that benefit from IABP therapy are listed in **Table 15-3**. IABP therapy offers valuable benefits by reducing cardiac work and MVO_2 and improving peripheral perfusion in patients with a variety of conditions and undergoing cardiovascular and surgical procedures.

Indications for IABP therapy include the following:

- Refractory ventricular failure
- Cardiogenic shock
- Unstable refractory angina
- Impending infarction
- Mechanical complications due to acute MI (ie, ventricular-septal defect [a hole in the wall between the ventricles], mitral regurgitation, or papillary muscle rupture)
- Ischemia-related intractable ventricular arrhythmias
- Cardiac support during high-risk general surgical or coronary angiography or angioplasty procedures
- Septic shock
- Weaning from cardiopulmonary bypass

- Intraoperative pulsatile flow generation
- Support for failed angioplasty and valvuloplasty

TABLE 15-3 Conditions or Situations That Benefit from IABP Therapy
Chest pain from unstable angina
Severe arrhythmias
Cardiogenic shock
Septic shock
Heart failure
Myocardial ischemia from unstable angina
MI
Ventricular irritability
Ventricular-septal defect (before surgical intervention)
Papillary muscle dysfunction (before surgical intervention)
Papillary muscle rupture (before surgical intervention)
Impaired cardiac function (before surgical intervention)
Support during diagnostic and interventional procedures (ie, coronary angiography and angioplasty)
Weaning from cardiopulmonary bypass
Failed angioplasty (until patient undergoes surgical intervention)
Unsuccessful valvuloplasty (until patient undergoes surgical intervention)
During cardiopulmonary bypass
Cardiac support during noncardiac surgery
Prophylactic support while awaiting cardiac surgery
Myocardial contusion
Mechanical bridge to other assist devices or heart transplantation
Abbreviations: IABP, intra-aortic balloon pump; MI, myocardial infarction.

In myocardial ischemia and chest pain associated with unstable angina, impending infarction, and intractable ventricular arrhythmias related to ischemia, the IAB can be beneficial in maintaining adequate coronary artery perfusion, relieving myocardial ischemia, and decreasing myocardial oxygen demand. If cardiac catheterization and further interventions are necessary, the patient can undergo these procedures in a more hemodynamically stable condition. The IAB can also be used in conjunction with cardiopulmonary bypass pumps to provide pulsatile flow. For patients in septic shock, myocardial

function may be impaired, and the IABP may be used to increase CO to meet the increased metabolic demands.

Patients who experience severe chest pain accompanied by ECG changes or arrhythmias and who do not obtain relief from drug therapy are at great risk of developing an MI. Improving coronary blood flow and reducing LV work will minimize chest pain and ECG changes associated with the MI. IABP therapy is often considered when symptoms persist despite maximal medical therapy. If cardiac catheterization and further interventions are indicated, the IABP improves the patient's hemodynamic status before the procedures.

Depending on the area of an acute MI, mechanical complications can occur. Although these complications involve a small percentage of MIs, the resulting hemodynamic compromise can have lethal consequences, especially if not treated immediately. Ventricular-septal defects, papillary muscle dysfunction, and papillary muscle rupture usually require surgical intervention, often on an emergency basis. If the patient undergoes cardiac catheterization or surgical intervention in a hemodynamically compromised state, the potential for mortality and morbidity increases significantly. The IABP provides temporary support to achieve hemodynamic stability before undertaking definitive measures.

Ventricular irritability, a frequent complication of acute MI, can lead to severe arrhythmias and further hemodynamic compromise. In most patients, conventional drug therapy and supportive measures are sufficient to reverse the irritability and arrhythmias. However, lack of response to conventional medical therapy results in high risk for further myocardial damage and death. IABP therapy has proven effective in stabilizing the hemodynamic condition of patients with ventricular irritability or arrhythmias by increasing coronary artery perfusion, reducing ischemia, and maintaining adequate peripheral perfusion.

In the already compromised heart, a decrease in arterial pressure can reduce myocardial oxygen supply and cause a loss of functional myocardial tissue. Prevention of worsening failure and cardiogenic shock requires prompt treatment of any signs of hemodynamic instability. Treatment aims at relieving LV workload and restoring the balance between myocardial oxygen supply and demand, allowing the myocardium time to heal and recover maximal function. IABP therapy assists in this effort by decreasing LV workload and increasing coronary artery perfusion.

LV failure following an acute MI may progress to cardiogenic shock. As with LV failure, the objectives of cardiogenic shock treatment are decreased cardiac work, increased myocardial oxygen supply, and lowered MVO_2. The combined effects of IABP therapy—increased oxygen supply, decreased afterload, and improved systemic perfusion—allow the heart to rest and help halt the subsequent vicious cycle that often occurs in acute MI.

Patients with impaired cardiac function are considered high-risk candidates for general surgery. Anesthetic agents and the procedure itself can place increased oxygen demands on the weakened heart. IABP therapy provides hemodynamic stability by helping balance myocardial oxygen supply and demand preoperatively, intraoperatively, and during the critical postoperative period when demands on the heart are particularly high.

The IABP works in conjunction with coronary angiography and angioplasty to support and stabilize the conditions of high-risk patients undergoing these procedures. Overall, IABP therapy can provide increased coronary artery perfusion and a reduction in cardiac work, thus lowering the risk of hemodynamic compromise due to reduced coronary flow during IAB inflation or acute coronary occlusion.

Weaning a patient from cardiopulmonary bypass may be difficult when support of the heart and lungs on the cardiopulmonary bypass machine is prolonged, surgical revascularization (placement of bypass grafts around blocked coronary arteries) is partially achieved, or preexisting myocardial dysfunction is present. Termination of cardiopulmonary bypass may result in hypotension and a low CO, despite

administration of inotropic and vasoactive drugs. IABP therapy in this setting decreases LV resistance, improves CO, and increases coronary artery and systemic perfusion pressures, facilitating the patient's removal from the cardiopulmonary bypass machine.

IABP therapy can support and stabilize the conditions of patients with severe LV failure resulting from failed angioplasty (attempts to open an occluded coronary artery in an interventional cardiac catheterization laboratory). Overall, the IABP can provide increased coronary artery perfusion and decreased cardiac work, thus reducing the risk of hemodynamic compromise because of reduced coronary flow or acute coronary occlusion. Unsuccessful valvuloplasty, however, a procedure in which a balloon-tipped catheter is used to dilate a stenotic heart valve, may produce cardiac dysfunction. The IABP helps support cardiac function until the patient can undergo definitive valve repair or replacement.

Septic shock is the consequence of overwhelming infection that affects all organ systems and dramatically increases metabolic demands. Characterized by low blood pressure, impaired neurologic function, decreased CO, and fever, it can lead to cardiogenic shock. The increased metabolic demands of a patient in shock lead to a mismatch between oxygen supply and demand in the tissues. This deficiency causes cells to carry on anaerobic respiration, creating lactic acid. As the lactic acid builds up in the body, it impairs the contractility of the heart and leads to impaired heart function. As discussed previously, this begins a vicious cycle of heart failure leading to decreased perfusion that worsens heart failure. For patients with an inadequate response to other supportive measures, IABP therapy can increase coronary blood flow, reduce LV workload by lowering systolic pressure, and improve tissue perfusion by maintaining adequate MAP.

In short, IABP therapy can be used for a variety of indications. **Table 15-4** summarizes the potential benefits of IABP therapy. Whatever the indication for use, the IABP fulfills basic roles: reducing the workload of the myocardium, improving coronary and systemic flow, and restoring the balance between the metabolic demands of the heart and body and the supply of oxygen available.

TABLE 15-4 Benefits of Intra-aortic Balloon Pump Therapy

Improves coronary artery perfusion
Increases cardiac output
Maintains peripheral perfusion
Reduces myocardial ischemia
Decreases myocardial oxygen demand
Improves hemodynamic stability
Decreases afterload

Contraindications

Although CCTPs do not insert the IAB, they may arrive before or during consideration of IAB insertion. It is useful to understand the contraindications to IABP therapy, which include the following:

- Severe aortic valvular insufficiency
- Abdominal or aortic aneurysm
- Severe calcific aortic or iliac arterial disease or severe peripheral vascular disease

These contraindications are discussed individually in the following sections.

Severe Aortic Insufficiency

IABP therapy is contraindicated in patients with severe aortic valve insufficiency. For the benefits of IABP therapy to be realized, the aortic valve must be competent. Balloon inflation in the setting of aortic valve insufficiency would force blood from the aorta across the valve into the ventricle. This **aortic regurgitation**, in turn, would increase cardiac work by overloading the ventricle with additional blood volume. In essence, the ventricle would have to pump the same blood back and forth, paradoxically increasing the workload. An incompetent aortic valve is also more likely to sustain damage from an improperly inserted IAB or poorly timed IAB inflation and deflation.

Aortic Aneurysm or Dissection

Use of the IABP is contraindicated if the patient has an abdominal or thoracic aortic aneurysm or aortic dissection. During insertion, the IAB catheter could become misdirected into the aneurysm itself or tear the weakened wall of the aorta. The increased pressure generated with counterpulsation could worsen the aneurysm or dissection.

Aortoiliac or Severe Peripheral Vascular Disease

Severe, calcific arterial or peripheral vascular disease is also a contraindication to IABP therapy, although some view it as a **relative contraindication**. The physician must decide whether the benefits of IABP therapy outweigh the risk of further compromised arterial blood flow. Peripheral vascular disease may limit the ability to advance the catheter through the atherosclerotic vessel. In addition, the presence of the catheter could cause plaque rupture or vessel occlusion, resulting in leg ischemia. This information is important to the IABP operator (usually a CCTP) because blood flow to the extremity distal to insertion must be monitored for evidence of circulation compromise.

Insertion Site Factors

Using a catheter without a sheath, or sheathless insertion, is not recommended if the patient has any of the following:

- A large amount of fatty tissue at the insertion site over the common femoral artery because the excessive distance between the skin and the femoral artery is likely to trap the catheter in the subcutaneous tissue
- Extensive scarring or fibrosis at the insertion site
- Other contraindications to percutaneous insertion

Side Effects and Complications

Side effects and complications of IABP therapy include the following:

- Limb ischemia (compartment syndrome may develop after the IAB is removed)
- Excessive bleeding from the insertion site
- Thrombocytopenia (owing to platelet destruction)
- Immobility of the balloon catheter
- Balloon leak
- Infection
- Aortic dissection
- Thrombosis
- Altitude changes during air transport

Management of specific complications is discussed later in this chapter.

IABP Operation During Transport

As with pacemakers and implanted cardioverter defibrillators, operation of an IABP varies somewhat from model to model. The fundamentals apply to all IABPs, however, so the material studied to this point will be useful for training with a specific IABP device. If possible, CCTPs should study and practice with the device that will be used on transports to help familiarize themselves with the specifics of IABP operation and common problems and their solutions.

Most critical care transport programs require that a CCTP be accompanied by a certified perfusionist or critical care registered nurse knowledgeable about the IABP, so CCTPs are working with a colleague with additional training in this area on calls that involve a patient receiving IABP therapy.

Vehicle Requirements

Patients receiving IABP therapy are candidates for transport in ground ambulances or in fixed- or rotary-wing aircraft. Some issues to consider when using these vehicles include battery life and vehicle power supply, space and weight constraints, and loading, unloading, and securing the pump in the vehicle. Addressing these issues when developing an IABP transport program will resolve most potential problems before the first transport.

Power Supply

Transport vehicles often have an inverter capable of converting the DC voltage generated by the engine to AC voltage suitable for powering the IABP. In addition to AC power, some aircraft produce 24-V DC power. Many IABPs are capable of operating on 110- to 120-V of AC power or 24-V of DC power, although the latter may require an adapter cable. Most IABP systems also incorporate batteries for limited periods of portable operation **Figure 15-21**. Ensuring a match between IABP power requirements and the available power supply in the transport vehicle is a crucial planning step when establishing an IABP transport program. Careful attention to transport-related physical and logistic considerations—well in advance of actual transport—ensures the ability of CCTPs to focus on patient-specific needs during transport.

Figure 15-21 A portable intra-aortic balloon pump.

Space, Weight, and Equipment Attachment

It is also necessary to evaluate space and weight constraints of the transport vehicle before transport and

the logistics of loading and unloading the IABP and the patient. Ground ambulances generally have minimal restrictions on weight, and the system will usually fit easily into the vehicle. In a transport aircraft, space limitations may require reconfiguration of the pump and monitor module. The additional weight of the system may also be an issue in air operations, another important planning point during transport program development.

Once the IABP is in the transport vehicle, it is critical to secure the pump so that it remains stable for safe operation throughout transport. It may be necessary to work with the IABP manufacturer, Federal Aviation Administration representatives, and the aircraft mechanic to ensure that an adequate system is in place to secure the IABP against air turbulence.

Equipment

The following equipment should be gathered before working with an IABP during transport:

- IABP with transport module
- Appropriately sized balloon catheters with insertion kit Spare helium tank (200 psi) Operator's manual
- Stopcocks
- 60-mL syringe (Luer tip)
- Skin electrodes
- IABP extension tubing
- Doppler ultrasound device (to assess distal pulses)
- Extra ECG cables
- 2″ cloth tape to secure IABP ECG electrodes and catheter to patient
- Arterial pressure monitoring transducer and setup
- Pressure transducer cables
- Arterial flush solution (500 mL of normal saline with 1,000 U of heparin)
- Adapters to fit other brands of balloon catheters to the IABP console
- Knee splint Soft restraints

Steps for Using the IABP

Skill Drill 15-1 describes the steps performed when beginning a call involving a patient receiving IABP therapy. These steps are as follows:

1. When entering the facility to transport a patient receiving IABP therapy, visually assess the surroundings to ensure that hallways, elevators, and routes of travel will accommodate necessary equipment and personnel **Step 1**.
2. Obtain the patient report **Step 2**.
3. Ascertain current IABP settings and note typical pressures (console systolic, diastolic, augmentation, and mean arterial pressures) **Step 3**.
4. Determine IABP catheter model, size, and insertion depth (measurement at insertion site) **Step 4**.
5. Ascertain that the balloon tip location has been verified by chest radiograph **Step 5**.
6. Conduct an assessment **Step 6**. IABP focus includes inspection of the insertion site for active bleeding, peripheral pulses in both lower extremities (may require Doppler confirmation), and a

radial pulse in the left upper extremity (to ensure subclavian blood flow).

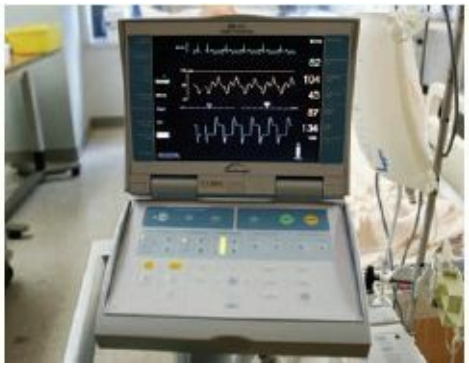
7. Attach new ECG leads and secure each lead over the electrode with 2″ cloth tape **Step 7**. This step will prevent lead disconnection and potential loss of trigger during transport.
8. Ensure that the IAB catheter is taped securely to the patient's leg **Step 8**.
9. Apply a knee immobilization splint to the leg in which the IAB was inserted to prevent leg flexion during transport **Step 9**.
10. Ensure that the appropriate connectors to attach the IABP to the transport console are available. Determine the console that will be used at the receiving facility, and be certain to take any necessary adapters or connectors (usually included in the IAB insertion kit).
11. Move the patient to the transport stretcher **Step 10**. Connect and secure all pumps, monitors, ventilators, and other equipment.
12. Transfer the IABP to the transport console at the bedside or in the transport vehicle (if the IABP console is mounted in the vehicle) **Step 11**.
13. Establish power to the transport IABP console **Step 12**.
14. Open the helium tank, and verify pressure **Step 13**.

Skill Drill 15-1

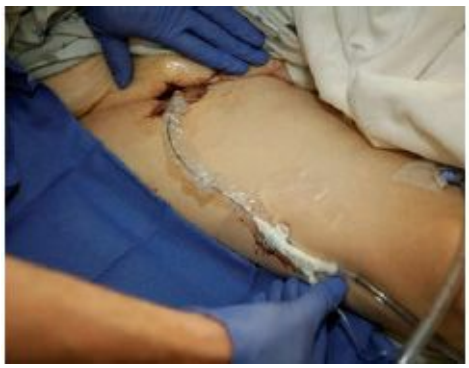
Operating the IABP During Transport

1 Visually assess the surroundings to ensure that hallways, elevators, and routes of travel will accommodate necessary equipment and personnel.

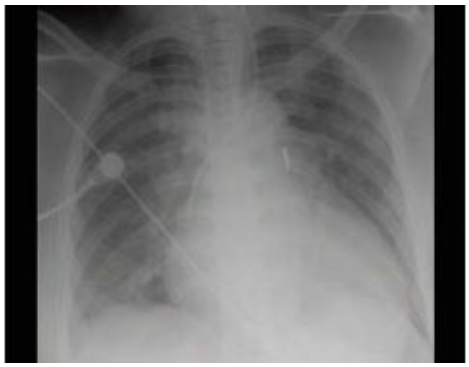
2 Obtain the patient report.



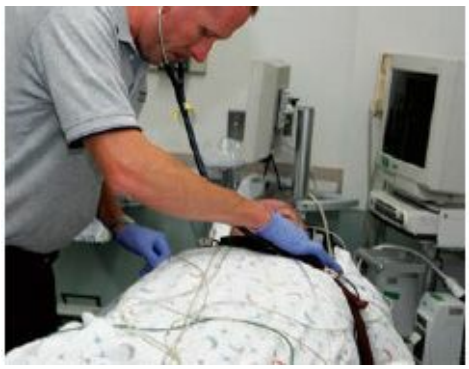
3 Ascertain current IABP settings and note typical pressures.



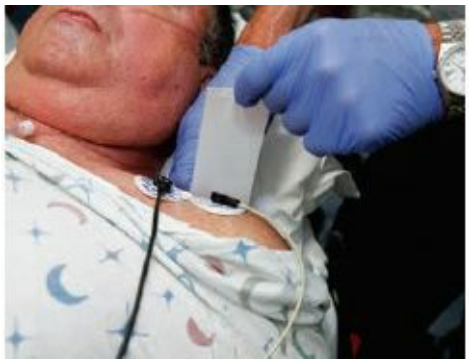
4 Determine IAB catheter model, size, and insertion depth.



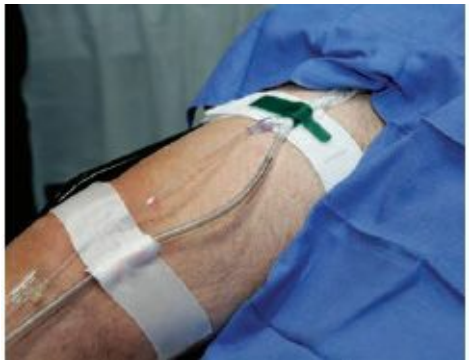
5 Ascertain that the balloon tip location has been verified by chest radiograph.



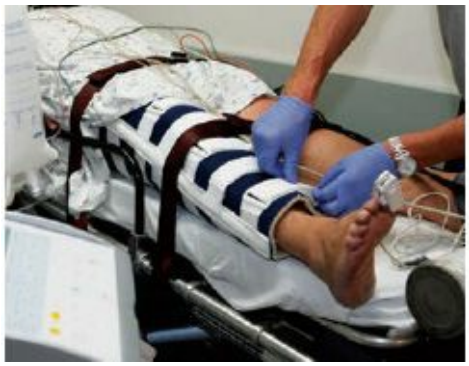
6 Conduct an assessment.



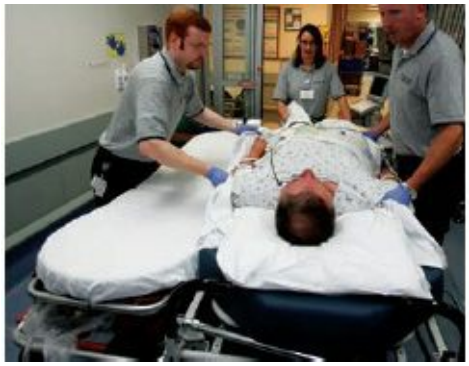
7 Attach new ECG leads and secure each lead over the electrode with 2″ cloth tape.



8 Ensure that the IAB catheter is taped securely to the patient's leg.

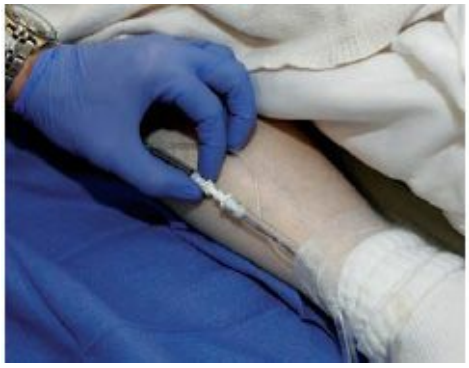


9 Apply a knee immobilization splint to the leg in which the IAB was inserted to prevent leg flexion during transport. Ensure that the appropriate connectors to attach the IABP to the transport console are available. Take any adapters that will be necessary at the receiving facility.



10 Move the patient to the transport stretcher. Connect and secure all pumps, monitors, ventilators, and other equipment.

11 Transfer the IABP to the transport console at the bedside or in the transport vehicle (if the IABP console is mounted in the vehicle).



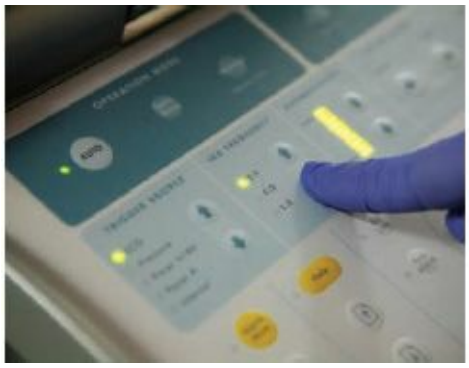
12 Establish power to the transport IABP console.

13 Open the helium tank and verify pressure.

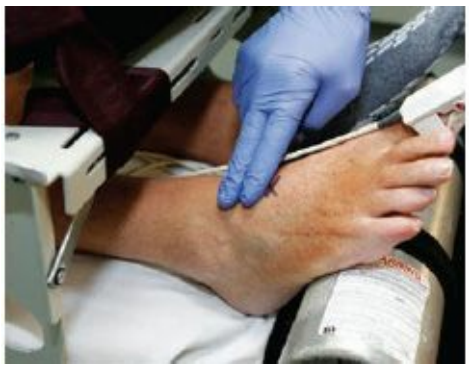
14 Follow the IABP console instructions for start-up (on console help screens or in manufacturer-provided user manual):

- Establish ECG and
  pressure waveforms from the patient.
- Confirm initial pump settings.

- Set timing.
- Initiate IAB pumping.
- Set console alarms.
- Confirm all pump settings.



15 Optimize IABP timing in a 1:2 mode:
- Set inflation by adjusting inflation to a later time until the dicrotic notch becomes visible, then move inflation to an earlier time until a crisp V pattern appears at the dicrotic notch.
- Set deflation to achieve the lowest possible diastolic pressure while maintaining maximal augmentation.



16 Assess pressures every 5 minutes or with any changes in the patient's condition during transport. Check the insertion site and pulses every 15 minutes.

15. Follow the IABP console instructions for start-up (on console help screens or in manufacturer-provided user manual) **Step 14**:
- Establish ECG and pressure waveforms from the patient.
- Confirm initial pump settings.
- Set timing.
- Initiate IAB pumping.
- Set console alarms.
- Confirm all pump settings.

16. Optimize IABP timing in the 1:2 mode **Step 15**:
- Set inflation by adjusting inflation to a later time until the dicrotic notch becomes visible, then move inflation to an earlier time until a crisp V pattern appears at the dicrotic notch.
- Set deflation to achieve the lowest possible diastolic pressure while maintaining maximal augmentation.

17. Assess pressures every 5 minutes or with any changes in the patient's condition during transport. Check the insertion site and pulses every 15 minutes **Step 16**.

Special Populations

Always immobilize the knee of the IABP leg prior to transport. This helps avert sudden leg movement, which may lead to bleeding or dislodgement of the IABP catheter. This is especially relevant to the geriatric patient whose tissues are less elastic and therefore more prone to damage from sudden movement.

Altitude Changes

Barometric pressure, the pressure exerted by gases in the ambient air, decreases with increasing altitude. During air transport, atmospheric pressure changes alter the gas volume in the IAB catheter and may affect IAB performance. As an aircraft ascends and atmospheric pressure decreases, the structural strength of the IAB restricts the rise in gas volume, but gas pressure in the IAB increases. Atmospheric pressure increases as the aircraft descends, reducing the gas volume in the IAB, which can decrease the effectiveness of IAB inflation.

Some IABPs compensate for atmospheric pressure changes by automatically initiating a refill cycle to adapt gas volume in the IAB to the external change. Initiation of one of these cycles typically begins when external pressure changes reach a preprogrammed threshold. Called autofill mode, this mode automatically purges and fills the IAB when local atmospheric pressure decreases or increases by 25 or 50 mm Hg, respectively. These pressure changes occur approximately every 1,000′ of ascent or 2,000′ of descent, respectively. The autofill mode should be used during air transport. If using an older model of IABP, its ability to compensate for altitude changes should be verified. If it is not capable of compensating for altitude changes, the IABP operator can manipulate the extent to which the balloon inflates to make use safer. The manual fill must be performed at the same intervals that an autofill would occur.

The potentially most catastrophic consequence that could result from IABP operation at an altitude would be a rapid ascent to altitude with the IAB inflating to full capacity. Theoretically, the gas will expand as the ascent occurs and the same volume of gas will cause the IAB to expand more, in theory causing the IAB to rupture. To prevent this complication, when operating an IABP console that is not capable of altitude adjustments, the pilot should be asked to coordinate altitude changes with the patient care crew. Slow ascents and descents allow the opportunity to adjust the pump. For ascent, the extent to which the IAB inflates should be reduced. Most IABP consoles allow a reduced inflation assist. When cruising altitude has been reached, the IAB should be refilled at the new altitude. For descent, the balloon should be kept inflated at full-assist capacity, and the procedure (refilling the balloon at the new altitude) should be repeated when on the ground.

Patient Assessment

Of the patients who are admitted to a community hospital with an acute MI, cardiogenic shock develops in 5% to 10%. Because the IABP can be an effective means of support for patients in LV failure and cardiogenic shock, personnel in these facilities may initiate IABP therapy. Unfortunately, the hospitals may not be equipped with cardiac surgery or cardiac catheterization laboratories or have the ability to perform interventional cardiology procedures. When the choice is to transfer patients with such conditions to a tertiary facility for further evaluation and treatment, early insertion of an IAB may make

transport safer by promoting hemodynamic stability. Some IABP-related considerations need attention before transport.

Assessment of a patient receiving IABP therapy revolves around two questions: (1) Is the therapy effective for the patient? (2) Are there complications resulting from the therapy? Patient assessment parameters that are useful for evaluation of the effectiveness of IABP therapy are vital signs, oxygenation, urine output, peripheral perfusion, central nervous system (mentation), and overall general condition. For a patient receiving correctly administered IABP therapy, one would expect, except in the most severe cases, that the patient would experience an improvement of each of the aforementioned parameters. Improvement of the vital signs, oxygenation, urine output, peripheral perfusion, and mentation of the patient can be expected because of the increase in CO. Some patients are confused during IABP therapy, which may be attributed to alterations in cerebral circulation induced by IAB counterpulsation. In the transport environment, the CCTP should be prepared to restrain or sedate an agitated patient receiving IABP therapy for the safety of the patient and crew. The CCTP should assess and document these parameters at least every 15 minutes or whenever there is a change in the patient's condition. Note that although the IABP will measure arterial blood pressure, it may be useful to measure the patient's noninvasive blood pressure (on the patient's arm with a blood pressure cuff), for reference.

Complications that may result from IABP therapy are most often related to the insertion site of the device or impairment of circulation by the balloon or the extremity where it is inserted. When in proper position, the IAB lies in the thoracic aorta just distal to the takeoff of the left subclavian artery. If during transport the IABP were to migrate toward the patient's head (**cephalad**), the distal portion of the IAB could occlude the takeoff to the left subclavian artery, which may be exhibited as a dampening or absence of the pulse in the left arm. Therefore, the CCTP should palpate or, if necessary, use a handheld Doppler ultrasound device to assess the left radial pulse and compare it with the right radial pulse when performing vital signs or patient assessments. Placing a pulse oximeter probe on a finger of the left hand would also provide early warning of compromised circulation to the left subclavian artery. In addition, the IAB may migrate toward the feet, causing an impairment in renal function. The depth of insertion of the IAB should be monitored and documented. A sudden decrease in urine output may also signal this impairment in renal blood flow, and, therefore, urine output should be noted and documented.

Special Populations

Be certain to evaluate mental status and level of orientation prior to transport. IABP therapy may lead to confusion necessitating physical or chemical restraint during transport. In geriatric patients, it is important to determine whether confusion is normal behavior for the patient (such as with dementia) or the result of altered cerebral blood flow secondary to IABP counterpulsation.

As mentioned earlier, the majority of IABs are inserted via the percutaneous technique in the femoral artery. This site should be inspected for evidence of bleeding, externally or internally. Any hematomas that have developed should be noted and marked on the skin to allow the CCTP or other health care provider to assess progression of the hematoma. Care should be taken to inspect the buttocks and posterior hip area because bleeding in this area may be difficult to detect. If the femoral artery is used as the insertion site, the CCTP should assess and document the presence of the distal pulses and capillary refill time (CRT) in the extremity used and compare it with the distal pulses and CRT in the other leg. The CCTP should splint the knee of the leg in which the IAB was inserted to prevent excessive movement that might lead to bleeding at the site or impairment of distal circulation. The sudden loss of pulses or decrease in CRT could signal that the IAB is occluding the femoral artery and causing ischemia in the

limb. If this complication occurs, medical control should be notified immediately and consideration given to the removal of the device in a facility as soon as feasible. In addition, because circulation in the extremity may be impaired, the potential for compartment syndrome to develop is high while the catheter is in place and after it has been removed. Compartment syndrome is heralded by the five Ps: Pain (out of proportion to what is expected), Pallor, Paralysis, Pulselessness, and Paresthesia. Also, the extremity might feel cold to the touch.

Emergency Interventions

There are several potential situations to troubleshoot during IABP therapy that are discussed in the following sections.

Cardiac Arrest

If cardiac arrest occurs, the trigger selection should be set to "pressure" and CPR started. The IABP will continue to operate in synchrony with compressions, augmenting diastolic flow and improving coronary perfusion. Defibrillation should be provided as necessary. There is no need to disconnect the IABP or associated monitoring equipment when shocking the patient. Like standard ECG and hemodynamic monitoring, IABP consoles are electrically isolated to prevent damage from defibrillators. Compression depth can be readily adjusted according to the MAP displayed on the IABP console. A minimum MAP of 60 mm Hg is necessary to perfuse vital organs during resuscitation.

Signs and Symptoms

Cardiac Arrest During IABP Therapy

- Pulselessness
- Flattening of unassisted arterial pressure waveforms. (The IABP will continue to generate regular waveforms if set to the ECG trigger and electrical complexes are present. *Do not* mistake the IABP pressure waveforms for evidence of cardiac output!)

Transport Management

Cardiac Arrest During IABP Therapy

- Switch the IABP trigger to pressure.
- Initiate CPR.
- Provide standard ACLS and BCLS resuscitation, including defibrillation, if indicated.
- Once return of spontaneous circulation occurs, MAP is adequate, and the ECG rhythm is stable, return the IABP trigger to the ECG mode.
- Reassess timing.

Balloon Rupture or Leak

Perhaps the most catastrophic complications that can result from IABP therapy are balloon rupture and air embolus. Although they are extremely rare occurrences, most balloon ruptures follow repeated friction of too large a balloon or a properly sized balloon located low in the thoracic aorta where sharp calcific

plaques tend to be most prominent. The CCTP should inspect the IAB tubing often for the presence of blood in the air channel. Although the central canal of the catheter functions like an arterial line and, therefore, is expected to occasionally have blood in the tubing, the gas lumen and channel of the catheter should never contain blood. If a chlorhexidine 2% skin preparation was used before insertion of the IAB catheter, flecks of the prep solution on the tubing may appear at a quick glance to be blood inside the tubing, so caution is required to distinguish blood inside the tubing from preparation solution or another substance on the outside surface. If blood is noted in the gas lumen of the catheter, IABP therapy should be discontinued immediately. The catheter should be disconnected, and, if signs or symptoms of gas embolus are present, place the patient in a left lateral recumbent position **Figure 15-22**, administer high-flow oxygen, and transport to the nearest appropriate facility. Although helium gas can leak into the bloodstream if the balloon ruptures, it is highly unlikely for two reasons: (1) Most IAB ruptures are miniscule holes. (2) During inflation, the gas pressure within the balloon is considerably less than the pressure in the aorta. Instead, blood is more likely to leak backward into the balloon, and this happens during deflation. A leak in the catheter may be signaled by an "excessive gas loss" alarm from the IABP console. Causes of gas loss alarms include high fever and significant tachycardia, both of which accelerate normal loss of gas from the circuit, as do loose connections or pinholes in the IAB tubing. If a gas loss alarm is triggered, the patient and tubing should be carefully inspected to determine the cause of the alarm. If after acknowledging this alarm, a source of a leak cannot be determined, the CCTP should assume that an IAB leak is a possibility and follow the aforementioned procedures.

A sudden loss of augmentation on the arterial line waveform and blood in the IAB catheter may be noted. If this occurs, the console to the patient should be shut off and medical control notified.

Console Failure

If the IABP console becomes disabled, manual inflation and deflation should be performed as soon as possible. The balloon must not remain idle (ie, immobile) in a patient for more than 30 minutes.

Skill Drill 15-2 gives the steps to manually inflate and deflate the IAB catheter.

1. Disconnect the IAB from the male Luer end of the extension tubing **Step 1**.
2. Attach the three-way stopcock and 60-mL syringe to the IAB catheter's male Luer **Step 2**. *Warning: Never inject air into the central lumen (female Luer hub).*
3. Aspirate to ensure that blood is not returned through the extracorporeal tubing. *Warning: If blood is aspirated from male Luer fitting of the extracorporeal tubing, immediately remove the IAB catheter owing to damage to the IAB.*
4. Inflate the IAB with 40 mL of air or helium, and immediately aspirate **Step 3**. Repeat every 5 minutes while the IAB is inactive.

The purpose of inflating and deflating the balloon manually is not to replicate the physiologic effects of IABP therapy, but rather to keep the balloon from accumulating clots in the folds while idle or immobile in the aorta.

Figure 15-22 The left lateral recumbent position.

Depletion of Helium

The IABP console user screen includes an indicator of the pressure in the helium tank. Most consoles also include a conventional pressure gauge in the proximity of the helium tank for use when the console is not in operation. A full helium tank will usually allow many weeks of IABP therapy without need for replacement. Should the tank require replacement, the console will provide visual and audible alerts. If the console indicates an empty helium tank, the CCTP should first ascertain that the tank valve is opened. Helium tanks operate in the same manner as oxygen tanks and are connected to the IABP console through a yoke similar to those found on oxygen regulators. The pin-index system for helium tanks will seat only into a helium yoke.

Should a tank require replacement, the valve on the depleted helium cylinder must be closed and the tank replaced with a full one. As with oxygen, a washer is required to ensure a tight seal between the tank and the yoke. Although it is unlikely that a helium supply would be completely depleted during a transport, it is possible for bumps and movement during transport to loosen or dislodge a helium tank from the IABP console, resulting in depletion of the helium supply. A spare (full) helium tank should always be carried during transport.

Excessive Bleeding

If excessive bleeding is noted from the insertion site, the CCTP should apply direct pressure over the site to control the bleeding. The CCTP should also consult with medical control for consideration of discontinuing infusions of anticoagulant medications or, if the bleeding cannot be controlled, diverting to the closest appropriate facility.

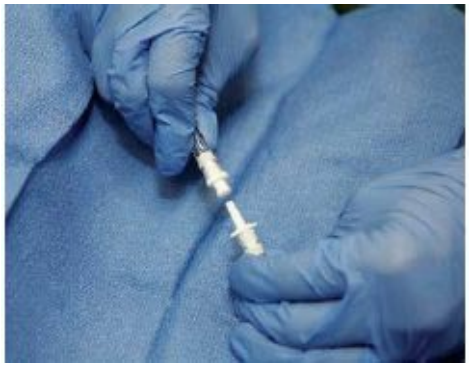
Catheter Migration or Accidental Removal

The uncontrolled environment of the prehospital arena presents unique challenges. The movement of patients presents opportunities for lines and attached devices to be removed accidentally. Before moving a patient, note the location and insertion mark of the IABP catheter; and before the patient is transferred to the stretcher, apply a knee splint to the leg in which the IAB was inserted. Tape the IAB catheter securely to the patient's leg. To reduce the chance of dislodging the IAB, IAB tubing connectors should not be taped together; if they become caught or tangled during movement, it is far better to have them pull apart. IAB catheters have sterile sheaths that allow for adjustment of the insertion depth without contamination of the catheter. If the catheter has moved significantly but has not become dislodged or disconnected from the sterile sheath protector, the IABP should be placed in standby mode (pumping stopped) and the catheter reinserted to the appropriate depth. Significant accidental movement of more than a few centimeters should not be adjusted without consultation with medical control or the referring physician. If the movement of the catheter occurred at the referring facility, obtaining a chest radiograph before

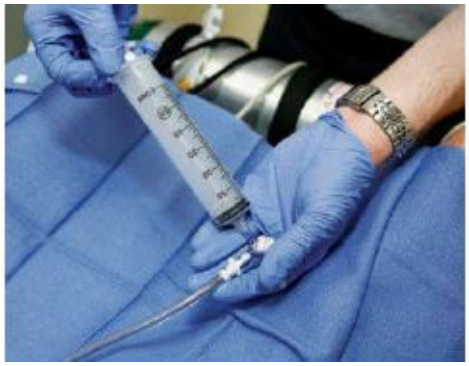
transport would verify the location of the catheter. If significant movement of the catheter occurred before transport, the physician who inserted the catheter can be asked to evaluate its position or potentially replace it before transport. If significant movement or accidental removal of the balloon occurs during transport, consider discontinuing IABP therapy after consultation with medical control. Allowing IABP therapy to continue with a misplaced balloon could cause significant complications, such as impaired renal blood flow or significant limb ischemia.

Skill Drill 15-2

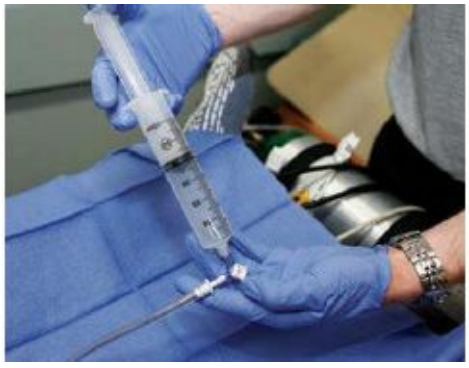
Manual Inflation/Deflation of an IAB



1 Disconnect the IAB from the male Luer end of the extension tubing.



2 Attach the three-way stopcock and 60-mL syringe to the IAB catheter's male Luer. *Never inject air into the central lumen*. Aspirate to ensure blood is not returned through the extracorporeal tubing. *Immediately remove the IAB catheter if blood is aspirated from the male Luer fitting of the extracorporeal tubing*.



3 Inflate the IAB with 40 mL of air or helium, and immediately aspirate. Repeat every 5 minutes while the IAB is inactive.

Transport Management

Excessive Bleeding at IAB Insertion Site

- Apply direct pressure.
- Be certain that pressure is applied over the arterial insertion site (not the external puncture site).
- If bleeding cannot be controlled, consult with medical control:
  - Consider discontinuing anticoagulant infusions (ie, heparin).
  - Consider diverting to the closest appropriate facility.

Special Populations

Control excessive bleeding with direct manual pressure to the IABP insertion site. This is particularly important in geriatric patients whose tissues are less elastic and therefore are more prone to injury and bleeding, for example as a result of sudden movement. Controlling excessive bleeding should not interfere with ongoing IABP therapy.

Cardiac-Assist Devices

In addition to the IABP, CCTPs may encounter a wide variety of cardiac- or circulatory-assist devices. Transport may be required for patients on **extracorporeal membrane oxygenation (ECMO)** or patients with a **ventricular-assist device (VAD)** (which can be an implanted right ventricular-assist device [RVAD], left ventricular-assist device [LVAD], or biventricular-assist device [BiVAD]). There are dozens of such devices presently in use worldwide, and newer, more sophisticated devices regularly appear on the market. The complexity of circulatory-assist technology precludes a complete review of every device, but there are some important considerations for the safe transport of these patients.

Figure 15-23 An extracorporeal membrane oxygenation circuit consists of a pump, a membrane oxygenator, and a heat exchanger, and is connected to the patient by an arterial or venous cannula.

ECMO is not therapy itself; it is an adjunct to oxygenation and ventilation and considered life support for a patient with cardiac and/or respiratory failure. ECMO ensures adequate oxygenation by

replacing the function of the lungs in gas exchange. An ECMO circuit is managed by a specially trained critical care registered nurse or by a perfusionist. The circuit includes an arterial or venous cannula that is connected to the patient and has a pump that directs bloodflow into the patient, a membrane oxygenator that adds oxygen and removes carbon dioxide, and a heat exchanger that replaces lost body heat **Figure 15-23**. Venous blood drains with gravity and the siphon effect of the pump. ECMO flow is nonpulsatile, meaning that ECMO pressure waveforms will be flat. Bleeding is the leading complication of ECMO because full-system anticoagulation is required to prevent thrombus formation. Other complications include pump failure, cannula dislodgement, cardiac arrest, or circuit problems such as an air embolus. Electrical and gas requirements need to be carefully assessed before transport to ensure an uninterrupted supply.

The most commonly encountered VAD is the LVAD, which can be fully implanted or externally delivered. Traditionally, LVADs have been a bridge-to-transplant option for patients with refractory end-stage heart failure. More recently, several VADs, including LVADs, RVADs, and BiVADs, have been approved as destination therapy, meaning they are an alternative to heart transplantation **Figure 15-24**. Temporary VADs are often used for patients with significant myocardial dysfunction as a consequence of infection, infarction, ischemia, or their inability to be weaned from cardiopulmonary bypass support following cardiac surgery. Criteria for placement of VADs require that patients be reasonable candidates for transplantation because it may not always be possible to wean a patient from VAD support.

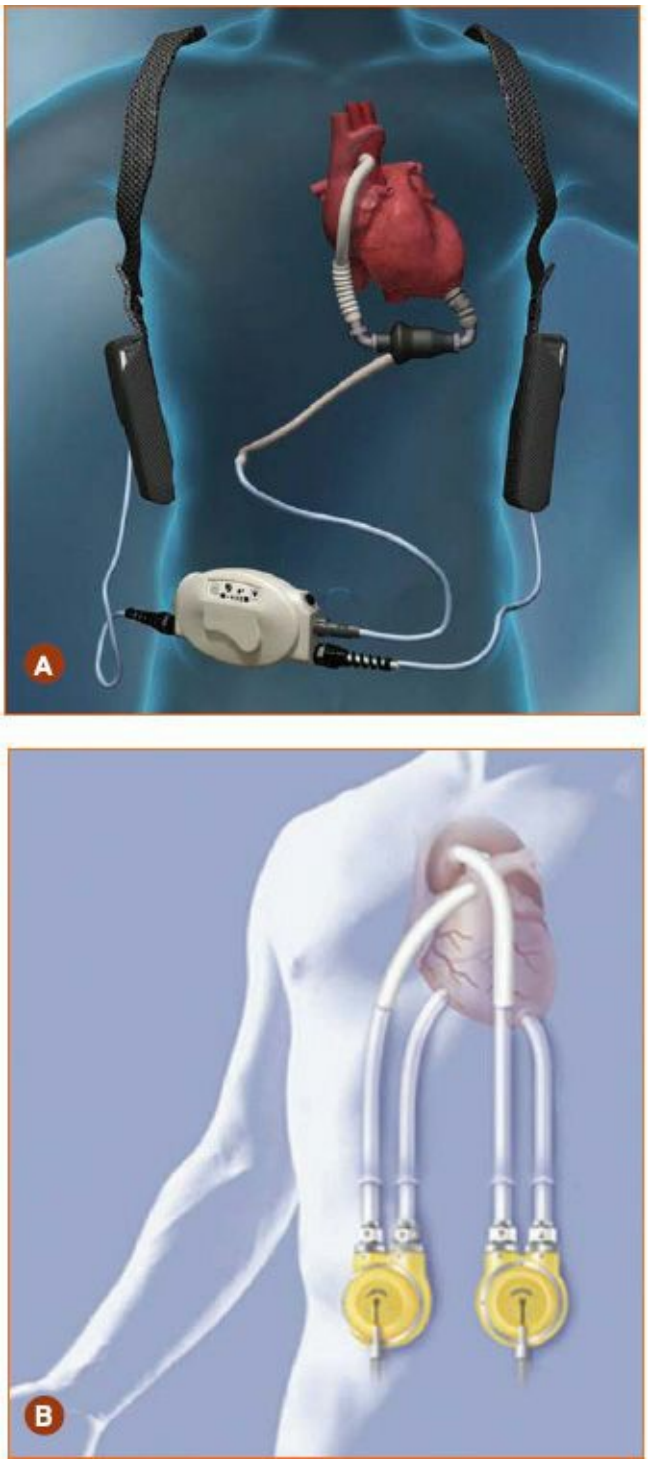


Figure 15-24 **A.** A left ventricular-assist device. **B.** A right ventricular-assist device.

Power requirements for VADs are usually less than those required for ECMO. Some VADs can operate for extended periods on batteries. The CCTP, in addition to ensuring that the transport vehicle has adequate power to supply or charge the VAD, should ensure that all necessary adapters, connectors, and charging units accompany the patient during transport. Almost all VADs have some ability to be manually operated using a hand or foot pumping accessory in case of mechanical or power failure. Be certain that such emergency equipment also accompanies the patient.

Complications of VADs in the early postimplantation period include bleeding, device malfunction, and thromboembolism. When a single-sided RVAD or LVAD is implanted, close observation and interventions to avoid failure of the opposite ventricle are extremely important. Later complications include infection and gastrointestinal problems (often seen when totally implanted VADs compress the gut, altering digestive processes). Anticoagulation requirements for VADs range from a single aspirin

taken daily to full systemic anticoagulation and close observation of clotting times. Point-of-care coagulation monitoring devices may be needed during transport.

VAD cannulas are usually tunneled through the chest into the heart. Invariably, these connections are large and may be affected by both cannula and patient positioning. As in ECMO, filling a VAD depends on gravity as well as patient fluid volume status. In cases of cardiac arrest, it may be inadvisable to perform CPR depending on the type and placement of VAD cannulas. Treatment of arrhythmias either pharmacologically or with defibrillation is typically attempted before considering CPR. Disconnection of VAD driver devices, while typically recommended by manufacturers, is not routinely done during defibrillation.

Finally, while most VADs provide pulsatile flow (meaning that pressure waveforms will be clearly visible on hemodynamic monitoring devices), some provide only centrifugal flow, which produces no pulsatile waveforms. Regardless of whether VAD flow is pulsatile, CCTPs should be aware that hemodynamic waveforms representing VAD flow will not match cardiac electrical activity on transport monitoring equipment. Additionally, pressure waveforms representing the cardiac activity of the patient may be visible interposed on pressure waveforms generated by the VAD. Interpretation of these data requires practice and experience.

A CCTP would not be expected to care for a patient with a VAD independently, nor should a CCTP ever accept that responsibility. A critical care registered nurse with specialized VAD training or a perfusionist should always accompany a patient with a VAD during transport. The CCTP provides an additional pair of skilled hands to assist these specialists.

Summary

Effective IABP therapy rests on an understanding of the mechanical cardiac cycle and the hemodynamics of cardiac blood flow and aortic pressures. The arterial waveform offers a window into this information.

Patients with a number of cardiac-, systemic-, and procedure-related conditions can benefit from IABP treatment. Common factors in many of these conditions include alterations in cardiac workload and its frequent consequence and disruption of the myocardial oxygen supply-demand balance.

Properly timed IAB pulsation produces a number of helpful hemodynamic effects: reduced afterload and HR, improved diastolic pressure, and lowered systolic pressure. These effects, in turn, lower MVO_2 and improve coronary artery and peripheral arterial perfusion.

IABP therapy is highly technical. In addition to the knowledge of physiology required, it requires a high degree of familiarity with the specific equipment used during transport, the ability to troubleshoot the equipment when problems arise, and—when transporting by air—awareness of the effects of atmospheric pressure changes on IAB function.

A CCTP may also need to assist a critical care registered nurse or perfusionist in transporting patients with VADs.

Case Study

YOU AND YOUR CRITICAL CARE TRANSPORT TEAM are dispatched to a suburban hospital for a 52-year-old man in the cardiothoracic intensive care unit (CTICU) with cardiogenic shock. On obtaining a more detailed patient report before leaving your base station, you learn that the patient underwent coronary artery bypass graft surgery and mitral valve replacement less than 24 hours ago. Since arriving in the CTICU after surgery, the patient's condition has been unstable, requiring the placement of an IABP, transfusion of numerous blood products, and administration of multiple inotropic and vasoactive medications. Your team has been requested to transport the patient by helicopter to a university hospital

for further and more advanced cardiac care not available at the referring facility. Your total flight time to the university hospital is 25 minutes.

On your arrival in the CTICU, you and your partner are met by the patient's nurse who says, "This patient is sick! He has required two-on-one nursing care ever since he has gotten to us. His blood pressure has been all over the place, and we have been titrating his 'pressors' continuously." The nurse takes you out to the hallway to give you a more detailed report. She reports to you that the patient had an acute inferior wall MI two days ago. He was brought to the local emergency department by an ALS ambulance, and an MI was diagnosed by the emergency department physician. The patient was immediately transferred to the cardiac catheterization lab, where he was found to have an ejection fraction of 20% accompanied by severe (> 95%) stenosis in five major vessels. The patient underwent stent placement, and an IABP was inserted to help the patient's heart muscle recover from the damage caused by the MI.

In the cardiac care unit 16 hours later, the patient experienced excruciating chest pain and shortness of breath. A 12-lead ECG confirmed that the patient was experiencing another MI caused by the occlusion of the just-placed stent and rupture of a papillary muscle, resulting in mitral valve regurgitation. A cardiothoracic surgeon was consulted, and the patient was taken to the operating room for an emergency coronary artery bypass graft procedure and valve repair. The patient was in surgery for 6 hours and has been in the CTICU for 12 hours.

As you walk in the patient's room to complete your assessment, you notice that the patient is intubated and receiving mechanical ventilation and the following infusions:

- Propofol (used for sedation during ventilator use)
- Milrinone
- Epinephrine
- Fentanyl
- Norepinephrine
- Insulin

The ventilator settings are as follows: tidal volume, 700 mL; rate, 12 breaths/min (assist control); fraction of inspired oxygen, 100%; and positive end-expiratory pressure, 5 cm H_2O. The patient has bilateral pleural chest tubes and one mediastinal drainage tube that are draining into a water-seal container with 900 mL of blood in the collection container. You also notice that he has a pulmonary artery catheter inserted through the right internal jugular vein, a left radial artery line, an indwelling urinary catheter, and a transvenous pacemaker. Despite receiving the aforementioned medications, the patient remains hypotensive with systolic readings as low as 60 mm Hg and no higher than 96 mm Hg. His CO is 3.0 L/min with a pulmonary artery pressure of 20/12 mm Hg and a dampened central venous pressure waveform reading of 2 cm H_2O. The IABP is on an assist ratio of 1:1 and is triggering from the patient's ECG rhythm. Other pertinent vital signs are as follows: noninvasive blood pressure, 76/32 mm Hg; arterial blood pressure, 80/41 mm Hg; pulse rate, 88 beats/min and normal sinus rhythm; Spo_2, 97%; respiratory rate, 12 breaths/min; and average hourly urinary output, 40 mL.

You and your partner prepare the patient for transfer to your helicopter by attaching your equipment to the patient. You maintain the infusions at their current rates because you are satisfied with the patient's current blood pressure and HR. Once in the helicopter, you notice that the patient is beginning to breathe more than the set ventilatory rate, his HR is increasing, and he seems to be becoming agitated. You and your partner discuss your standing protocols for ongoing sedation. The decision is made to titrate the propofol infusion from 10 µg/kg/min to 20 µg/kg/min and initiate a 1,000-mL normal saline fluid bolus to

assist in preventing a drop in blood pressure.

You arrive at the university hospital and prepare to transfer your care to the accepting nurse. You note that the patient is much less agitated and is no longer breathing at more than the set ventilatory rate. His blood pressure has responded appropriately to the normal saline fluid challenge and is significantly improved.

1. For this trip, what additional equipment might you need that is not already located in the helicopter?
2. Do you think a consultation with medical control should be made before departure? If so, what kind of orders are you going to request from the command physician?
3. What was the rationale for the insertion of the IABP in the case?

Analysis

Because most flight programs do not always carry their IABP, the flight crew must remember to reconfigure the aircraft to make room for the IABP. Additional IV pumps and tubing might be needed owing to the complexity of this patient's condition. It is always better to have extra supplies than not enough.

It is imperative that a consult with medical control be made before leaving the referring facility. The patient has multiple signs of dehydration and underresuscitation, which need immediate attention. When you call the medical control physician, make sure you have all pertinent lab results (ie, electrolytes, coagulation studies, and blood gases) so you can relay them to the physician. Remember, you are the medical control physician's eyes; the physician cannot see the patient, so you need to paint the clearest picture possible.

This patient is in desperate need of albumin and/or packed red blood cells for fluid replacement, which the flight crew did not address. You may also want to discuss blood pressure management if needed during transport. It is a good idea to plan ahead and ask the command physician which pressor to titrate if the blood pressure should fall. In this case, the flight crew did not consult medical control but, for obvious reasons, should have made the call.

This patient had a massive MI that affected a large part of his heart muscle. The damage to his heart muscle has left him with a very inefficient "pumping heart." The IAB was inserted to relieve the heart of the stressors and to take over the workload while giving the heart a rest. This patient has gone into cardiogenic shock despite use of the IABP, which should tell you that there is little collateral assistance from within the heart. This patient may even be a candidate for heart transplantation. This patient's CO is extremely poor, and the pulmonary artery catheter readings suggest that the patient is volume depleted, which needs to be addressed.

Prep Kit

Ready for Review

- In IABP therapy, a balloon connected to a pump via a catheter has been inserted into the aorta to provide temporary assistance to a failing heart.
- The IAB catheter is typically threaded into the descending aorta through the femoral artery.
- The balloon is connected to a console to shuttle helium gas, inflating the balloon at the onset of diastole and deflating just before systole (ventricular ejection).
- An ECG machine determines when the balloon should be inflated and deflated.

- Inflation and deflation are timed to the cardiac cycle, resulting in significant augmentation of flow to the coronary and renal arteries during diastole (increased supply) and substantially reduced afterload during systole (decreased demand).
- One of the CCTP's main roles in transporting a patient receiving IABP therapy is to ensure proper timing by reading the patient's ECG and pressure waveforms and continuously monitoring the patient's condition and all devices, modifying the timing if necessary.
- Other CCTP responsibilities include securing the patient to prevent movement of the balloon and ensuring that all equipment has the necessary power supply.
- The AV valves (mitral and tricuspid) lie between the atria and the ventricles, and the semilunar valves (aortic and pulmonic) are the exits from the ventricles. The purpose of the cardiac valves is to prevent the backward flow of blood in the heart.
- The AV valves have chordae tendineae, which attach the edge of the valve to the papillary muscles attached to the endocardium and hold the AV valves in place in their closed position and prevent them from prolapsing into the atria during systole or ventricular contraction. The semilunar valves do not have chordae tendineae; opening and closing are related to the pressure changes in the heart, aorta, and pulmonary artery.
- Systole and diastole include five phases: atrial systole, isovolumetric contraction, ventricular ejection, isovolumetric relaxation, and ventricular filling.
- In atrial systole, the pressure in the ventricles increases. The atria contract, extra blood is ejected from the atria into the ventricles, mitral and tricuspid valves are open, and pulmonary and aortic valves are closed.
- Isovolumetric contraction occurs between the closure of the AV valves and the opening of the semilunar valves (in which there is no change in ventricular blood volume) immediately before the blood is ejected through the semilunar valves. The pressure in the ventricles rises abruptly.
- During ventricular ejection, pressure in the left ventricle exceeds pressure in the aortic root, resulting in the aortic valve opening.
- During isovolumetric relaxation, pressure in the ventricles is below the arterial pressure. Ventricles are no longer contracting, mitral and tricuspid valves are closed, and the pulmonary and aortic valves are closed.
- Ventricular filling occurs once the mitral and tricuspid valves open.
- Pressure waveforms are one of the main variables evaluated to determine the effectiveness of IABP therapy.
- The dicrotic notch, a slight bump in the aortic pressure waveform that signals the onset of diastole, occurs during isovolumetric relaxation as the valves bulge under the pressure of the blood from the aorta and then spring back slightly.
- In heart failure, myocardial oxygen supply and demand are mismatched. IABP therapy offers a mechanical means of rebalancing them by helping to increase the blood flow to the heart muscle, thereby decreasing the workload.
- Each IABP console allows the operator to control the volume of gas shuttled back and forth to the balloon with each counterpulsation cycle. This control is variously referred to as augmentation or balloon volume.
- The arterial pressure waveform that the IABP console monitors may come from the central lumen of the IAB catheter, from the fiberoptic catheter, or from a peripheral arterial catheter in the patient.

- IAB inflation and deflation can be set to trigger from the ECG, pressure changes, a pacemaker, an internally preprogrammed rate, or combinations of these. The most reliable is the ECG. The provider caring for the patient chooses the trigger best suited to the patient.
- The IABP device can pump in several modes, called assist ratios or IABP frequency. IABP waveform evaluation is usually easiest in the 1:2 assist ratio, which allows comparison of pressure waves of the unassisted cycle with the assisted one. In this mode, every other contraction is augmented by the balloon pump.
- When evaluating a waveform in the 1:2 mode, the peaks created by the inflation of the IAB are usually the highest pressure peaks and are referred to as the diastolic augmentation waveforms. The pressure waveform that follows is referred to as the assisted systole waveform because the deflation of the IAB assists the ventricle in ejecting blood. The third pressure waveform is referred to as the unassisted systole because the balloon pump is not moving and the waveform reflects what would normally occur without IABP intervention.
- To evaluate for correct IABP timing, the pressure peaks, slopes, and inflation and deflation points of each of these pressure waveforms must be evaluated in a systematic manner, as follows:
  - Identify systole and diastole of each pressure waveform.
  - Identify diastolic augmentation, assisted systole, and unassisted systole.
  - Identify and evaluate the point of inflation.
  - Identify and evaluate the end-diastolic dip in pressure created by balloon deflation.
  - Evaluate the slope of diastolic augmentation pressure and the slope of assisted systole.
  - Evaluate diastolic augmentation peak pressure.
  - Compare the peak pressure of assisted systole with the peak pressure of unassisted systole.
- Early inflation is reflected on the waveform by a shortened normal pressure decrease that follows diastole or as an opened V and with a lessened slope on the diastolic augmentation waveform. Early inflation can force the premature closure of the aortic valves, decrease the stroke volume, and damage the aortic valve.
- Late inflation causes little harm but does not maximize the benefits of IABP therapy.
- Early deflation also results in the benefits of IABP therapy not being fully realized.
- Late deflation, which can be extremely detrimental, is characterized by a widened appearance of the diastolic augmentation waveform base and a prolonged rate of rise in the assisted systolic waveform. Late deflation *worsens* myocardial ischemia and needs to be avoided.
- IABP therapy reduces cardiac work and myocardial oxygen consumption and improves peripheral perfusion to patients with a variety of conditions and undergoing cardiovascular and surgical procedures.
- Patients receiving IABP therapy are candidates for transport in ground ambulances or in fixed- or rotary-wing aircraft. Some issues to consider when using these vehicles include space and weight constraints, battery life and vehicle power supply, and loading, unloading, and securing the pump in the vehicle.
- Many IABPs are capable of operating on 110- to 120-V AC power or 24-V DC power, although the latter may require an adapter cable. Ensuring a match between IABP power requirements and the available power supply in the transport vehicle is crucial.
- In aircraft, space limitations may require reconfiguration of the pump and monitor module.
- Once the IABP is in the transport vehicle, it is critical to secure the pump so that it remains stable for

safe operation throughout transport.

- Steps directly related to operating an IABP during transport include the following (other steps are needed for quality care):
  - Ascertain current IABP settings and note typical pressures.
  - Determine IAB catheter model, size, and insertion depth.
  - Ascertain that the balloon tip location has been verified by chest radiograph.
  - Attach new ECG leads and secure each lead over the electrode with 2″ cloth tape.
  - Ensure that the IAB catheter is taped securely to the patient's leg.
  - Apply a knee immobilization splint to the leg in which the IAB was inserted to prevent leg flexion during transport.
  - Ensure that the appropriate connectors to attach the IABP to the transport console are available. Determine the console that will be used at the receiving facility, and be certain to take necessary adapters or connectors.
  - Move the patient to the transport stretcher. Connect and secure all pumps, monitors, ventilators, and other equipment.
  - Transfer the IABP to the transport console.
  - Establish power to the transport IABP console.
  - Open the helium tank and verify pressure.
  - Follow the IABP console instructions for start-up.
  - Optimize IABP timing in a 1:2 mode.
  - Assess pressures every 5 minutes or with any changes in patient condition. Check insertion site and pulses every 15 minutes.
- Atmospheric pressure changes can affect IAB performance. With ascent, balloon pressure and volume can rise. With descent, balloon pressure and volume can decrease. Some IABPs compensate for pressure changes automatically.
- The autofill mode should be used during air transport; if the autofill mode cannot be used, a manual fill must be performed at the same intervals that an autofill would occur.
- Patient assessment parameters useful for evaluation of effectiveness of IABP therapy are vital signs, oxygenation, urine output, peripheral perfusion, central nervous system (mentation), and overall general condition of the patient.
- To detect compromised circulation to the left subclavian artery, the CCTP should palpate or use a handheld Doppler ultrasound device to assess the left radial pulse and compare it with the right radial pulse. A pulse oximeter probe on a finger of the left hand also provides early warning of this complication.
- Inspect the insertion site, buttocks, and posterior hip area for evidence of bleeding. Note hematomas, and mark their borders to monitor progression.
- If the femoral artery is used as the insertion site, assess and document the presence of the distal pulses and CRT in the extremity used and compare the pulses and CRT with those in the other leg. Splint the knee to prevent excessive movement of the leg. The sudden loss of pulses or CRT could signal IAB occlusion of the femoral artery, causing ischemia in the limb. Notify medical control immediately.
- Emergency situations to troubleshoot when an IABP is in use include cardiac arrest, balloon rupture or leak, console failure, an empty helium tank, excessive bleeding, and catheter migration or accidental removal.

- If cardiac arrest occurs, the trigger select should be set to "pressure" and CPR started. Defibrillation should be provided as necessary. There is no need to disconnect the IABP or any associated monitoring equipment when shocking the patient.
- Balloon ruptures are rare. Inspect the IAB tubing often for the presence of blood in the air channel of the tubing. The gas lumen and channel of the catheter should never contain blood. If blood is noted in the gas lumen of the catheter, IABP therapy should be discontinued immediately. For signs or symptoms of gas embolus, the patient should be placed in left lateral recumbent position, high-flow oxygen should be administered, and the patient should be transported to the nearest appropriate facility.
- Carefully inspect the patient and tubing to determine the cause of a gas loss alarm. If a source of a leak cannot be determined, assume that an IAB leak is a possibility.
- If the IABP console becomes disabled, perform manual inflation and deflation as soon as possible. The balloon must not remain idle (ie, immobile) for more than 30 minutes, or clots can accumulate in the folds of the balloon.
- An empty helium tank should be indicated by the IABP console screen. First ascertain that the tank valve is opened. Should a tank require replacement, close the valve on the depleted helium tank and replace the tank with a full one.
- If excessive bleeding is noted from the insertion site, apply direct pressure over the site to control the bleeding. Consult with medical control about discontinuing infusions of anticoagulant medications or, if bleeding cannot be controlled, diverting to the closest appropriate facility.
- Accidental catheter migration or removal can occur. Note the location and insertion mark of the IAB catheter before moving the patient.
- ECMO is life support for patients with cardiac and/or respiratory failure and must be managed by a specially trained critical care nurse or perfusionist.
- The leading complication of ECMO is bleeding, but complications can also include pump failure, cannula dislodgement, cardiac arrest, or air embolus. CCTPs need to carefully assess electrical and gas requirements before transport.
- CCTPs should ensure that all emergency equipment accompanies the patient with VAD, including manually operating materials in case of mechanical or power failures. Complications of VADs include bleeding, device malfunction, and thromboembolism in the early postimplantation period. Infection and gastrointestinal problems can also occur.
- Coagulation monitoring devices should be considered for transport.
- CCTPs should treat arrhythmias pharmacologically or with defibrillation before considering CPR for patients with VADs.
- CCTPs should know that the hemodynamic waveforms representing VAD flow will not match cardiac electrical activity, and interpretation of the data requires practice and experience.
- CCTPs should never be expected to care for patients with VADs independently.

Vital Vocabulary

afterload An expression of cardiac work related to the forces that must be overcome to eject blood from the ventricle.

aortic regurgitation Backward flow of blood through the aortic valve from the aorta into the left

ventricle.

assisted systole The pressure waveform that follows the diastolic augmentation peak.

assist ratio A setting on the IABP that allows the operator to determine how often the pump inflates the balloon. A 1:1 setting means the balloon inflates with each heartbeat; and 1:2, with every other heartbeat, etc; also called IABP frequency.

atrioventricular (AV) valves The valves (mitral and tricuspid) that separate the atria from the ventricles.

caudal Pertaining to or in the direction of the feet.

central lumen The lumen or port of the IAB catheter used to guide initial catheter insertion and to monitor arterial pressure during operation.

cephalad Toward the patient's head.

chordae tendineae The thin, fibrous strands stretching out from the apexes of some of the papillary muscles of the ventricles, extending upward and attaching to the edges of the AV valves, which help keep the cusps of the valves from turning inside out when pressure builds up in the ventricles.

diastole The relaxation phase of the heart cycle, in which the ventricles are dilated and filling with blood.

diastolic augmentation The increase in aortic pressure during diastole that IAB inflation produces, improving coronary and peripheral perfusion, and that may be thought of as a "second systole."

diastolic augmentation waveforms The waveforms caused by IAB inflation during diastole.

dicrotic notch The brief increase in aortic pressure reflected in a notching of the wave; caused by the sudden closure and spring back of the aortic valve leaflets; signals the start of diastole.

extracorporeal membrane oxygenation (ECMO) An adjunct to oxygenation and ventilation that consists of pumps, oxygenators, and heat exchangers to ensure adequate patient oxygenation, thereby replacing the function of the lungs in gas exchange.

Frank-Starling curve States that the force of the cardiac muscle contraction is proportional to the amount of stretch placed on the muscle fibers (meaning that the more the heart is stretched by the incoming blood, the more forcefully the ventricles contract and the more blood that is ejected); demonstrates how changes in ventricular preload lead to changes in stroke volume.

gas lumen The lumen or port of the IAB catheter that carries helium between the IAB and the pump console to inflate and deflate the balloon.

intra-aortic balloon pump (IABP) therapy A procedure involving a balloon inserted into the aorta and connected to a pump via a catheter, helping to increase the blood flow to the coronary arteries during diastole (inflation) and decrease afterload of blood from the left ventricle (deflation).

isovolumetric contraction The early stage of ventricular contraction during which ventricular blood volume is unchanging because all valves are closed (the semilunar valves have not yet opened).

isovolumetric relaxation The early stage of ventricular relaxation during which ventricular blood volume is unchanging because all valves are closed (the AV valves have not yet opened).

myocardial oxygen consumption (MVO_2) The volume of oxygen that the heart muscle consumes; an expression of the level of oxygen demand in the heart.

papillary muscles A type of muscle in the ventricle from which the chordae tendineae extend and attach to the cusps of the AV valves.

preload The amount of blood that flows into the ventricle passively from the atria; related to the overall volume status of the patient.

relative contraindication A condition that makes a particular treatment or procedure somewhat inadvisable but does not completely rule it out.

semilunar valves The valves (aortic and pulmonic) that are the exits from the left and right ventricles into the aorta and the pulmonary artery, respectively.

systole The contraction phase of the heart cycle when the ventricles pump blood out of the heart through the aorta and the pulmonary artery into the systemic and pulmonary circulatory systems.

timing In the context of IABP therapy, a method for coordinating the IAB inflation-deflation cycle with the cardiac cycle (inflation during diastole and deflation synchronous with systole).

unassisted systole Pressure waveform reflecting what would normally occur without IABP assistance.

ventricular-assist device (VAD) Sometimes an alternative to heart transplantation, this mechanical pump is surgically implanted and helps maintain a heart that can no longer function properly. It has primarily been used to support patients who are waiting for a heart transplant.

Bibliography

Guyton AC, Hall JE. *Textbook of Medical Physiology.* 10th ed. Philadelphia, PA: WB Saunders; 2000.

Joseph D, Shinn A. Concepts of intraaortic balloon counterpulsation. *J Cardiovasc Nurs.* 1994;8:45–60.

Joseph D. Timing waveform analysis. *Crit Care Nurs Clin North Am.* 1994;8:1996.

Joseph DL, Spadoni SM. CD-ROM: *Theory and Practice of Intraaortic Balloon Counterpulsation.* New York, NY: Center for Biomedical Communications, Columbia Presbyterian Medical Center; 1998.

Kahn JK. *Intra-aortic Balloon Pumping: Theory and Clinical Applications: A Monograph for the Clinician.* Princeton Junction: Communications Media for Education, Inc.

Kern MJ. Intra-aortic balloon counterpulsation. *Coronary Artery Dis.* 1991;2:649–660.

Maccioli GA, Lucas WJ, Norfleet EA. The intra-aortic balloon pump: a review. *J Cardiothorac Anesth.* 1988;2:365–373.

Mertlich G, Quaal SJ. Air transport of the patient requiring intra-aortic balloon pumping. *Crit Care Nurs Clin North Am.* 1989;1:443–457.

Pappas G, Winter SD, Kopriva CJ, Steele PP. Improvement of myocardial and other vital organ functions and metabolism with a simple method of pulsatile flow (IABP) during clinical cardiopulmonary bypass. *Surgery.* 1975;77:34–44.

Quaal SJ. *Comprehensive Intra-Aortic Balloon Pumping.* 2nd ed. St Louis, MO: Mosby; 1993.

Gastrointestinal and Genitourinary Emergencies

Authors

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Metabolic Regulation of

Acid-Base Status: Andrew Bartkus, RN, MSN, CEN, CCRN, CFRN, NREMT-P, FP-C

Objectives

1. Understand the anatomy and physiology of the gastrointestinal system, including the alimentary canal and accessory organs (p 630–634).
2. Understand the anatomy and physiology of the genitourinary system, including the urinary system and the male and female reproductive systems (p 634–638).
3. Differentiate between upper and lower gastrointestinal bleeding (p 639).
4. Understand the pathologies related to common disorders of the gastrointestinal system, including peptic ulcers, gastritis, esophageal varices, Mallory-Weiss syndrome, diverticulitis, angiodysplasia, inflammatory bowel disease, and ulcerative colitis (p 639–642).
5. Properly assess the signs and symptoms as well as describe the management of the various gastrointestinal conditions (p 643–646).
6. Describe laboratory results as they relate to specific gastrointestinal system disorders (p 645–646).
7. Describe gastrointestinal system imaging, including endoscopy, colonoscopy, angiography, and scintigraphy, as well as other in-hospital assessment and management techniques (p 646).
8. Understand the pathologies related to diseases of the gastrointestinal system, including intestinal obstructions, ileus, liver disease, choledocholithiasis, and pancreatitis (p 648–660).
9. Understand the pathologies related to common disorders of the genitourinary system, including both acute and chronic renal failure, urinary tract infections, testicular torsion, penile fracture, and priapism (p 661–666).
10. Properly assess the signs and symptoms as well as describe the management of the various pathologies discussed (p 646–665).
11. Describe genitourinary system laboratory results as they relate to the specific disorders (p 662–666).
12. Describe various gastrointestinal- and genitourinary-related feeding and drainage tubes, including their assessment, maintenance, and potential complications. Examples include various feeding tubes, different types of urinary catheters, ostomies, Jackson-Pratt drains, and T-tubes (p 667–674).
13. Understand flight considerations for patients with feeding or drainage tubes (p 672).
14. Understand acid-base physiology (p 674).
15. Examine how the body's chemical buffer system mitigates acid-base imbalances (p 674–675).
16. Describe how to interpret blood gas samples (p 675–677).
17. Understand the pathologies related to an imbalance of acid-base, their clinical features, and their treatment. Pathologies include metabolic alkalosis, metabolic acidosis, and renal metabolic acidosis (p 674–679).

18. Understand flight considerations for patients with gastrointestinal and genitourinary tract complications (p 679–680).

Introduction

Homeostasis (stability in the body's internal environment) depends on a number of metabolic processes that involve the gastrointestinal (GI) and genitourinary (GU) systems. Unfortunately, these processes can be the target of a number of pathologies causing a range of complications, such as malnourishment, pain, and shock. These conditions require a thorough assessment and diagnosis followed by appropriate management—a process that is inherently difficult because the signs and symptoms that accompany abdominal conditions may be obscure. A detailed understanding of related anatomy and physiology is therefore required for the CCTP to successfully manage a patient with a GI/GU condition during transport, along with any complications that may arise.

Anatomy and Physiology

Intricately complex yet stunningly efficient, the GI system consists of a specifically arranged network of organs and ducts devoted to the digestion of food and the extraction of its nutritional content. The alimentary canal (also referred to as the GI tract) consists of the mouth, pharynx, esophagus, stomach, small intestine, and large intestine; food travels through these structures and finally exits the body via the rectum. The alimentary canal is lined with continuous specialized tissue that allows for the absorption of digested nutrients from food while maintaining a protective barrier from waste and microorganisms. During mechanical and chemical digestion, food turns into the basic molecular building blocks required by the body's cells. Enzymes that aid in this digestive process are secreted by a variety of accessory organs, including the salivary glands, liver, gallbladder, and pancreas.

Unnecessary side products from the digestion process are either defecated via the anus or excreted by the GU system. Cooperation between the liver and kidneys allows for safe passage of toxic nitrogen-containing compounds during their exit from the body.

GI System

GI Tract Tissue

Although the alimentary canal contains numerous specialized cells and tissues, four layers are seen almost continuously from the esophagus to the rectum **Figure 16-1**: the mucosa, the submucosa, the muscularis externa, and the serosa.

The **mucosa** forms the first and innermost layer that is exposed to the lumen, or cavity, of the canal. This moist mucous membrane consists of three sublayers:

- The surface epithelium consists of a thin layer of simple columnar epithelium interspaced with goblet cells that secrete mucus. This mucus moistens the surface of the GI tract and protects the lining from being digested itself. Specialized absorption cells are found in the epithelium as well.
- The lamina propria, located immediately above the epithelium, consists of loose areolar connective tissue filled with capillaries and lymph nodules. It provides limited access to the circulation for digested molecules.
- A layer of smooth muscle called the muscularis mucosae lies above the lamina propria, creating folds in the tissue to increase the surface area available for nutrient absorption.

The **submucosa** consists of connective tissue just beyond the mucosa. Blood and lymphatic vessels weave through this layer to connect the vessels of the mucosa to systemic circulation. The submucosal

nerve plexus resides here, permitting innervation of smooth muscle and secretory cells. The elastic character of the submucosa allows for restoration of a structure's original shape after it is distended by food.

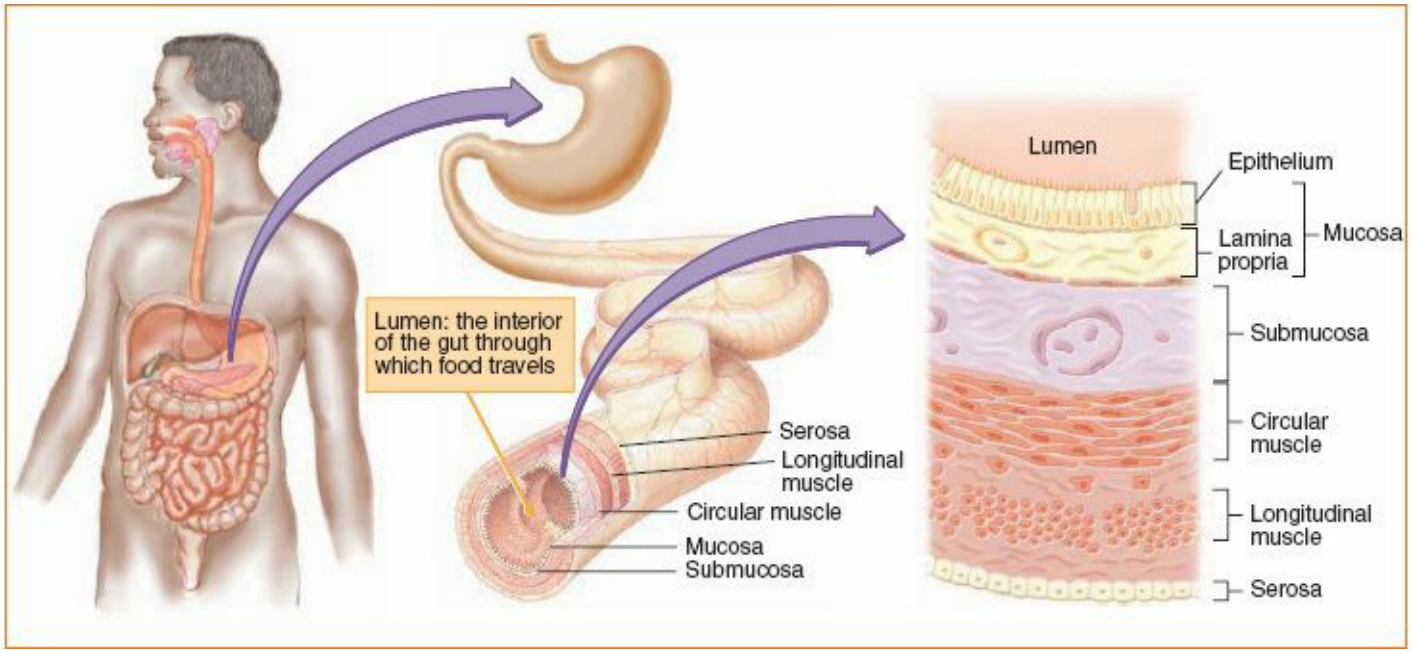


Figure 16-1 Gastrointestinal tract layers.

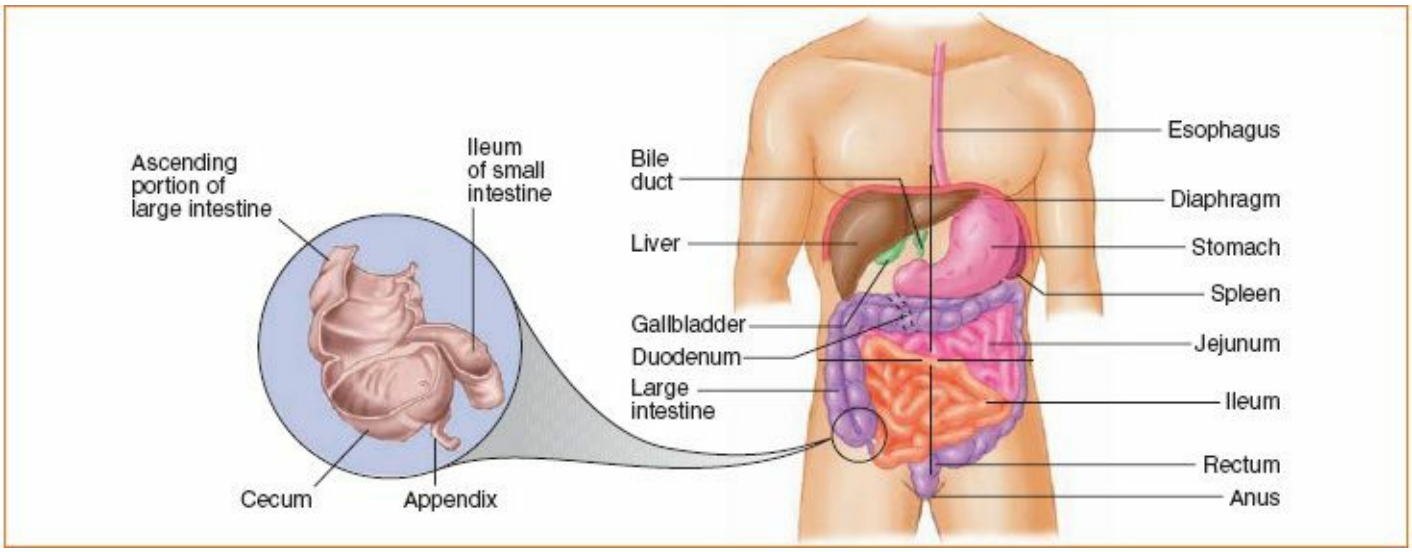


Figure 16-2 Overview of the gastrointestinal system.

The **muscularis externa** forms the muscle bulk of the GI tract. It is divided into two layers of smooth muscle: an inner circular layer and an outer longitudinal layer. The arrangement of these two layers facilitates the mechanical digestion methods of segmentation and peristalsis.

The **serosa** (commonly referred to as the visceral peritoneum) consists of areolar connective tissue lined with simple squamous epithelium, called the mesothelium. The serosa is meant to be a protective layer, covering all peritoneal GI organs.

Alimentary Canal

The alimentary canal **Figure 16-2** maintains a chemical environment that is beneficial for the digestion of food, without altering the environment of the rest of the body.

Mechanical digestion begins the moment food enters the oral cavity. Mastication (chewing) tears and

grinds food into more manageable chunks. The increase in the surface area of the food that results from this process facilitates chemical digestion, which begins when food within the mouth is moistened with saliva. Saliva contains salivary amylase, an enzyme that breaks down starch.

The food bolus, once sufficiently chewed, is pushed into the oropharynx by the tongue in preparation for swallowing. Swallowing becomes a largely involuntary process once initiated, albeit a fairly complicated one. More than 22 muscles are used to block off the nasopharynx with the soft palate and uvula, the larynx with the epiglottis, and the mouth with the tongue, thereby forcing food into the esophagus.

The esophagus is a 25-cm tube of muscle connecting the oropharynx with the stomach. A substantial flow of blood is supplied to the muscles of the esophagus via several arteries and returned through a large network of veins in the submucosa. Peristalsis, a series of wavelike contractions of the esophagus, carries food to the stomach in less than 8 seconds. The bolus of food arrives in the stomach via the gastroesophageal sphincter, which closes following the passage of a bolus, preventing backflow.

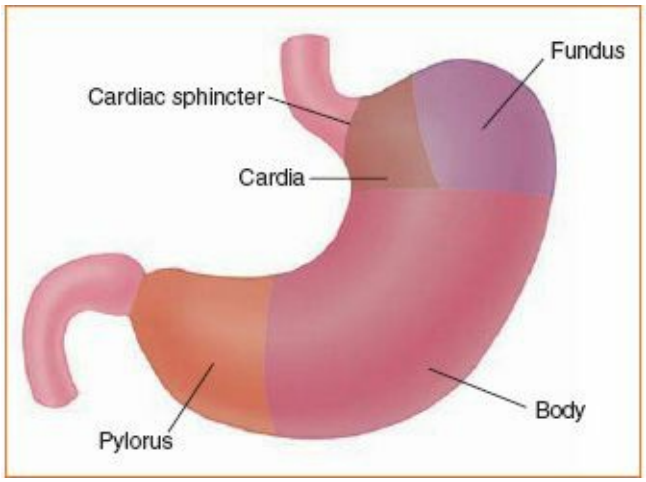


Figure 16-3 Anatomy of the stomach.

The stomach stores food while continuing chemical and mechanical digestion through a variety of enzymes and muscles. The stomach, which is bound superiorly by the esophagus and inferiorly by the small intestine, is a 15- to 25-cm J-shaped widening of the GI tract. When the stomach is full, it can expand up to 4 L. Mechanical digestion is continued and completed by the stomach muscles. A bolus of food enters the stomach at the cardia region via the cardiac sphincter. The stomach terminates at the pyloric sphincter, which regulates the exit of materials from the stomach **Figure 16-3**.

Throughout the stomach, glands consisting of specialized mucous neck cells produce a number of secretions, collectively termed gastric juice. Included in this juice are mucin, hydrochloric acid, intrinsic factor, pepsinogen, and lipases, all of which aid in the digestive process. The end result of the digestive actions that occur in the stomach is chyme, a liquid mixture of partially digested food, gastric juice, and water that is ready for entry into the small intestine.

The small intestine is a long, 2.5-cm-wide canal that twists throughout all four quadrants of the abdomen. Digestion is completed, and the bulk of nutrient absorption takes place here. This organ is divided into three areas: the duodenum, the jejunum, and the ileum. The superior mesenteric artery provides blood supply to all three of these regions via its vast network of vessels in the mesentery.

Chyme begins its journey through the small intestine at the duodenum. The small intestine also receives bile and pancreatic juice at the duodenum, both of which aid in further digestion.

The large intestine facilitates the transformation of chyme into feces and inevitable expulsion from the alimentary canal via the rectum and anus. The large intestine is divided into six regions: cecum, ascending colon, transverse colon, descending colon, sigmoid colon, and rectum **Figure 16-4**. The more

proximal sections (cecum, ascending colon, and transverse colon) provide water and electrolyte absorption, whereas the latter areas (descending colon, sigmoid, and rectum) provide storage for feces and muscles for propulsion to the rectum.

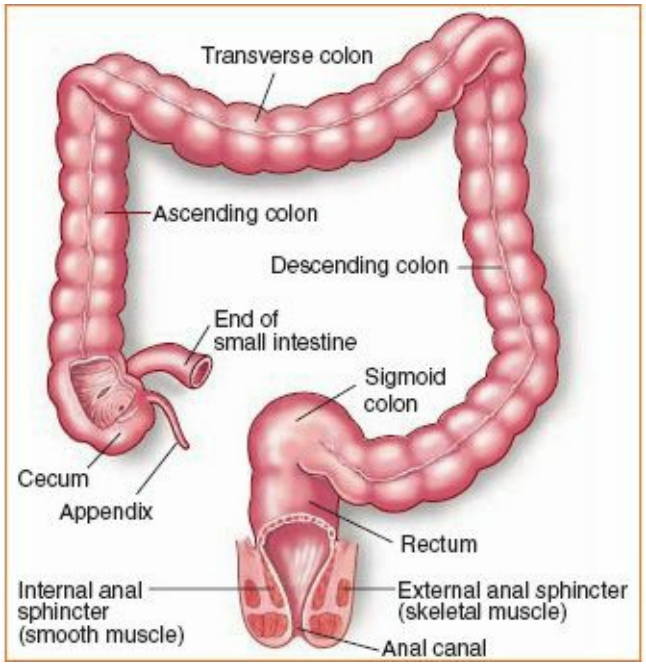


Figure 16-4 Anatomy of the large intestine.

Chyme is deposited into the cecum from the ileum of the small intestine, beginning the final leg of its journey through the body. The appendix attaches posteromedially to the cecum, about 2.5 cm inferior to the ileocecal valve. Chyme is pushed upward into the ascending colon, where water and electrolytes are reclaimed from unusable end products of digestion. Anaerobic bacteria residing here prepare a significant amount of vitamins B and K for absorption as well.

The transverse colon proceeds across the abdomen until it makes an inferior turn, after which it becomes the descending colon. These two sections function mainly to store feces. The muscularis externa begins to thicken gradually along the length of these areas to assist with the movement of increasingly compact feces.

The sigmoid colon starts at the level of the pelvis, taking an S-shaped path into the rectum, a 13-cm muscular tube. A defecation reflex is stimulated by the stretching of the rectal wall by feces buildup, resulting in the opening of the internal anal sphincter. The external anal sphincter is voluntarily controlled. Once it is opened, feces are defecated through the anus.

Accessory Organs

The alimentary canal does not accomplish the feat of digestion on its own. Similar to any other functioning system, it receives outside support from several accessory organs—namely, the salivary glands, liver, gallbladder, and pancreas.

Three pairs of extrinsic salivary glands inhabit the tissues surrounding the oral cavity, secreting saliva to initiate the chemical digestive process **Figure 16-5**. The saliva produced by these extrinsic salivary glands aids in digestion not only by providing enzymes that break down starches in food, but also by moistening food to facilitate bolus formation. In addition, saliva cleanses the mouth. Tiny internal salivary glands are located throughout the oral cavity as well, supplementing the secretion of the extrinsic glands. Secretion of saliva by all of these glands is amplified by stimulation of the salivatory nuclei in the brain stem, whether it is by the sight, smell, or taste of food. Alternatively, sympathetic nervous system stimulation promotes secretion of a thicker saliva with a higher concentration of mucin, resulting in a dry

mouth.

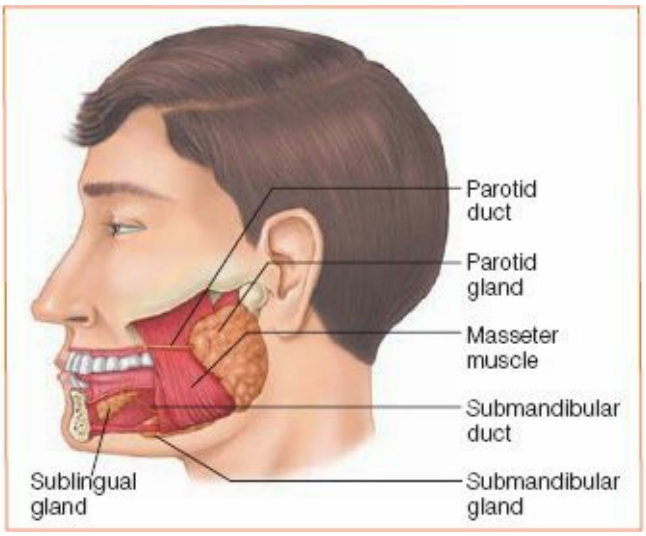


Figure 16-5 The salivary glands.

The liver is the largest solid organ in the body, as well as the most vascular, containing more than one fifth of a person's total blood volume at any given time **Figure 16-6**. It serves as a central terminal through which all blood returning to the central circulation from the alimentary canal must pass. It provides three functions critical to the body's functioning: metabolic regulation, hematologic regulation, and bile production. Although the liver dominates the right upper quadrant of the abdomen, the majority of its bulk actually rests within the right thoracic cage; in this position, the ribs are able to provide some protection from trauma. During forceful exhalation, the liver rises as high as the fourth intercostal space just below the right nipple, and sprawls past the midline approaching the left nipple. Normally the liver can be palpated inferior to the right ribs during inspiration only, remaining within the thoracic cage during expiration.

The porta hepatis provides the gateway into the liver for the hepatic portal vein, hepatic artery, and common hepatic duct, all of which arrive via the lesser omentum. These three vessels bifurcate upon entry, separating the liver into right and left functional lobes that are distinct from the superficial lobes. The hepatic portal vein and hepatic artery continue to branch out until they eventually form the liver's functional unit, a lobule. Inside a lobule, the incoming oxygen- and nutrient-rich blood is drained into liver sinusoids, where it comes into direct contact with hepatocytes and Kupffer cells. Hepatocytes, the most metabolically active cells in the body, account for 80% of the cells in the liver. They produce and secrete bile, process fundamental molecules into macromolecules (such as glucose into glycogen), store fatsoluble vitamins, and turn toxic nitrogen-containing wastes into urea. Kupffer cells are macrophages that reside in the sinusoids and engulf bacteria and old cells. The liver sinusoids drain blood into central veins that will eventually fuse to form the hepatic vein and ultimately the inferior vena cava, returning the blood to the systemic circulation. The secreted bile drains into a bile duct, which passes it into the common hepatic duct. From there the bile is either secreted directly into the duodenum or stored in the gallbladder.

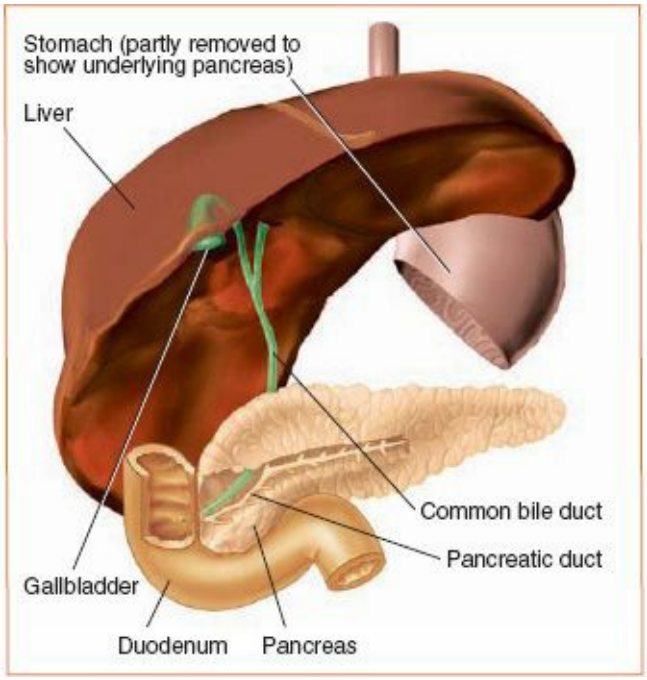


Figure 16-6 The liver, gallbladder, and pancreas.

Bile is a fat emulsifier required for the digestion of fats within the small intestine. As much as 1 L of bile is produced per day, and excess amounts are stored in the gallbladder, a small muscular sac located just inferior to the right lobe of the liver and protruding from under the liver at the level of the ninth rib. The gallbladder is about 10 cm long and can hold up to 50 mL of bile. Water and ions are reabsorbed here, concentrating the bile. Bile enters and exits via the 4-cm-long cystic duct, which connects to the common hepatic duct, or bile duct, allowing transit to the duodenum. The intestinal hormone cholecystokinin (CCK) regulates contraction of the gallbladder. CCK also relaxes the hepatopancreatic sphincter, permitting entry of bile and pancreatic juice into the small intestine via the major duodenal papilla.

Pancreatic juice is a solution of digestive enzymes secreted by the pancreas, a 12.5- to 15-cm-long retroperitoneal organ found in the left upper quadrant. The head of the pancreas is framed by the duodenum, the body runs along the inferior aspect of the stomach, and finally a tail reaches the spleen. The release of pancreatic juice is an exocrine function, but the pancreas also has an equally important endocrine function—the production of insulin and glycogen.

Microscopically, the pancreas contains millions of acini lobules, each consisting of a ring of secretory cells around an intralobular duct through which the secreted pancreatic juice drains into the main pancreatic duct. This duct empties into the common bile duct; thus, it shares a pathway to the duodenum with bile. Zymogen granules within the acinar cells secrete trypsinogen, chymotrypsinogen, procarboxypeptidase, pancreatic amylase, lipases, and nucleases, which mix with water and bicarbonate to make up pancreatic juice. Enzymes such as trypsin within the duodenum convert the various proteins to their active forms, preventing any self-digestion by these powerful enzymes. Collectively, these enzymes account for the majority of digestion that occurs in the small intestine. The bicarbonate balances the acidity of newly arriving chyme from the stomach, and the water serves to further dilute the chyme prior to absorption.

Scattered throughout the acini lobules are islets of Langerhans, cells of the pancreas that secrete a variety of endocrine hormones directly into the bloodstream. Numerous insulin-secreting beta cells work alongside alpha cells, which produce glucagon, and delta cells, which produce somatostatin. Insulin facilitates the absorption of free glucose molecules into cells from the bloodstream. Glucagon stimulates glycogen production in the liver during times of glucose surplus. Somatostatin has numerous regulatory

functions, including the inhibition of overall pancreatic function.

GU System

Urinary System

The many nutrients and ions absorbed into the bloodstream by the tissues of the alimentary canal serve a useful purpose to the body for only a certain period of time. Eventually, as a result of metabolic reactions, the molecules and atoms are either incorporated into useful products or leftover as waste. The renal system provides a path for waste to leave the body **Figure 16-7**. The kidneys continuously filter blood, managing its volume, maintain appropriate balances between acids and bases, and discard toxins and excesses by producing urine. Because the kidneys are extremely vascular, about one fourth of the cardiac output passes through them every minute. The remainder of the urinary system—the ureters, urinary bladder, and urethra—provides for transport and storage of urine during its journey out of the body.

The kidneys are a pair of retroperitoneal organs that stretch from the T12 to the L3 vertebrae. The position of the liver forces the right kidney to be slightly lower. Kidneys weigh an average of 150 g, with a length of 12 cm, a width of 6 cm, and a depth of 3 cm. The adrenal glands, seated on top of the kidneys, are important in endocrine function because they produce catecholamines in response to stress. Although the 12th ribs provide the kidneys some protection, they remain relatively vulnerable to external trauma. Three layers of tissue provide some measure of protection from such trauma:

- The renal fascia, the outermost layer, consists of dense fibrous connective tissue that encloses the kidneys and adrenal glands, securing them to the abdominal walls.
- A layer of adipose tissue directly beneath the renal fascia provides a cushion from sudden movements.
- The renal capsule, which is made up of more fibrous connective tissue, coats the kidney itself.

Internally, the tissues of the kidneys can be divided into three zones: the outermost renal cortex, the medulla, and the innermost pelvis. The functional unit of the kidney, the nephron, lies across the border between the cortex and medulla. The renal pelvis is an open space that allows pooling of urine and drainage into the ureter. **Figure 16-8** and **Figure 16-9** show the nephrons of the kidney and the glomerulus, respectively.

Upon entering the kidney, the renal artery immediately branches off into vessels of decreasing diameter. This culminates in the afferent arteriole that feeds blood into the glomerulus, a meshwork of capillaries contained within the glomerular capsule. The high pressure of the incoming blood forces fluid and solutes through openings in the capillary cell wall, which are then collected by the glomerular capsule and drained into the proximal convoluted tubule. The size of the openings in the capillary walls prevents erythrocyte and large-protein leakage, while podocytes that wrap around the capillaries help filter out macromolecules.

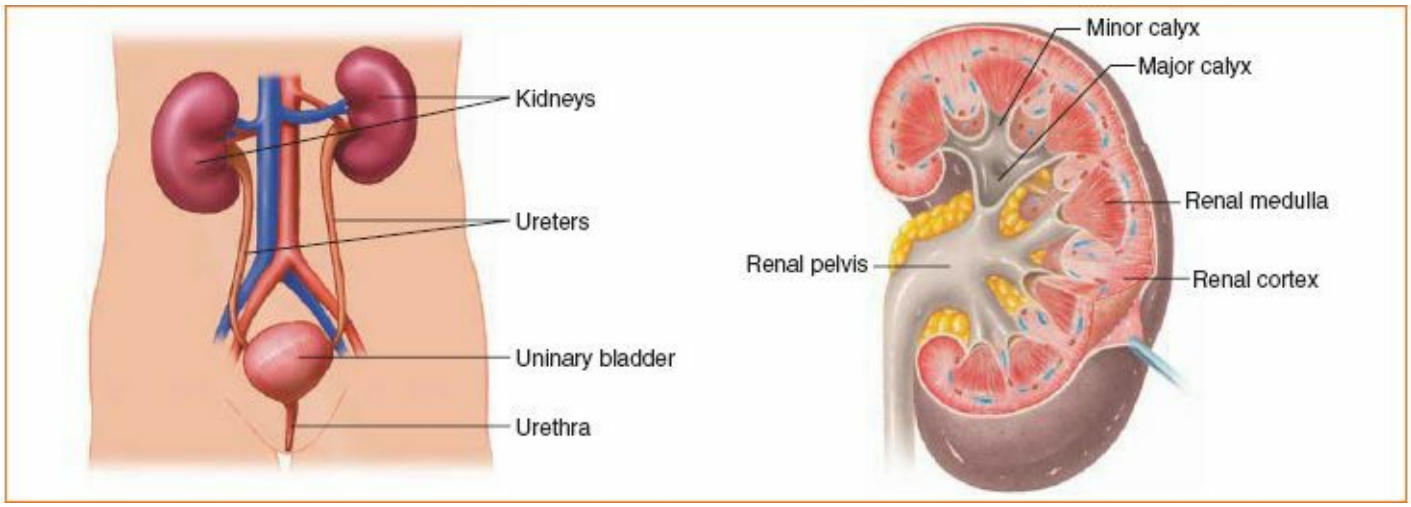


Figure 16-7 Organs of the urinary system.

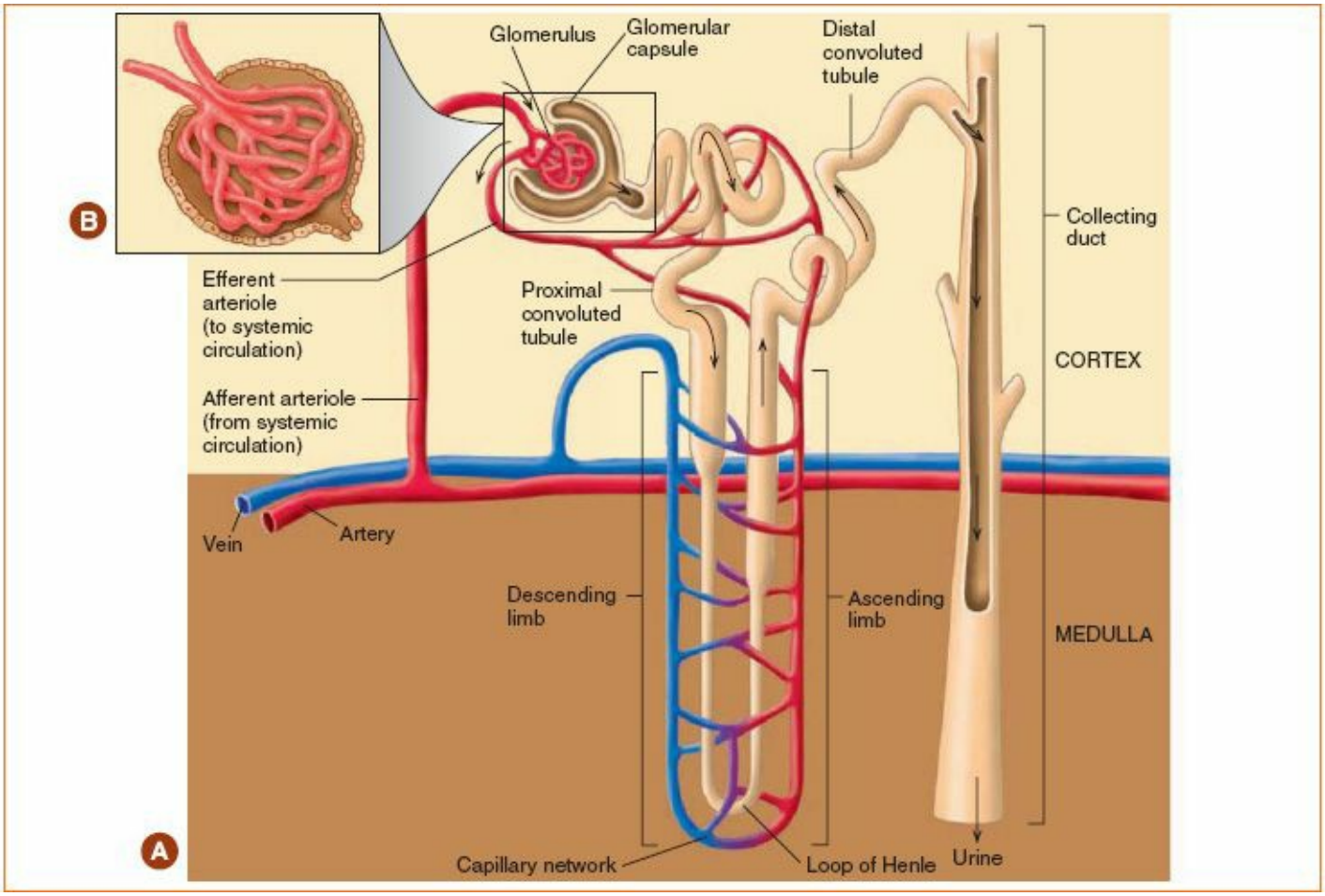


Figure 16-8 **A.** The nephrons of the kidney. Part of the nephron is located in the cortex, and part is located in the medulla. **B.** The glomerulus, close up.

Once filtered, blood exits the glomerulus by way of the efferent arterioles, which then divide into the peritubular capillaries. The proximal convoluted tubule descends into the renal medulla, and subsequently straightens out. After a distance it turns 180° to take a parallel path back to the cortex. This portion, called the loop of Henle, is where the bulk of ion absorption and reabsorption occurs. A series of osmotic gradients between the lumen of the loop of Henle and the medullary tissues result in ion exchange as material moves along this pathway, which plays a significant role in the dilution and concentration of urine.

The loop of Henle becomes the distal convoluted tubule upon its reentry to the cortex, eventually making its way to the collecting duct. The peritubular capillaries are entangled with the proximal and distal convoluted tubules as well as the loop of Henle, reabsorbing water and solutes not meant to be excreted.

The effectiveness of the glomerulus in filtering blood provides a benchmark for the evaluation of overall renal function, termed the **glomerular filtration rate (GFR)**. The normal GFR in an adult is about 125 mL/min. Various pathologies can affect the GFR, and a decrease in the rate will result in a number of metabolic alterations.

A second test frequently employed in assessing glomerular function—and hence renal function—is creatinine clearance. **Creatinine** is the metabolized form of creatine that is eliminated through the urine. The normal elimination rate for men is 95 to 145 mL/min, and the normal rate for women is 75 to 115 mL/min.

Specialized cells known as juxtaglomerular cells are present in the walls of the afferent and efferent arterioles. These cells secrete renin, an enzyme that plays an important role in the activation of the renin–angiotensin–aldosterone system (which helps regulate blood pressure). In between the afferent and efferent arterioles and the distal convoluted tubule is another class of specialized cells, the macula densa. These cells detect chemical changes in the concentration of the filtrate. This region is collectively referred to as the juxtaglomerular apparatus, and is present in every nephron.

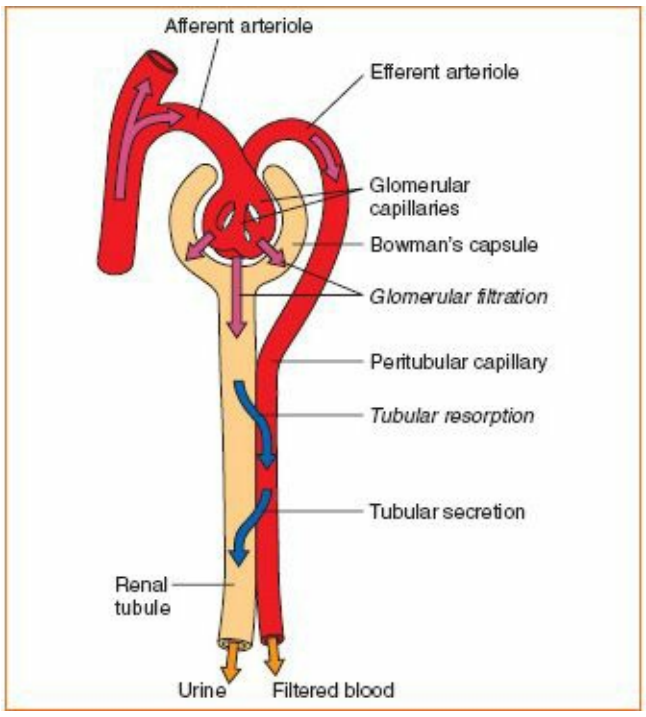


Figure 16-9 The glomerulus of the kidneys. The nephron carries out three blood-filtering processes: glomerular filtration, tubular reabsorption, and tubular secretion.

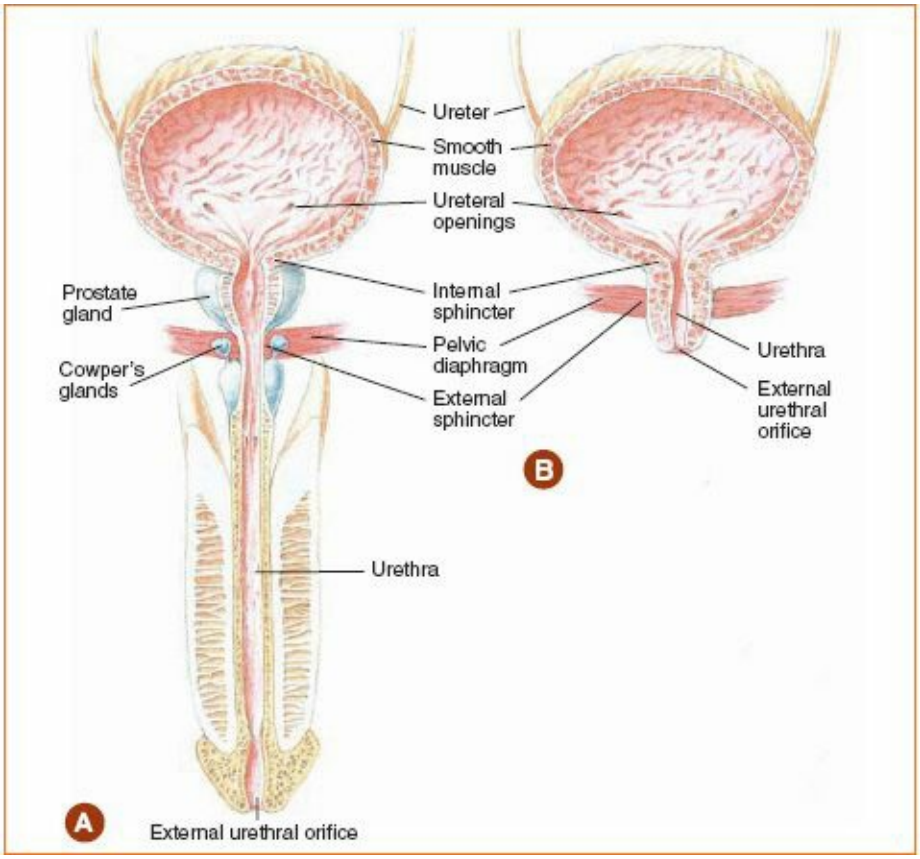


Figure 16-10 Structure of the urinary bladder and urethra. **A.** Male. **B.** Female.

The ureters, a pair of 30-cm long muscular tubes running a course to the urinary bladder, are essentially a continuation of the renal pelvis. Although gravity plays a role in the movement of urine, **peristalsis** is also a significant contributor. The ureters enter the urinary bladder in the trigone region at its base. The urethra also drains the bladder here, so there are actually three connections to the bladder at this point (hence the prefix "tri"). The bladder is a hollow, muscular organ that is located directly posterior to the pubic symphysis. It has a urine storage capacity upwards of 0.5 L and can be palpated in the lower abdomen when distended.

The urethra is another muscular tube, about 20 cm long in men and 3 to 5 cm long in women. It drains the bladder, providing an exit route for urine from the body **Figure 16-10**.

Male Reproductive System

The purpose of the male reproductive system is to generate sperm and provide a means for its delivery to a fertilizable egg in the female partner. The testes are the fundamental male reproductive organ; they consist of a pair of 4- × 2.5-cm oval-shaped spheres, suspended external to the body in a sac of skin called the scrotum. Housing outside the body is required to allow sperm production at 91°F (33°C), about 3° lower than core body temperature. Each testis is covered by two distinct layers of tissue: the outer tunica vaginalis, which folds over itself, and the inner tunica albuginea. The tunica albuginea is a tough fibrous capsule that protects the testis and penetrates it, dividing it into hundreds of lobules. Inside these lobules, sperm are created in the seminiferous tubules. The testes receive their blood supply from the testicular arteries and drain into their complementary veins. These blood vessels branch from the abdominal aorta, and along with auto-nomic nerve fibers travel to and from the testes through the spermatic cord **Figure 16-11**.

Newly made sperm drain into the efferent ductules and then move to the epididymis, where they will mature. When stimulated by smooth-muscle contractions arising from ejaculation, sperm are forced into the ductus deferens (vas deferens), a 45-cm tube that exits the scrotum via the spermatic cord. The ductus

deferens travels anterior to the pubic bone and then posterior to the bladder, finally joining with the seminal vesicle duct to form the ejaculatory duct. The two ejaculatory ducts pass through the prostate gland and terminate in the urethra, which serves as an exit route for both sperm and urine in the male.

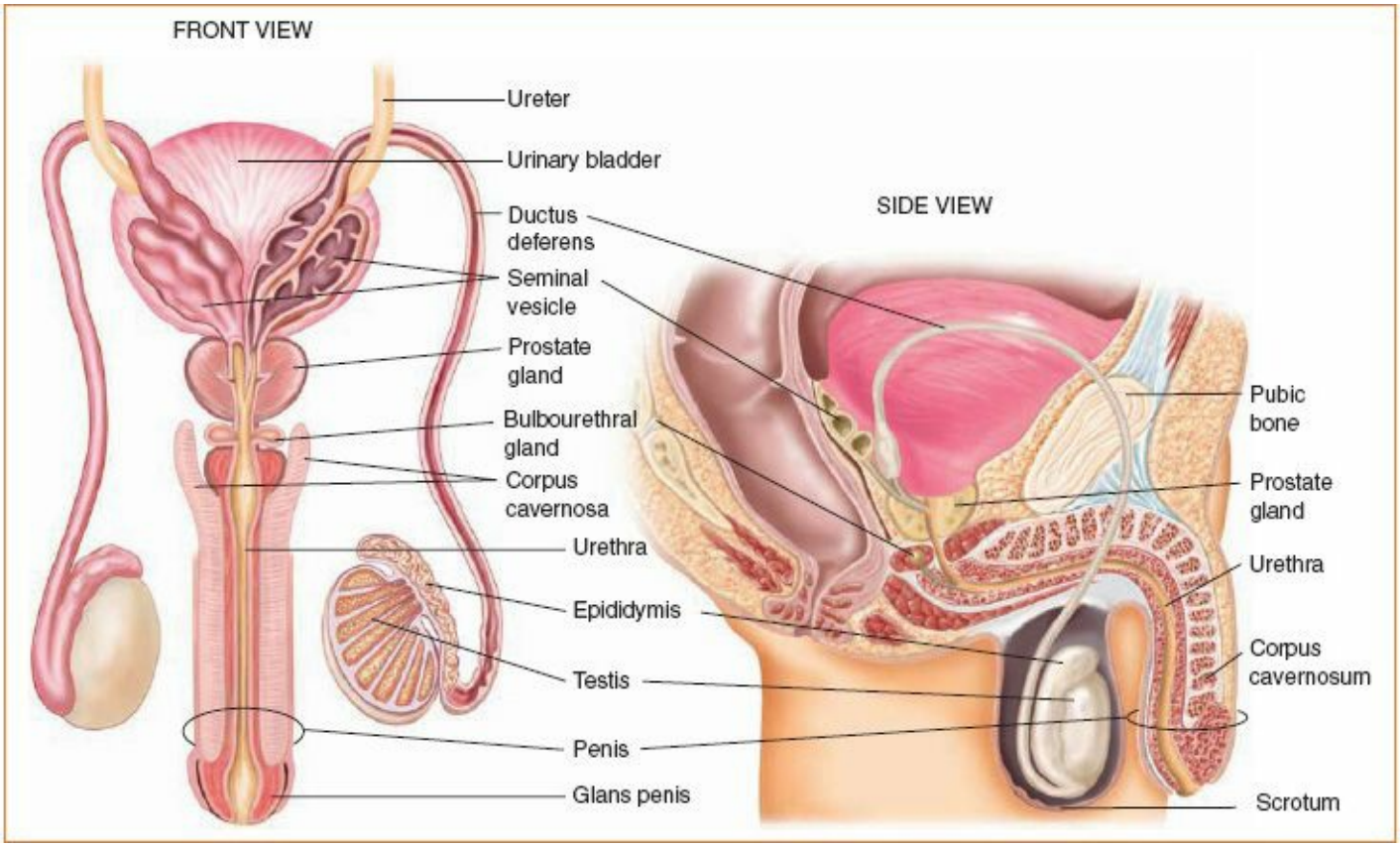


Figure 16-11 The male reproductive system.

The male reproductive system also includes five accessory glands: a pair of seminal vesicles, a pair of bulbourethral glands, and the prostate gland. The seminal vesicles are each about 6 cm long and lie on either side of the posterior bladder. The alkaline secretions of the seminal vesicles both provide the sperm with energy and facilitate their journey by relaxing the cervix and uterus in the female. The bulbourethral glands, which are much smaller than the seminal vesicles, are located just inferior to the prostate gland. The secretions of these glands neutralize any acidic urine encountered in the urethra, thereby protecting the sperm from degradation. The prostate gland surrounds the urethra and ejaculatory ducts and sits immediately inferior to the bladder. Its secretions activate sperm and provide additional nutrients for the sperm's journey.

Taken collectively, the secretions of the seminal vesicles, bulbourethral glands, prostate gland, and sperm are termed semen. Semen is the transport medium by which sperm enters the female's reproductive system, permitting introduction to an egg and potential fertilization. The final leg of the semen's journey in the male sends it through the penis, which the urethra passes through. Three vascular spaces made up of erectile tissue surround the urethra within the penis: the corpus spongiosum and a pair of corpora cavernosa. When the male is sexually stimulated, these three compartments engorge with blood following a parasympathetic release of nitric oxide, a local vasodilator. This process results in penile erection, turning the normally flaccid penis into a rigid structure.

Female Reproductive System

The female reproductive system produces and develops eggs, preparing them for fertilization by a sperm. In addition, the uterus of the female provides a home for a developing embryo during gestation. The main female reproductive organs are the ovaries, which are located on either side of the uterus within the

peritoneum **Figure 16-12**.

Ovarian follicles within the ovary contain oocytes, or immature eggs. During the process of ovulation, an oocyte is dispelled from a vesicular follicle and the ovary each month in a female of childbearing age. The fallopian (uterine) tubes, a pair of 10-cm tubes originating adjacent to the ovaries and terminating in the uterus, take in the expelled oocyte.

Cilia within the fallopian tubes project the oocyte toward the uterus. Situated between the bladder and rectum, the uterus is the cavity in which embryonic development occurs following fertilization. The uterus flexes superiorly around the urinary bladder and is divided into four portions: the superiormost fundus, the central body, the inferior isthmus, and the cervix. The cervix is the narrow point of attachment to the vagina, commonly referred to as the cervical canal. The uterus consists of three thick tissue layers: the outermost perimetrium, the muscular myometrium, and the inner endometrium. A developing embryo will lodge within the endometrium for the duration of gestation. The uterine arteries provide the uterus with its significant blood supply through a network of arcuate arteries. Every month a major portion of the endometrium of the uterus is shed and regenerated through the menstrual (uterine) cycle unless fertilization has occurred.

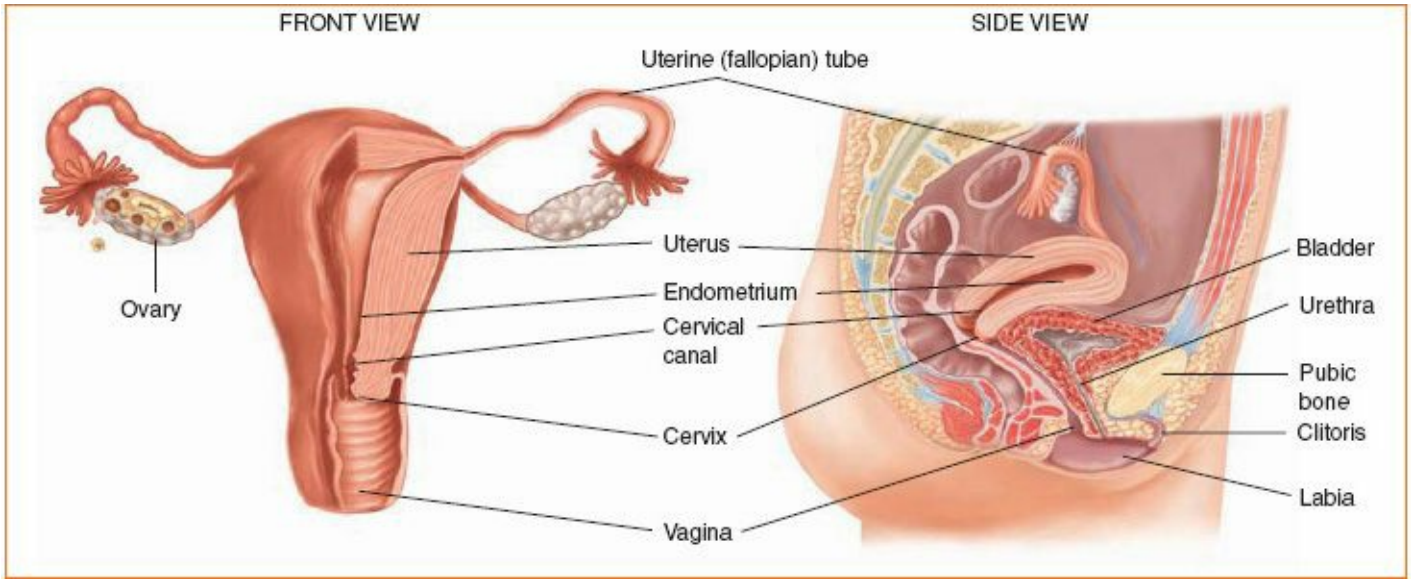


Figure 16-12 The female reproductive system.

The vagina connects the uterus to the external vaginal orifice. During intercourse, the vagina receives semen from the penis, guiding it into the uterus for possible fertilization. The vagina also serves as the birth canal during childbirth. In females, the urethra exits the body just anterior to the vagina, separating the urinary and reproductive tracts.

Special Populations

Injuries and conditions that involve the GI system may be initially missed in pregnant patients. As the gravid uterus grows, the abdominal viscera stretches to accommodate the growing uterus. As this viscera stretches, the pain perceived by the pregnant patient may indeed be referred pain. For example, a large right lower lobe pneumonia may be "felt" by the pregnant woman in the right lower quadrant of the abdomen. Although these patients are typically good historians, where they feel discomfort in the abdominal region may not be the cause or initiation of the pain.

The abdominal/uterine area of a pregnant patient should never be tender or painful. When you are assessing pregnant patients, the uterus and abdominal region should be gently palpated. In addition to

feeling for uterine contractions and fetal movement, the uterus/abdomen should be assessed for tenderness. This tenderness may reflect a uterine/placental or abdominal injury. It may also reflect chorioamniotitis and infection of the amniotic membranes. This infection can lead to profound and overwhelming sepsis in the pregnant patient.

Conditions Affecting the Alimentary Canal

Epidemiology and Pathophysiology

The GI tract contains a multitude of structures operating under harsh conditions. A variety of pathologies are capable of disrupting its normal activities, many of which reach the clinical level only upon breaching the protective layers of intestinal mucosa. Disturbance of the mucosa can expose mesenteric vasculature, nerves, lymph, and inner tissue layers to the extreme changes in pH levels and digestive enzymes of the GI lumen. This exposure accounts for the common symptoms of GI abnormalities: abdominal pain, tenderness, and bleeding. Bleeding is the most common significant finding.

Overall, GI bleeding can be classified into one of two categories based on the location of the bleeding. Upper GI bleeding originates proximal to the **ligament of Treitz**, which supports the junction of the duodenum and the jejunum. Any bleeding distal to this location is referred to as lower GI bleeding.

Upper GI bleeding occurs up to six times more often than lower GI bleeding, but they share a comparable mortality rate of 6% to 10%. Extrinsic factors such as alcohol and tobacco use, diet, and nonsteroidal anti-inflammatory drug (NSAID) use are known to increase the incidence of upper GI bleeding. Recent GI surgery and illness increase the risk as well. In addition, the severity of the bleeding increases steadily with age for those older than 60 years. In patients younger than 60 years without significant comorbidities, the mortality rate is less than 0.1%. The amount of bleeding can vary greatly, from microscopic amounts of blood to massive hemorrhaging.

Although a large percentage of people may develop some degree of lower GI bleeding in their lifetime, it accounts for less than 1% of hospital admissions in the United States. Incidence is closely correlated to age, increasing 200-fold between 30 and 80 years of age. Males are 40% more likely to experience lower GI bleeding than females.

Differentiating between upper and lower GI bleeding can prove difficult, because 80% of GI bleeding presents with rectal bleeding of some kind **Table 16-1**. Endoscopy is the preferred method of locating the bleeding source.

Conditions Affecting the Upper GI Tract

Peptic Ulcers

Peptic ulcers are the leading cause of upper GI bleeding, accounting for more than 50% of acute cases **Figure 16-13**. They are characterized by an erosion of the mucosal lining of the GI tract in either the stomach (gastric ulcer) or the duodenum (duodenal ulcer). Once that defensive lining is disrupted by 1 to 3 mm or more, exposure to the acids and pepsin in the pylorus and duodenum can cause ulceration. An ulceration that is deep enough can penetrate a blood vessel, leading to major or minor bleeding. Continued ulceration can result in a perforation of the gastric or duodenal wall, causing peritonitis. Finally, scar tissue from past ulcers can obstruct the GI tract. Peptic ulcers are common in the United States, with a lifetime prevalence of 10%. Despite this, they only account for about 0.1% of US hospital admissions. Although psychological stress is commonly indicated as a contributing factor to ulcer formation, there is a significant lack of evidence to support this theory.

TABLE 16-1 Causes of Upper and Lower GI Bleeding

Type of Bleeding	Cause
Upper GI bleeding	Peptic ulcers Gastritis Esophageal varices Mallory-Weiss syndrome
Lower GI bleeding	Diverticulitis Angiodysplasia Inflammatory bowel diseases (ulcerative colitis, Crohn's disease, cancer)

Abbreviation: GI, gastrointestinal.

TABLE 16-2 Causes of Peptic Ulcers

Helicobacter pylori infection
Nonsteroidal anti-inflammatory drugs
Alcohol use
Tobacco use
Stress-related erosive syndrome
Curling's syndrome
Cushing's syndrome
Zollinger-Ellison syndrome
Gastritis

The bacterium ***Helicobacter pylori*** is the most frequent cause of peptic ulcers, being involved in 70% to 80% of cases. *H pylori* are highly motile spirilla bacteria that colonize the mucosa of the stomach and duodenum. Once inside the mucous layer, they remain protected from gastric acid and begin to produce toxins that cause local tissue inflammation. The inflammation leads to tissue damage and erosion, which results from the tissue's increased susceptibility to the acids and digestive enzymes in the lumen. NSAIDs such as aspirin inhibit the defenses of the mucosa against irritation by reducing production of prostaglandins, the hormones that mediate the inflammatory response in tissues. When their production diminishes, mucosal damage from acidic gastric juice is not corrected promptly and erosion ensues. Use of alcohol and tobacco increases the acidity of the gastric juice, further irritating the lining.

Other causes of peptic ulcers are listed in Table 16-2. **Stress-related erosive syndrome** is a frequent cause of peptic ulcers in critically ill patients, with an incidence of nearly 100%. Poor mucosal perfusion as a result of multiple comorbidities, trauma, or burns also reduces the efficiency of the mucous barrier. This general decrease in resistance to gastric juice allows the formation of numerous shallow ulcers throughout the esophagus, stomach, and duodenum. Ulcers associated with severe burns are termed Curling's ulcers, and those accompanying episodes of increased intracranial pressure are Cushing's ulcers. It is thought that the increased intracranial pressure causes excess vagal stimulation, which results in a marked increase in acidic gastric secretions. Less common causes of ulcers include hypersecretion of acid into the GI lumen, such as by a tumor, as seen in **Zollinger-Ellison syndrome**.

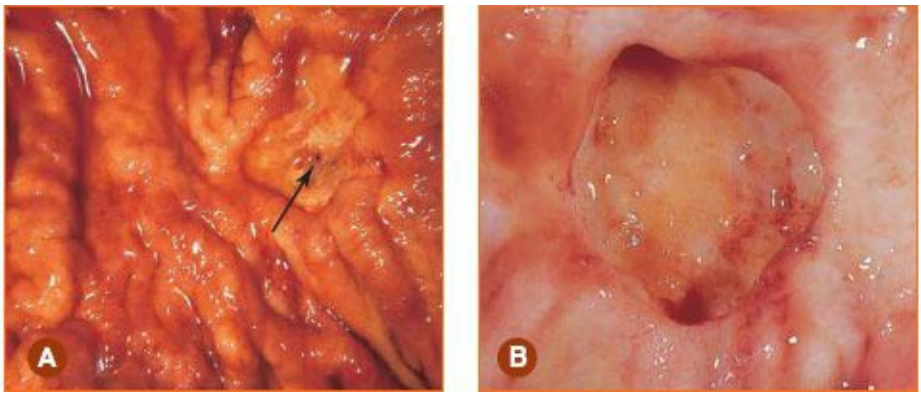


Figure 16-13 Peptic ulcers. **A.** Gastric ulcer, which eroded a blood vessel in the base of the ulcer (arrow) and bled profusely. **B.** Large chronic duodenal ulcer.

Gastritis

Inflammation of the gastric mucosa, termed **gastritis**, is a common precursor to upper GI bleeding. Causes of gastric inflammation vary widely and can be broadly separated into acute or chronic pathologies. Common causes of gastritis are listed in **Table 16-3**.

As previously mentioned, the mucosal lining of the esophagus, stomach, and duodenum forms an important barrier between gastric contents and internal tissues of the body. A dynamic equilibrium is established across this barrier between the aggressive and defensive components. Hydrochloric acid and pepsin in the gastric juice assault the mucosa, whereas bicarbonate, prostaglandins, and rapid cell renewal protect it. When this equilibrium is disturbed, inflammation of the epithelial cells occurs as a result of the ensuing immune system response. An inflamed lining is unable to provide its typical defensive mechanism and becomes susceptible to erosion into the well-perfused submucosal layer. As expected, gastritis is a common cause of peptic ulcers. Once the blood vessels in submucosa are breached, GI bleeding begins.

TABLE 16-3 Causes of Gastritis
Corrosive chemical ingestion
Alcohol abuse
Helicobacter pylori infection
Autoimmune disorders (type A gastritis)
Reactive gastritis
Radiation exposure

Acute gastritis is characterized by a rapid onset of mucosal inflammation frequently accompanied by mild to severe upper GI bleeding **Figure 16-14**. Ingestion of corrosive chemicals, for example, can burn the epithelial lining of the GI tract within a short time period. Depending on the chemical, these burns can cause significant inflammation and subsequent necrosis of tissues with varying degrees of hemorrhage. Chemical burns commonly affect external perioral tissues, the oral cavity, and the tongue as well. Many household cleaners such as bleach are potent enough to trigger this process.

Many cases of acute gastritis are an exacerbation of a chronic GI condition. Alcohol-induced gastritis, commonly referred to as hemorrhagic gastritis, presents as numerous lesions along the gastric

mucosa. Extensive tissue damage is found in up to 20% of chronic alcoholics. Minor bleeding occurs frequently and often goes unnoticed in these patients, but an episode of severe hemorrhage can occur suddenly after even a small intake of alcohol. *H pylori* cause acute gastritis during the primary period of infection. The initial immune system response to the invading bacteria triggers an inflammation of the mucosal tissues, which is self-limiting and resolves within 11 to 14 days. Occasionally, the bacteria are fully eliminated from the body following the immune defense. More typically, however, chronic gastritis develops.

Autoimmune disorders targeting the GI system can cause chronic inflammation of mucosal lining and also inhibit certain metabolic pathways crucial to digestion. In **type A gastritis**, the intrinsic factor secreted by mucosal parietal cells is attacked by autoantibodies. The inhibition of intrinsic factor results in a decreased absorption of vitamin B_{12}, resulting in pernicious anemia. A more common form of chronic gastritis is **type B gastritis**, or antral-predominant. In this case, inflammation of varying severity is caused by *H pylori*, with an incidence approaching 100% in the elderly.

Reactive gastritis is a chronic mucosal edema that results from recurring contact of the mucosa with antagonistic substances such as bile, pancreatic juice, or NSAIDs. Also commonly referred to as reflux or chemical gastritis, reactive gastritis is most commonly seen in patients following gastric surgery. Bile or pancreatic juice refluxed from the duodenum into the stomach will damage the surface of epithelial cells and cause a decreased mucosal blood flow. Diminished blood flow inhibits immune response and, as a result, inflammation in this situation is minor to nonexistent.

Radiation exposure causes varying forms of gastritis depending on the amount of radiation involved. Substantial tissue necrosis occurs in the mucosa during the first few weeks following exposure. An accompanying decrease in blood perfusion to the area will inhibit digestive processes. Scar tissue formation is common following cell replacement. Ulcerations may occur as well, either acutely or chronically.

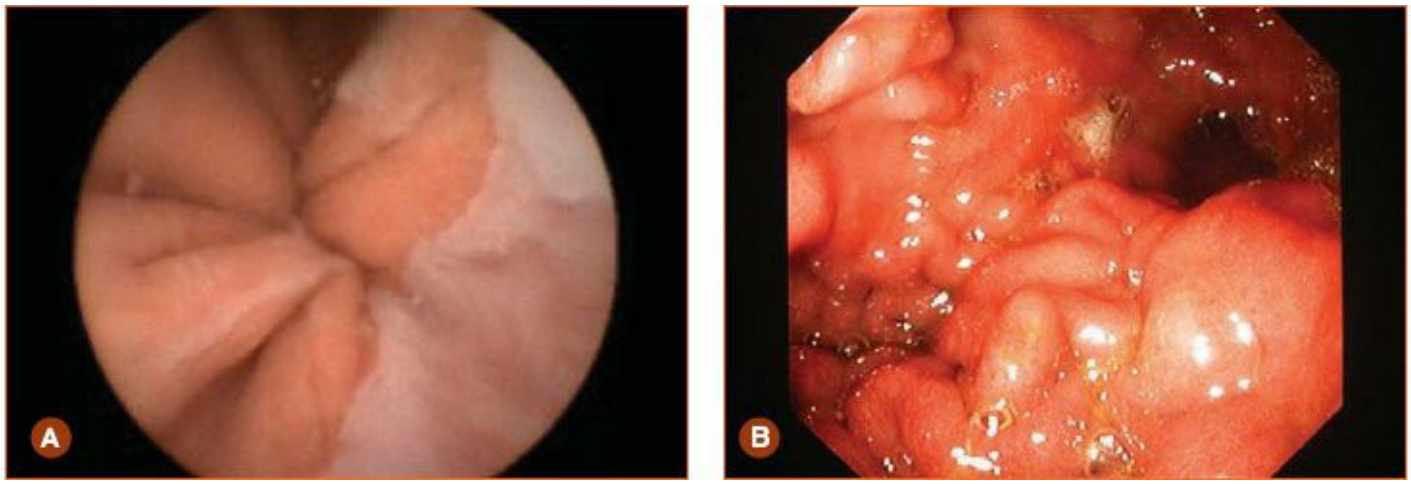


Figure 16-14 **A.** Normal stomach. **B.** Gastritis.

Esophageal Varices

Just as erosion of mucosal lining into a blood vessel will initiate GI bleeding, so will the reverse process —swelling of esophageal veins into the lumen of the esophagus, called an **esophageal varix** **Figure 16-15**. These exposed veins are susceptible to sudden rupture, resulting in severe hemorrhage; this phenomenon accounts for about 14% of upper GI bleeding. Mortality immediately following a variceal rupture ranges from 35% to 40%. Unlike other causes of GI hemorrhage, in which active bleeding typically stops without intervention in 80% to 90% of cases, esophageal varices spontaneously stop bleeding only 50% of the time. If left untreated, mortality rises to 70% after 1 year.

Swelling of the esophageal veins is a result of increased venous pressure in the hepatic portal system. When **portal hypertension** occurs, venous blood will use alternate pathways for its return to the vena cava via the **azygos system**. The azygos system also serves to drain a portion of the esophageal venous blood, and overuse of this system causes pooling in these veins. The lower portion of the esophagus drains blood into the left gastric vein, which is adversely affected by portal hypertension as well. Increases as small as 5 mm Hg in portal venous pressure are capable of producing varices, with the risk of rupture growing exponentially as pressure continues to rise. Although varices can occur anywhere along the esophagus, stomach, and duodenum, ruptures leading to GI hemorrhage most frequently occur at the gastroesophageal junction.

Figure 16-15 Mucosal surface of the esophagus illustrating varices, which appear as tortuous elevations of the mucosa (*arrows*).

Portal hypertension results from liver damage, usually cirrhosis. In **cirrhosis**, buildup of fatty acids, along with chronic destruction of liver tissue and fibrosis, obstructs blood flow. In the United States, liver cirrhosis is primarily caused by chronic alcohol use or hepatitis C, which together account for more than 60% of all diagnoses.

Mallory-Weiss Syndrome

Mallory-Weiss syndrome involves a longitudinal laceration of the esophageal mucosa **Figure 16-16**, typically in the distal esophagus at the gastroesophageal junction. The tear develops as a result of repeated significant changes in local pressure, such as those seen in repeated vomiting. GI bleeding occurs once the tear reaches the vascular submucosa. Mallory-Weiss tears are implicated in about 5% of upper GI bleeds, with a mortality of less than 10%. In as many as 90% of cases, bleeding stops spontaneously. Cofactors increasing the likelihood of a tear include hiatal hernias, chronic alcohol use, and increasing age.

Conditions Affecting the Lower GI Tract

Lower GI bleeding is frequently the result of an exacerbation of a chronic condition. This type of bleeding, which is characterized by bleeding distal to the ligament of Treitz, can occur in the jejunum, ileum, large intestine, rectum, and anus. A number of conditions can cause bleeding. The severity of

bleeding can vary greatly.

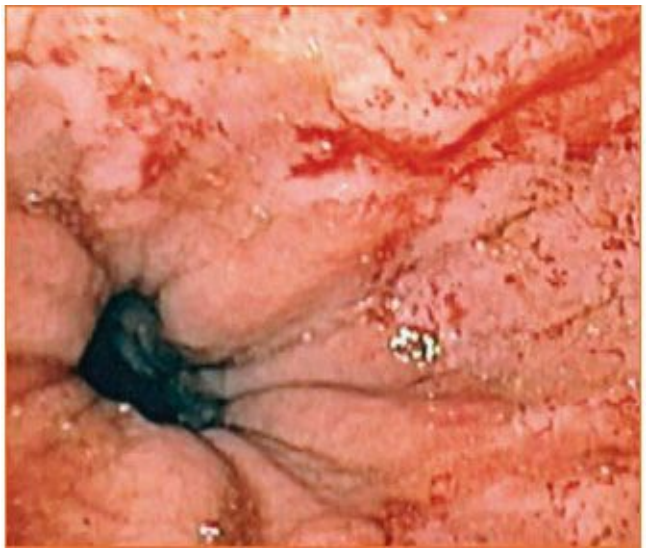


Figure 16-16 Mallory-Weiss syndrome.

Diverticulosis and Diverticulitis

Diverticulosis is a common disease of the lower GI tract in the elderly. Small pouches called **diverticula** outcrop from the mucosal lining of the large intestine, typically in the descending colon and sigmoid colon **Figure 16-17** and **Figure 16-18**. These outcroppings are a consequence of strong Haustral muscle contractions, which push tissue against a mucosa that weakens with age. The diverticula are generally painless and can be found in half of all US men older than 60 years.

Diverticulitis, an inflammation of the diverticula, occurs in as many as 25% of diverticulosis cases. It is frequently a result of infection from invading intestinal bacteria. Inflamed diverticula are painful, and 3% to 5% of them bleed.

Diverticulosis is the most common cause of lower GI bleeding, being responsible for 42% to 55% of cases. Bleeding can be heavy because arterial blood vessels are incorporated into developing diverticula. Certain risk factors are known to increase the incidence of diverticulosis, including a low-fiber diet, chronic constipation, and NSAID use.

Angiodysplasia

Angiodysplasia is a malformation of submucosal blood vessels in the GI tract **Figure 16-19**. Although it can occur anywhere in the alimentary canal, more than three fourths of all cases present in the cecum and ascending colon. These thin-walled, winding vessels are highly susceptible to rupture. Bleeding as a result of angiodysplasia is the second most common cause of lower GI bleeding, accounting for 20% to 30% of cases.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is a collective term covering two idiopathic colon pathologies that are together responsible for about 10% of the cases of lower GI bleeding: ulcerative colitis and Crohn's disease **Figure 16-20**. Common causes of inflamed colon or colitis are listed in **Table 16-4**.

Ulcerative colitis is an inflammation of the rectal mucosal and submucosal tissues. In this condition, ulcers that occasionally bleed form throughout the colon. Inflammation spreads into the colon in 75% of cases, a situation termed pancolitis. The incidence of ulcerative colitis is highest in whites aged 20 to 25 years.

Crohn's disease is a less organized inflammation of the GI tract in which all layers of the mucosa

may be affected. This inflammation is not contiguous, as is seen in ulcerative colitis, but rather results in scattered ulcerations and fibroses throughout the large and small intestines. Minor bleeding occasionally occurs from these ulcers as well as from small tears that may develop in the mucosa. Only about 1% of patients diagnosed with Crohn's disease experience significant GI bleeding. The disease is thought to be hereditary and is found most commonly in whites.

TABLE 16-4 Causes of an Inflamed Colon or Colitis
Ulcerative colitis
Crohn's disease
Bacterial infection
Viral infection
Radiation colitis

As ulcerative colitis and Crohn's disease move into chronic states, scar tissue formation over ulcerations can result in a thickening of the mucosa and partial bowel obstructions. Because both diseases share similar symptoms, 20% of cases are diagnosed as indeterminate colitis.

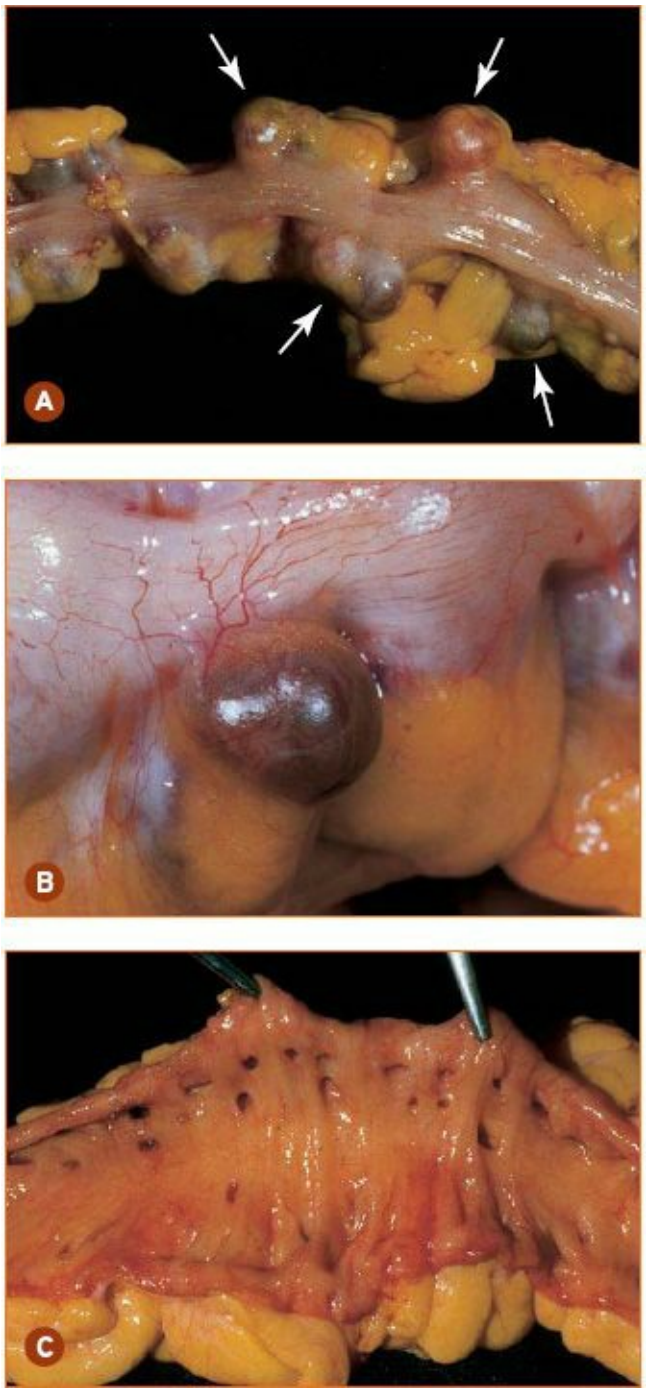


Figure 16-17 Diverticulosis of the colon. **A.** Exterior of the colon illustrating several diverticula projecting through the wall of the colon (arrows). **B.** Closer view of the diverticulum. **C.** Interior of the colon, illustrating openings of multiple diverticula. Several of the openings are well demonstrated in the mucosa just below the clamps.

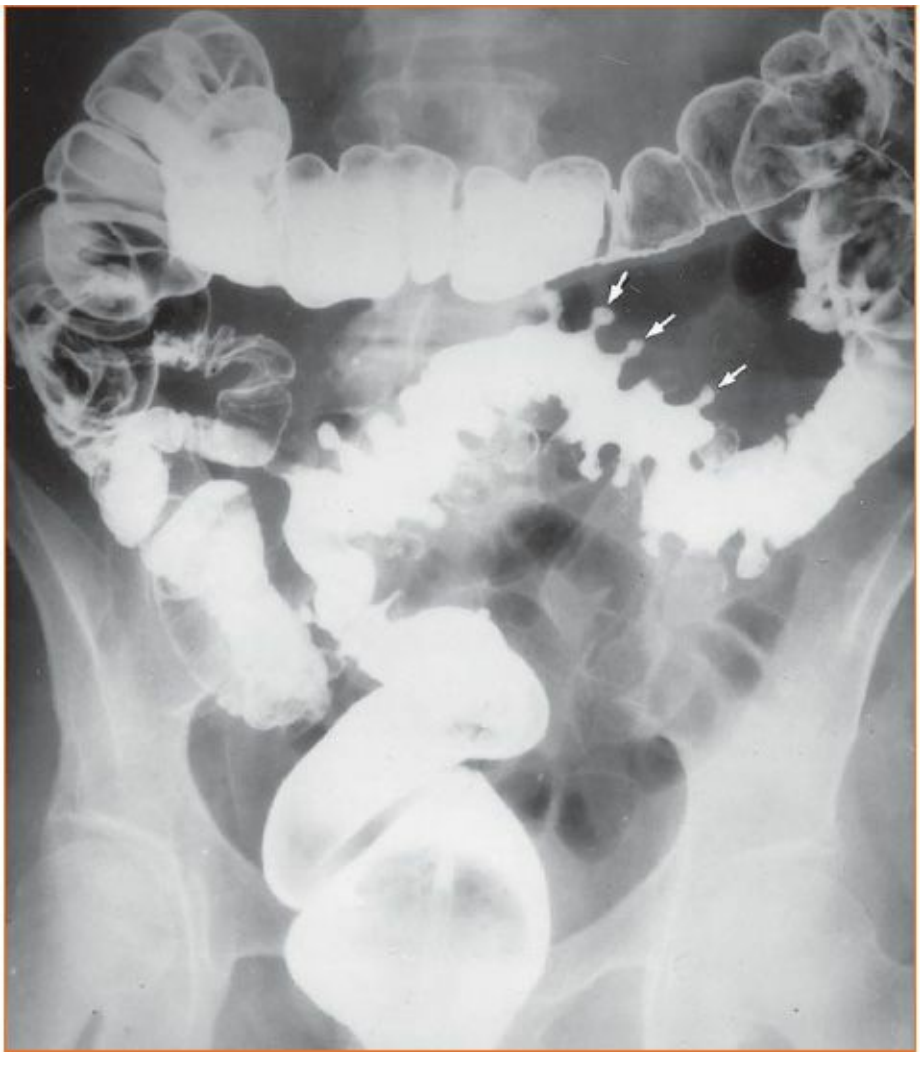


Figure 16-18 Diverticula of the colon demonstrated by injection of barium contrast material into the colon (barium enema). Diverticula filled with contrast material appear as projections from the mucosa (arrows).

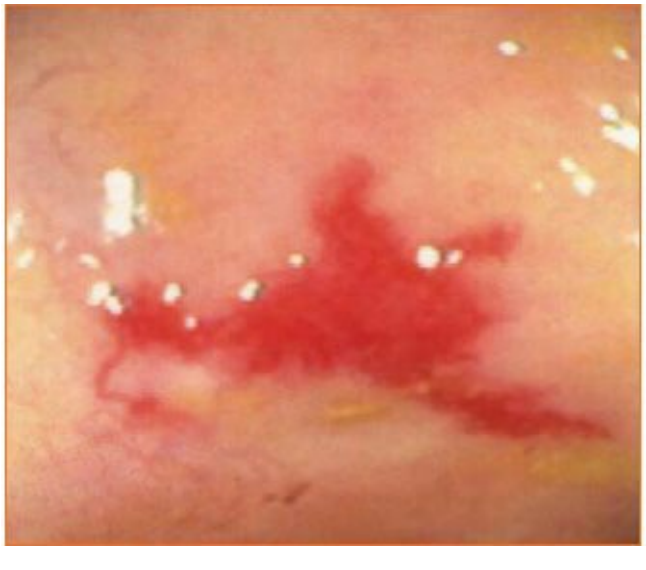


Figure 16-19 Angiodysplasia.

A variety of infectious diseases of the colon are also commonly referred to as IBD. These cases are self-limiting and rarely result in significant bleeding, including bacterial infections by *Shigella, Salmonella*, and *Campylobacter*. Viral causes include the Norwalk-type virus and cytomegalovirus in

immunosuppressed patients. Patients on continued antibiotic therapy may experience growth of *Clostridium difficile*, which can cause permanent mucosal damage.

Radiation colitis occurs in 75% of patients who have received radiation doses of 40 Gy or more to the abdomen or pelvis. Chronic mucosal thickening can result, as well as ulcerations throughout the colon. Although the major complication of this condition is abdominal pain and diarrhea, anemia and lower GI bleeding can occur as well.

Assessment

Signs and Symptoms

The signs and symptoms of patients who experience GI bleeding vary depending on the origin and severity of the bleeding. Slow, chronic bleeding can result in the presence of **melena**, black tarry stool containing partially digested blood. If the bleeding in either the upper or lower GI tract is minimal enough, blood in the stool may be detectable only by laboratory testing. **Table 9-8** describes the various signs and symptoms associated with the stages of blood loss. Diffuse abdominal pain accompanies even minor bleeding, and will be described as a chronic stomachache or nonspecific burning sensation. The presence of even small amounts of blood in the stomach can cause irritation and subsequent vomiting. Emesis containing partially digested blood, known as **hematemesis**, has a coffee-ground appearance. Many patients experience weight loss, anemia, and malnutrition as a result of poor nutrient absorption over damaged mucosa.

Complaints of frequent dizziness and syncope are common in patients with GI bleeding. Fever as a result of infection can occur in patients who experience GI bleeding as well. Therefore, temperature should be assessed in patients with GI dysfunction or bleeding. Often it is necessary to obtain a rectal temperature because of intubation. In such cases, gently insert the thermometer into the rectum without causing further pain to the patient. It is important to be aware that rectal temperatures are normally higher than oral ones, with a normal reading of around 99.5°F

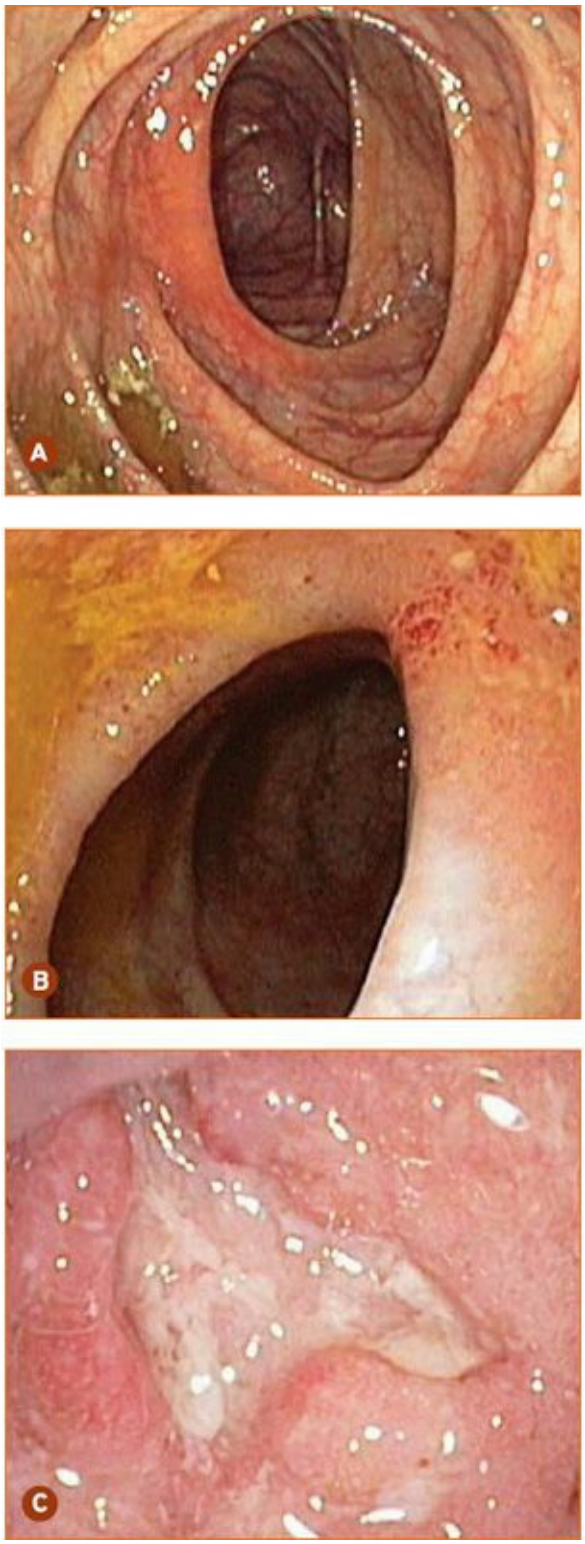


Figure 16-20 **A.** Normal colon. **B.** Ulcerative colitis. **C.** Crohn's disease.

Diarrhea is the most common symptom associated with lower GI disorders, as a result of reduced water absorption efficiency associated with damaged or infected mucosa. Dehydration frequently results from this problem, as do electrolyte imbalances that can trigger cardiac arrhythmias.

Significant GI bleeding is usually acute and can present with hemodynamic instability and shock. Vomiting large amounts of bright red blood can signify acute bleeding in the upper GI tract—for example, from ruptured varices, an ulcerated submucosal blood vessel, or Mallory-Weiss syndrome. **Hematochezia**, the presence of bright red blood in the stool, indicates active lower GI bleeding. When coupled with diarrhea, significant blood and fluid loss can result. Hypotension may or may not be immediately apparent, and testing for orthostatic changes should be performed. Other signs of shock such

as tachycardia, diaphoresis, and altered mental status accompany blood loss. When paired with a decreased level of consciousness, vomiting can result in airway compromise.

When assessing for rectal bleeding, the CCTP should be aware that external sources of bleeding may exist in the area of the anus, such as **pressure ulcers**. Pressure ulcers arise from prolonged pressure on body tissues, and are commonly seen in patients confined to a bed. As many as 25% of nursing home patients are observed to have pressure ulcers, with their treatment incurring considerable costs on the health care system. Pressure ulcers are classified on a scale of stages 1 through 4 **Figure 16-21**. Stage 1 ulcers are reversible, characterized by warmth and some blanching; stage 4 ulcers have full-thickness tissue necrosis, often with muscle and bone involvement. If ulcers are present, the CCTP should take care to avoid further pressure on the area while bandaging any exposed bleeding tissue. It is unlikely that these ulcers would produce life-threatening blood loss, but they do cause severe pain and infection can also become severe.

Some abdominal diseases produce distinctive pain in specific locations, aiding in their diagnosis. For example, peptic ulcers cause a burning epigastric pain that peaks 1 to 3 hours after eating and is usually relieved by antacids. Heartburn pain frequently accompanies peptic ulcers. Gastric ulcer pain is exacerbated with a full stomach, whereas duodenal ulcers are more painful at night, when the stomach is empty. Esophageal varices present initially with painless bleeding that develops into a burning pain. Diverticulitis typically presents with sharp intermittent pain to the lower left abdominal quadrant, commonly referred to as a left-sided appendicitis. Gastritis and inflammatory bowel disease generally cause vague abdominal discomfort, making it difficult to differentiate between them based on signs and symptoms alone. The patient's medical history and presence of risk factors for certain diseases should always be considered during the assessment to aid the prehospital provider in selecting the appropriate treatment.

Signs and Symptoms

GI Bleeding

- Diffuse abdominal pain
- Chronic stomachache
- Nonspecific burning sensation
- Vomiting, often with blood present
- Hematemesis
- Melena
- Hematochezia
- Weight loss
- Anemia
- Malnutrition
- Dizziness
- Syncope
- Fever
- Diarrhea
- Dehydration
- Hypotension

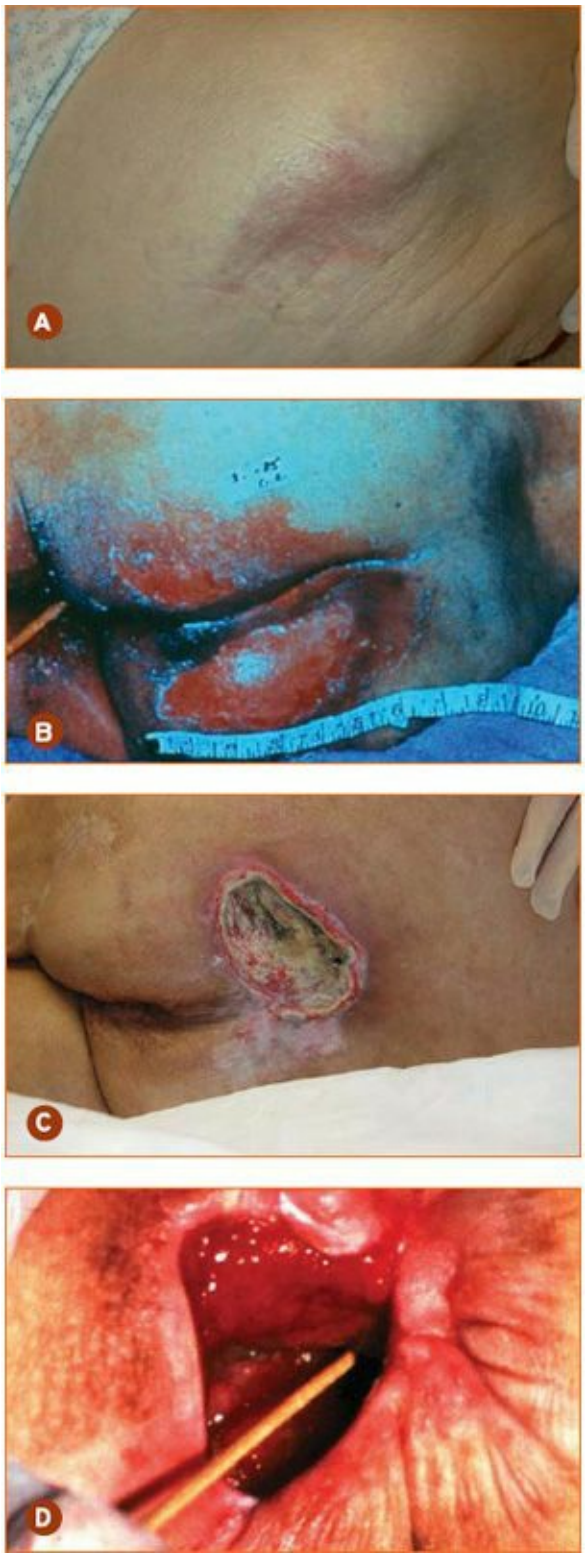


Figure 16-21 A pressure ulcer develops when pressure decreases blood supply and thus oxygenation to an area of tissue. **A.** Stage 1. **B.** Stage 2. **C.** Stage 3. **D.** Stage 4.

Differential Diagnosis

GI Bleeding

- Peptic ulcer disease
- Gastritis
- Esophageal varices

- Zollinger-Ellison syndrome
- Abdominal aortic aneurysm
- Gastric tumor
- Esophageal cancer
- Cholecystitis
- Cirrhosis
- Esophagitis
- Syncope
- Angiodysplasia
- Disseminated intravascular coagulation
- Celiac sprue (syndrome in which intestines lose their ability to absorb)
- Irritable bowel syndrome
- Hemorrhoids
- Antibiotic use
- Infectious diarrheal processes

Transport Management

GI Bleeding

- Provide airway control: suction and intubation.
- Provide ventilatory support with oxygen.
- Designate nothing by mouth.
- Place two large-bore IV tubes.
- Provide fluid resuscitation.
- Draw blood samples, provide blood infusion.
- Continuously monitor blood pressure.
- Continuously monitor pulse oximetry.
- Continuously monitor the electrocardiogram (ECG).
- Obtain lab values.
- Place nasogastric (NG) or orogastric (OG) tube.

Laboratory Data

Laboratory testing and monitoring are essential in the diagnosis and management of chronic GI bleeding **Table 16-5**. Hemoglobin and hematocrit values appear normal during the first 24 to 48 hours of bleeding owing to an equivalent loss of plasma and red blood cells. Eventually the hematocrit value decreases as interstitial fluid shifts to blood vessels in the body's attempt to maintain blood pressure. Hematocrit values also fall as blood is diluted with crystalloid fluids during resuscitation. Fluctuations in the hematocrit value over time indicate active bleeding.

A coagulation profile can determine the existence of clotting deficiencies requiring specific treatment. Prolonged prothrombin time (PT) and activated partial thromboplastin time (PTT) are suggestive of liver disease, and a liver function test should be performed. Thrombocytopenia may be chronic or a result of platelet loss from hemorrhage. Infusion of fresh frozen plasma and platelets may be

required in these patients.

Blood urea nitrogen (BUN) levels increase following upper GI bleeding owing to digestion and absorption of blood proteins in the large intestine. A decreased GFR due to hypoperfusion of the kidneys secondary to hypovolemia will exacerbate this increase. An elevated BUN-to-creatinine ratio rules out kidney failure as the cause of the increased BUN level. A ratio greater than 36 is highly suggestive of upper GI bleeding.

An adrenergic response to GI bleeding results in hyperglycemia, whereas the insult to mucosal tissues stimulates an increase in the white blood cell (WBC) count. Serum electrolyte disturbances can occur as well, including hypernatremia and hyperchloremia from fluid resuscitation and hypokalemia from excessive vomiting. The CCTP needs to review the most recent labs in the chart as part of his or her assessment before transfer.

Imaging

The most effective method of localizing the origin of GI bleeding is to search the mucosa. **Endoscopy**—specifically, esopha-gogastroduodenoscopy—is the method of choice because it is capable of visualizing more than 90% of the upper GI tract with a 90% to 95% diagnosis rate. Endoscopes are equipped with therapeutic tools that allow immediate treatment of an actively bleeding site upon discovery. Hemostatic methods available include the use of heater and bipolar electrocautery probes to cauterize a bleeding vessel and injection sclerotherapy (injection of small doses of either epinephrine or clotting factors). Gastric lavage is required prior to endoscopy to facilitate viewing of the internal structures. Complications from endoscopy are relatively rare, with an incidence of less than 1%.

Visualization of lower GI mucosa is possible via **colonoscopy**, although the procedure is more complicated than endoscopy. To view the mucosa effectively, bowel preparation methods are required to clear the colon. Colonoscopy allows visualization of the entire rectum and large intestine, up to the ileocecal junction.

TABLE 16-5 Normal vs Abnormal Laboratory Values

Test	Normal	Abnormal
Hemoglobin	14-17.5 g/dL	< 14 g/dL
Hematocrit	41%-50%	< 41%
Prothrombin time	10-13 s	Varies
Partial thromboplastin time	< 40 s	Varies
Blood urea nitrogen (BUN)	8-23 mg/dL	> 23 mg/dL
Creatine, serum	0.6-1.2 mg/dL	Varies (unchanged or small rise)
BUN:creatine ratio	10:1	> 10:1
Glomerular filtration rate	125 mL/min	< 125 mL/min
Blood glucose	70–110 mg/dL	> 110 mg/dL (early)
White blood cells (leukocytes)	4,500-11,000/μL	Varies, elevation indicates infection

Angiography is an alternative to endoscopy or colonoscopy when significant active bleeding is present. When this imaging technique is used, dye injected into blood vessels will leak into the lumen of

the GI tract at the site of the bleeding. A bleeding rate of at least 0.5 mL/min is required for effective assessment via this means. Angiography is more frequently used to diagnose lower GI bleeding because of the lower success rate of colonoscopy as opposed to endoscopy in such cases.

Technetium-99m–labeled red blood cell **scintigraphy** can detect GI bleeding rates as low as 0.1 mL/min, although with less specificity than angiography. In this technique, red blood cells attached to radioisotopes are injected into the bloodstream, and subsequent scanning of the abdomen detects their presence in the GI lumen. The tagged cells remain in the circulation for as long as 48 hours, allowing repeat studies to be performed if necessary. Successful detection of GI bleeding occurs as often as 93% of the time, although localization is accomplished with a frequency of only 67% to 97%.

Various noninvasive imaging techniques are available as well and form a cornerstone of acute abdominal assessment. These techniques include abdominal ultrasounds, computed tomography (CT) of the abdomen, and magnetic resonance imaging (MRI). **Figure 16-22** shows a normal abdominal CT scan.

Management

The fundamentals of patient care must be ensured before any specific treatment is attempted. In particular, it is important to control and maintain the patient's airway. During acute GI bleeding, airway compromise occurs primarily by either of two ways: physical obstruction or mechanical obstruction. Physical obstruction of the airway by blood and/or vomit must be cleared with suctioning. Mechanical obstruction of the airway by the tongue or head positioning as a result of a decreased level of consciousness, secondary to hypovolemic shock, must be managed by airway adjuncts and intubations. Rapid intubations are indicated in patients with a high risk for aspiration, such as patients who are lethargic or have an altered mental status.

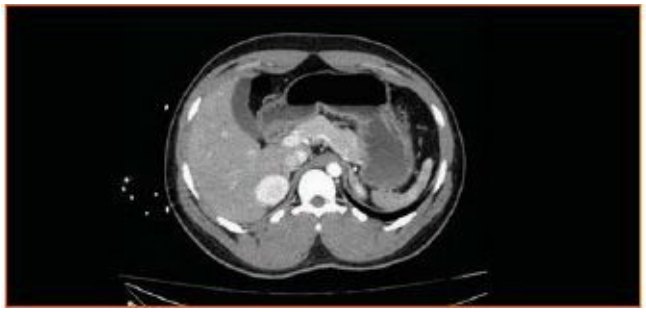


Figure 16-22 A normal abdominal computed tomographic scan.

Supplemental oxygen should be administered via non-rebreathing mask in the alert, conscious patient. Ventilations should be assisted as necessary with a bag-mask device and high-flow oxygen.

Any patient with acute GI bleeding should be designated NPO (nothing by mouth) because of the likelihood of undergoing endoscopy and/or surgery shortly after hospital admission. Two large-bore IV catheters (14 to 16 gauge) should be placed immediately. Fluid resuscitation should begin immediately in any patient who exhibits signs of shock, to consist of an initial bolus of 20 mL/kg of isotonic crystalloid solution.

Blood samples should be drawn concurrently with IV placement to determine a type and crossmatch, complete blood count, electrolyte levels, and PT and PTT values. Blood loss estimated at greater than 1,500 mL requires administration of packed red blood cells along with fluid replacement. Platelet or fresh-frozen plasma infusion may be indicated based on lab findings as well as volume of packed red blood cells given.

Controversies

Several states across the United States have been grappling with the issue of certified individuals and the administration of blood and blood products. Some states may not allow a non-licensed individual to administer blood or blood products during transport. This may force the sending facility to complete or even discontinue the administration before transfer. Facilities should be aware of their individual state regulations and the composition (as in certified vs licensed) of their critical care transport teams when faced with these types of transfers.

Continuous blood pressure and pulse oximetry monitoring is required to evaluate the patient's progress and the effectiveness of fluid resuscitation efforts. Care must be taken to avoid fluid overload secondary to excessive fluid administration in patients with comorbidities, specifically those with cardiac-related conditions. Placement of a pulmonary artery catheter or other hemodynamic monitoring device should occur as soon as possible because it allows for a detailed and current analysis of hemodynamic status. Continuous ECG monitoring is necessary to observe the patient for a variety of arrhythmias that may develop secondary to electrolyte imbalances or hypoxia.

Along with treating the immediate concerns of GI bleeding, other factors such as incontinence, diarrhea, and constipation need to be addressed. Incontinence will most often be controlled with a Foley catheter placed prior to transport. Foley catheter placement and management is discussed in detail later in this chapter. Diarrhea control, particularly during extended transports, is necessary. This can usually be accomplished with loperamide treatment. Oral rehydration therapy is important for patients with chronic diarrhea; IV fluids may be used if the oral route is unavailable. In infectious processes, the patient will commonly be administered an antibiotic to control the cause of the diarrhea. Chronic constipation is typically managed with an osmotic laxative such as milk of magnesia. When managing any of these three conditions, it is important to ensure that any underlying cause of the condition is being addressed.

Upper GI Bleeding

NG or OG tube placement is indicated in any patient with upper GI bleeding to facilitate clearing of gastric contents. Suctioning via these tubes permits an estimation of blood loss and an evaluation of the consistency of the gastric contents as coffee-ground, bright red, or blackened. Gastric lavage should be performed with 250 to 500 mL of water or saline to remove the remaining stomach contents. Gastric lavage is a necessary precursor to endoscopy, allowing the provider to determine the extent and activity of the bleeding. However, gastric lavage is not performed during transport; it is an in-house procedure that must occur before transport.

Endoscopy is performed at the hospital when the patient's condition is stable. It may serve both diagnostic and therapeutic functions. Once the bleeding site is located, injection sclerotherapy—injection of small doses of either epinephrine or clotting factors into the insult site—can be performed via endoscopy. Epinephrine causes a local vasoconstriction that results in decreased blood flow to the area; clotting stimulators such as human thrombin or fibrin glue will slow or stop the bleeding process significantly.

Endoscopic variceal band ligation can be used to control bleeding esophageal varices. This technique involves the placement of rubber bands around the varix, which shuts off its blood supply. Cauterization methods and hemostatic clip placement are alternatives to these treatments.

Figure 16-23 Sengstaken-Blakemore tube. © C. R. Bard, Inc.

Vasopressin administration induces vasoconstriction of the splanchnic arteries, which reduces mesenteric blood flow and thereby reduces portal hypertension. This effect can be beneficial to the patient with esophageal varices, in that it substantially reduces blood flow to the region. Nitroglycerin is commonly administered in conjunction with vasopressin to control subsequent rises in coronary artery blood pressure. An alternative to vasopressin is octreotide, a synthetic version of somatostatin. Many cardiac complications associated with vasopressin-nitroglycerin treatment may be avoided by initiating in the patient the more selective vasoconstriction that octreotide provides.

Treatments for endoscopic and pharmacologic refractory variceal hemorrhage include balloon inflation and shunt placement. Balloon tamponade of variceal bleeding is effective in controlling the hemorrhage in as many as 90% of cases. In this procedure, a **Sengstaken-Blakemore tube** **Figure 16-23** or a **Minnesota esophagogastric tamponade tube** is inserted nasally and introduced to the stomach via the esophagus. A gastric balloon is then inflated to place pressure on the cardia region of the stomach. A second esophageal balloon can be inflated if necessary to further tamponade bleeding. Although balloon placement has a high success rate in initial bleeding control, it is associated with a high rate of bleeding recurrence upon removal. Given this fact, it is rarely used today.

A more frequently employed procedure is the placement of a **transjugular intrahepatic portosystemic shunt** to control variceal bleeding **Figure 16-24**. The introduction of a shunt into the systemic venous circulation results in a significant decrease in hepatic portal pressure and, therefore, a significant decrease in the pressure in the esophageal collateral veins. Shunt placement has a greater than 90% success rate with less than 30% bleeding recurrence.

Proton pump inhibitors should be administered to reduce gastric acidity in any patient suspected of having a peptic ulcer. Antacids can greatly reduce gastric acidity in a short period of time, thereby reducing ulcer irritation. Prophylactic acid inhibition is common in patients admitted to an intensive care unit to prevent the development of stress ulcers. Patients testing positive for *H pylori* should be treated with antibiotics specific to the bacteria, such as omeprazole and clarithromycin.

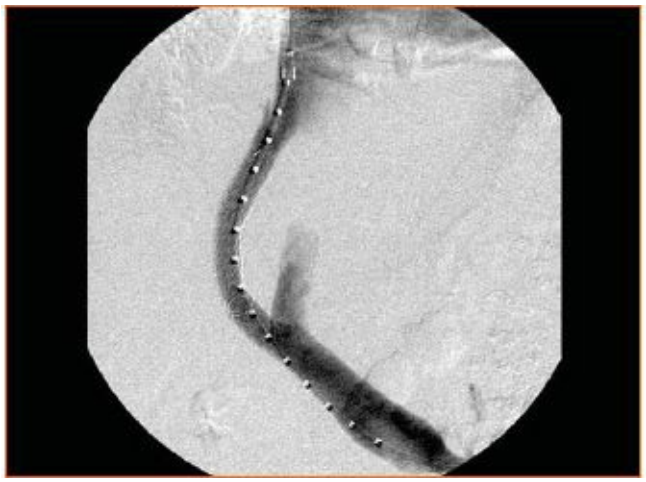


Figure 16-24 Transjugular intrahepatic portosystemic shunt placement. Reprinted with permission from the *American Journal of Roentgenology*.

Lower GI Bleeding

Management of the patient with lower GI bleeding is similar to that of the patient with upper GI bleeding. Attention must be paid to fluid loss through water shifts in the colon and the associated dehydration and electrolyte abnormalities that can occur. Endoscopic methods of cauterization and coagulation are common first-line treatments. Intra-arterial injection therapy with vasopressin or various hemostatic agents is associated with a 70% success rate. Surgical methods such as colon resection and removal are indicated in bleeding refractory to endoscopic and colonoscopic techniques, but are infrequently required.

Intestinal Obstructions

Blockage of the GI tract can occur in the small or large intestine through a variety of pathologies. The convoluted small intestines are far more susceptible to obstruction than are the large intestines, which are wider in diameter. Obstructions are classified as partial, simple, complete, or strangulated. Although a partial intestinal obstruction can cause some discomfort and malnutrition, a complete blockage stops normal GI function, resulting in death if not treated promptly.

Epidemiology and Pathophysiology

Postoperative adhesions are the major cause of intestinal obstructions, accounting for up to 60% of obstructions. Other common causes include tumors, hernias, and Crohn's disease. Of those patients diagnosed as having an intestinal obstruction, 40% are strangulated obstructions. Overall, intestinal obstructions are fairly common in the United States, accounting for up to 20% of all acute surgical admissions to hospitals.

Mechanical Obstruction

A mechanical obstruction is the result of a physical blockage of the intestinal lumen and can be classified into one of three categories: extrinsic, intrinsic, or intraluminal **Table 16-6**.

Extrinsic causes, which originate external to the intestines, include adhesions, hernias, volvulus, and masses. **Adhesions** are bands of connective tissue that can distort the normal anatomy of the abdomen. They are most often the result of improper healing or scar tissue growth following abdominal surgery. A **hernia** is a protrusion of an organ from its tissue lining. Overall, postsurgical adhesions are the most common cause of mechanical intestinal obstruction, followed by hernias. A **volvulus** is a twisting of the intestine onto itself, usually resulting in strangulation. **Masses** often originate from surrounding abdominal

organs that put pressure on the intestines and may be tumors, aneurysms, or abscesses.

Intrinsic causes arise from the intestinal lining itself, such as diverticula, neoplasms, or intussusception. An **intussusception** is a prolapse of the intestine into an adjacent segment.

An intraluminal obstruction is commonly the result of an ingested foreign body or a fecal impaction. Figure 16-25 through Figure 16-28 show intestinal obstructions caused by hernias, volvulus, a mass, intussusception, and a foreign body, respectively.

Chyme, fluids, and digestive products cannot pass an intestinal obstruction, and immediately begin to accumulate proximal to the blockage. This buildup, along with swallowed air, will distend the lumen proximal to the obstruction. GI tract distention stimulates an increase in digestive juice secretion and peristalsis, worsening the situation. The continually increasing intraluminal pressure also increases the permeability of mucosal capillary beds, allowing vascular fluid to flow into the intestinal lumen. This phenomenon, which is termed **third spacing**, can lead to dehydration, electrolyte imbalances, and hypovolemia.

Over time, the stagnation of intestinal contents facilitates significant bacterial growth. As bacteria enter the mucosal tissues, infections leading to peritonitis or sepsis may occur. If luminal distension proximal to the blockage site continues unchecked, a bowel rupture can occur. Once ruptured, the contents of the GI tract will irritate surrounding tissues and likely cause peritonitis.

TABLE 16-6 Causes of Intestinal Obstruction

Type of Obstruction	Cause
Mechanical	Extrinsic • Adhesions • Hernias • Volvulus • Masses Intrinsic • Diverticula • Neoplasms • Intussusception Intraluminal • Foreign body ingestion • Fecal impaction
Nonmechanical (ileus)	Postoperative Acute colonic pseudo-obstruction Abdominal inflammation Peritonitis Heavy metal poisoning Metabolic abnormalities Spinal cord injury

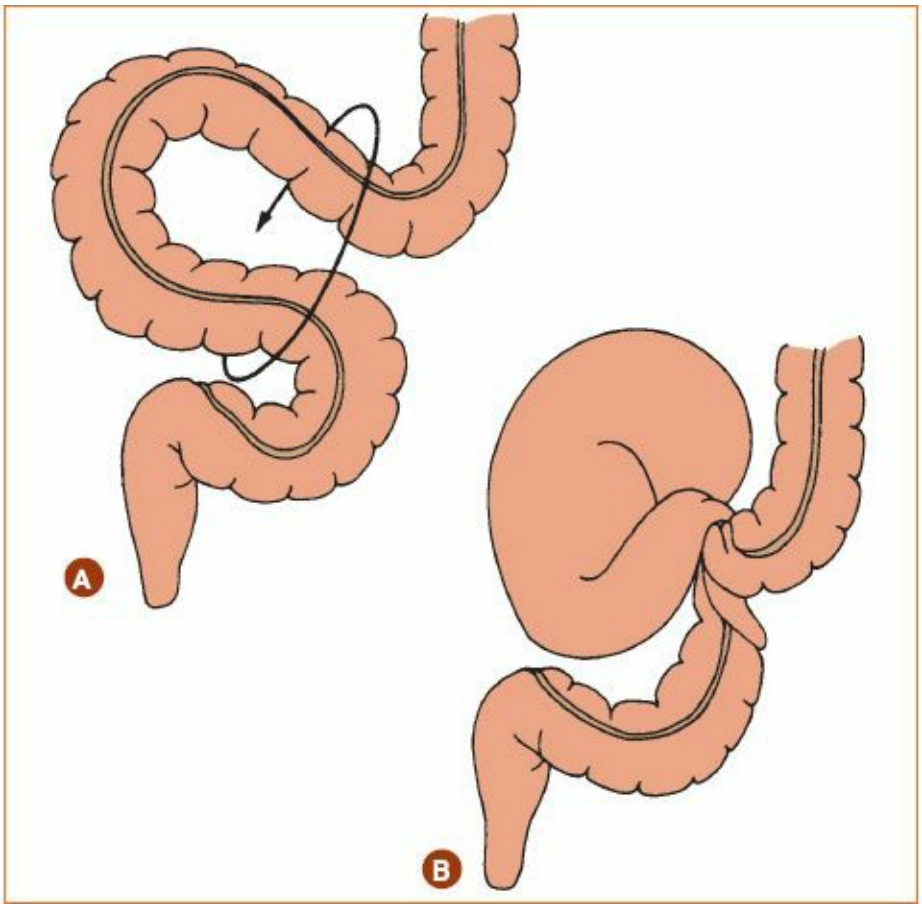


Figure 16-25 The pathogenesis of volvulus. **A.** Rotary twist of the sigmoid colon on its mesentery. **B.** Obstruction of the colon and interruption of its blood supply caused by volvulus.

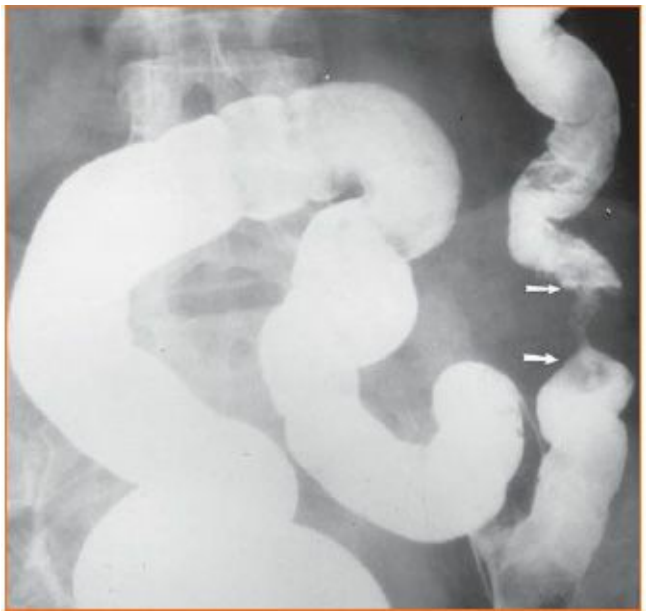


Figure 16-26 A colon carcinoma demonstrated by barium enema. The tumor narrows the lumen of the colon, which appears as a filling defect in the column of barium (*arrows*).

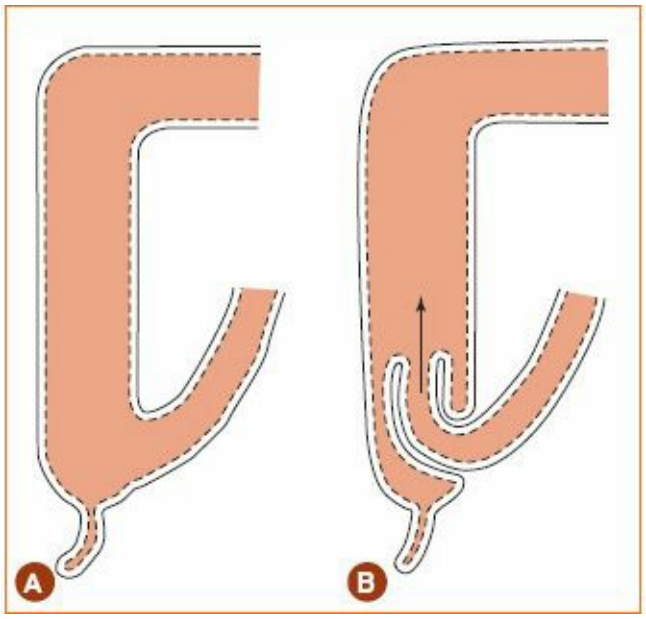


Figure 16-27 The pathogenesis of intussusception. **A.** Normal anatomic relationships. **B.** Vigorous peristalsis carries the distal ileum into the cecum. The *dashed line* indicates mucosa.

Distal to the obstruction site, tissue ischemia occurs secondary to arterial occlusion. This ischemia inhibits any residual GI functioning and eventually results in necrosis.

Ileus

Ileus is the lack of movement of the GI contents through the intestines in the absence of a mechanical obstruction. The most common type occurs postoperatively and is idiopathic, possibly resulting from the use of an anesthetic, which interferes with mesenteric innervation. The use of opiate-based medications inhibits intestinal motility, and the frequent administration of these medications after surgery may be a factor in postoperative ileus as well. Postoperative ileus is transient, usually lasting 48 to 72 hours after surgery. This type of ileus and other types of reversible ileus are also known as acute colonic pseudo-obstruction, or Ogilvie's syndrome. Other causes of ileus include abdominal inflammation, peritonitis, heavy metal poisoning, metabolic abnormalities, and spinal cord injury.

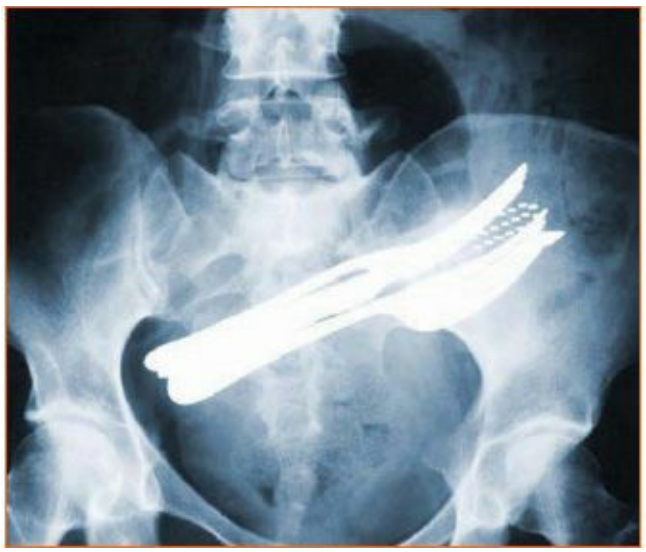


Figure 16-28 A foreign body.

Assessment

Signs and Symptoms

Moderate to severe abdominal pain accompanies distention proximal to the intestinal obstruction, with eventual vomiting. Excess bacterial growth causes bad breath, along with exceptionally foul-smelling vomit. The vomit may contain large amounts of bile and may have an appearance similar to feces depending on the location of the obstruction.

The intestinal mucosa become inflamed secondary to ischemia and necrosis. This inflammation, along with bacterial infiltration into the tissues, can cause a fever as the patient's immune response develops. Shock can develop from third spacing of fluids, vomiting, and diarrhea, manifesting as tachycardia, altered mental status, cool and clammy skin, and hypotension.

In a patient with an intestinal obstruction, the abdomen is tender and distended on palpation, typically with diffuse pain. Palpation of the mass may suggest obstruction. Initially, hyperactive and high-pitched bowel sounds may accompany an obstruction, followed by an eventual cessation of sound as GI function is increasingly inhibited.

Laboratory Data and Imaging

Laboratory testing functions mostly to determine the degree of dehydration and electrolyte imbalance from an intestinal obstruction. Hemoglobin and hematocrit values become elevated as the blood becomes more concentrated following excessive vomiting. Metabolic alkalosis frequently occurs as electrolyte shifts compensate for the loss of hydrogen ions through vomiting. White blood cell counts increase as the extent of tissue ischemia and necrosis expands.

Signs and Symptoms

Intestinal Obstruction

- Moderate to severe abdominal pain
- Tender, distended abdomen
- Foul-smelling vomit that may contain bile or appear similar to feces
- Diarrhea
- Bad breath
- Fever
- Shock
- Tachycardia
- Altered mental status
- Cool, clammy skin
- Hypotension
- Hyperactive, high-pitched bowel sounds initially
- Eventual cessation of bowel sounds

Differential Diagnosis

Intestinal Obstruction

- Diverticulitis
- Cholelithiasis
- Endometriosis

- Foreign body
- Gastroenteritis
- Inflammatory bowel disease
- Pancreatitis
- Appendicitis
- Urinary tract infection
- Pelvic inflammatory disease
- Ovarian torsion
- Cholangitis

Transport Management

Intestinal Obstruction

- Stabilize the ABCs.
- Provide fluid resuscitation.
- Provide supplemental oxygen.
- Continuously monitor the ECG.
- Designate NPO.
- Place an OG or NG tube.
- Administer prophylactic antibiotics.

Plain radiography can diagnose an intestinal obstruction by detecting abnormally high air volumes proximal to the obstruction site. A lack of air distal to the obstruction also can be detected, permitting a fairly accurate localization of the problem. **Enteroclysis** can be used in conjunction with radiography to provide a more accurate diagnosis. In this procedure, a barium contrast dye is infused into the duodenum through an OG or NG catheter. The progression of the catheter through the intestines can be monitored on a fluoroscope screen in real time, allowing for observation of the blockage.

A CT scan can be used to diagnose a bowel obstruction with exceptional specificity, and this technique is often used when prior imaging studies produce indeterminate results. An abdominal CT scan is useful in differentiating the cause of the obstruction and observing tissue alterations and inflammations. Abdominal ultrasonography can also be used to diagnose an intestinal obstruction with great accuracy.

Management

After the stabilization of any life threats to the patient's airway, breathing, or cardiac functioning, initial treatment focuses on fluid resuscitation. Isotonic fluid boluses should be administered as needed to maintain adequate perfusion. Supplemental oxygen should be provided through either a nonrebreathing mask or a nasal cannula. The patient's ECG should be continuously monitored for signs of arrhythmias secondary to electrolyte imbalances.

Patients with a suspected intestinal obstruction should be placed on NPO restrictions, and an OG or NG tube should be placed to relieve distention proximal to the blockage. Prophylactic antibiotics are frequently administered to reduce the likelihood of infection and subsequent sepsis.

Foley catheter placement can assist in monitoring fluid resuscitation progress. The Foley catheter may or may not be placed by the CCTP depending on regional protocols. Patients with chronic intestinal

obstruction may receive total parenteral nutrition (TPN) to help them maintain adequate nutrition. Emergency surgery is required in patients with a strangulated bowel. Techniques are directed toward reducing the obstruction through laparoscopic lysis or circumventing the obstruction by a bowel resection. Specific treatment for ileus is aimed at correcting the underlying cause.

NG Tube Insertion

The following equipment is needed to place an NG tube:

- NG tube
- 50-mL irrigation syringe
- Water-soluble lubricant
- Adhesive tape
- Saline for irrigation
- Emesis basin
- Gloves
- Stethoscope
- Low-powered suction unit

Indications for NG tube placement are as follows:

- Evacuation of stomach contents
- Dilution or lavage of poisons in the stomach
- Removal of blood in patients with GI hemorrhage

Contraindications to NG tube placement include severe facial trauma, croup, and epiglottitis. Potential complications of NG tube placement include improper positioning of the tube: (1) turbinate insertion, causing bleeding and pain; or (2) tracheal insertion, causing the patient to cough and choke. If the tube cannot be advanced to its predetermined length, then it has most likely curled in the mouth, throat, or trachea.

Skill Drill 16-1 shows the steps for NG tube placement as follows:

1. Use BSI precautions.
2. Assemble your equipment.
3. Explain the procedure to the patient, and oxygenate the patient if necessary. Position the patient in the sitting position. Ensure that the patient's head is in a neutral position and suppress the gag reflex with a topical anesthetic spray **Step 1**.
4. Examine the patient's nose for deformity or obstruction; determine the best side for insertion.
5. Constrict the blood vessels in the nares with a topical alpha-agonist **Step 2**.
6. Measure the tube from patient's earlobe to the tip of the nose.
7. Measure from the patient's earlobe to the bottom of the xiphoid process.
8. Total the two measurements and mark the correct length on the tube with adhesive tape **Step 3**.
9. Lubricate 6″ to 8″ of the tube with a water-soluble gel **Step 4**.
10. Insert the tube into one nostril. Gently advance the tube toward the posterior nasopharynx **Step 5**.
11. When you feel the tube at the nasopharyngeal junction, rotate it 180° inward toward the other nostril.

12. Gently advance the tube until it is in the nasopharynx. As the tube enters the oropharynx, instruct the patient to swallow **Step 6**.
13. Pass the tube to the predetermined point **Step 7**. Do not force the tube if resistance is encountered.
14. Check placement of the tube in two ways: auscultate over the epigastrium while injecting about 20 to 30 mL (no more than 50 mL) of air into the tube and/or observe for gastric contents in the tube **Step 8**. There should be no reflux around the tube.
15. Apply suction (at a low setting) to the tube to aspirate the gastric contents, and secure the tube in place **Step 9**.

OG Tube Insertion

The following equipment is needed to place an OG tube:

- OG tube
- 50-mL irrigation syringe
- Water-soluble lubricant
- Adhesive tape
- Saline for irrigation
- Emesis basin
- Gloves
- Stethoscope
- Low-powered suction unit

Indications for OG tube placement are as follows:

- Threat of aspiration
- Pressure of the stomach on the diaphragm
- Patient is unconscious

Contraindications to OG tube placement include:

- Esophageal disease or esophageal trauma (use extreme caution)
- Esophageal obstruction

Complications include:

- Patient biting tube

Skill Drill 16-2 shows the steps for OG tube placement:

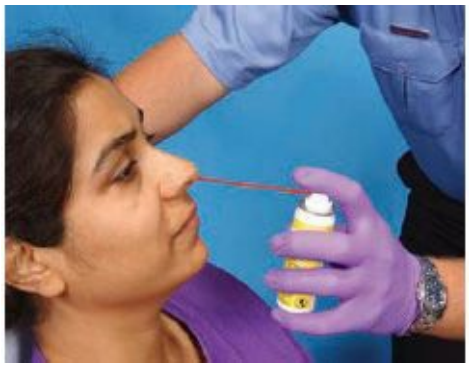
1. Position the patient's head in a neutral or flexed position **Step 1**.
2. Measure the tube for the correct depth of insertion (mouth to ear to xiphoid process) **Step 2**.
3. Lubricate the tube with a water-soluble gel **Step 3**.
4. Introduce the tube at the midline, and advance it gently into the oropharynx **Step 4**.
5. Advance the tube into the stomach **Step 5**.
6. Confirm proper placement: auscultate over the epigastrium while injecting 30 to 50 mL of air and/or observe for gastric contents in the tube. There should be no reflux around the tube **Step 6**.
7. Apply suction to the tube to aspirate the stomach contents, and secure the tube in place **Step 7**.

An NG or OG tube may already be in place upon the CCTP's first encounter with the patient, particularly during interfacility transports. In such cases it is important to ensure continuous proper placement of the tube and to be alert for any potential complications that may arise. Correct placement may also be ensured through pH testing. To accomplish this, use a pH indicator test strip and place a small amount of aspirated stomach contents on it. Proper placement will test acidity, with a pH of less than 6. Aspiration is always a concern for patients with an NG or OG tube inserted, whether for suction or feeding. If a patient appears to be in distress with a tube in place, first attempt to ensure proper placement by injecting a small air bolus into the tube while auscultating the epigastrium. If the patient continues to gag or is vomiting, suctioning and removal of the tube may be necessary. Following such action, reinsertion may be performed if permitted by regional protocols and if the patient's condition allows.

Skill Drill 16-1

Inserting a Nasogastric Tube in a Conscious Patient

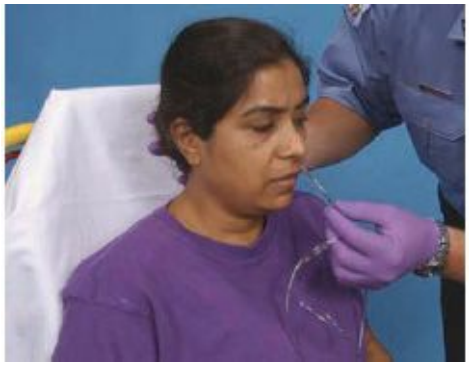
1 Explain the procedure to the patient. Ensure that the patient's head is in a neutral position and suppress the gag reflex with a topical anesthetic spray.



2 Constrict the blood vessels in the nares with a topical alpha-agonist.

3 Measure the tube for the correct depth of insertion (nose to ear to xiphoid process). Mark the correct length on the tube with adhesive tape.

4 Lubricate the tube with a water-soluble gel.



5 Advance the tube gently along the nasal floor.

6 Encourage the patient to swallow or drink to facilitate passage of the tube.

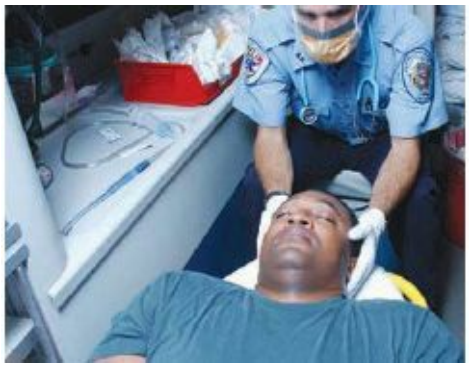
7 Advance the tube into the stomach (to the predetermined point).

8 Confirm proper placement: auscultate over the epigastrium while injecting 30 to 50 mL of air into the tube, and/or observe for gastric contents in the tube. There should be no reflux around the tube.

9 Apply suction (at a low setting) to the tube to aspirate the gastric contents, and secure the tube in place.

Skill Drill 16-2

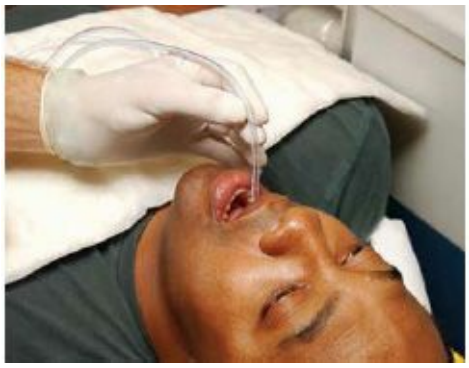
Orogastric Tube Insertion



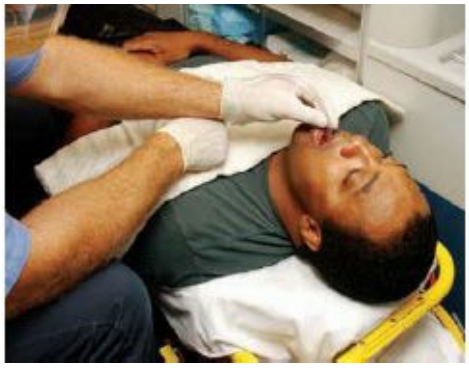
1 Position the patient's head in a neutral or flexed position.

2 Measure the tube for the correct depth of insertion (mouth to ear to xiphoid process).

3 Lubricate the tube with a water-soluble gel.

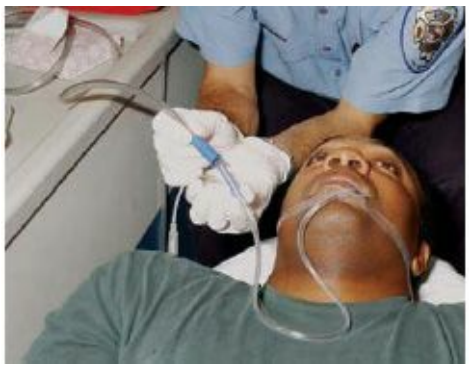


4 Introduce the tube at the midline, and advance it gently into the oropharynx.



5 Advance the tube into the stomach.

6 Confirm proper placement: auscultate over the epigastrium while injecting 30 to 50 mL of air and/or observe for gastric contents in the tube. There should be no reflux around the tube.



7 Apply suction to the tube to aspirate the stomach contents, and secure the tube in place.

Liver Disease

Considering the amount of vasculature that passes through the liver, it is easy to imagine the far-reaching implications that any insult to this organ could cause. In general, an insult to the liver is either infectious or noninfectious. If the insult resolves within 6 months without any permanent function deficit, it is termed acute. When symptoms persist longer, chronic liver disease has developed. Regardless of the cause, most cases of chronic liver disease find a common ending—cirrhosis.

Epidemiology and Pathophysiology

Types of Liver Disease

Hepatitis, an inflammation of the liver, is the physiological result of any liver disease. Viral infection by hepatitis types A, B, C, D, and E accounts for 90% of acute hepatitis cases. The remaining 10% of cases result from excessive alcohol consumption, autoimmune disorders, toxins, and drugs. Infection by **hepatitis C virus (HCV)** is especially prominent when the disease becomes a chronic condition, with this infection evolving into chronic hepatitis with a frequency of 80%.

Fulminant hepatic failure is the result of a sudden significant insult to the liver. It occurs less commonly than the hepatitis process, but is associated with mortality rates ranging from 50% to 90%. The leading causes of fulminant hepatic failure are acetaminophen toxicity (39%), idiosyncratic drug reactions (13%), and viral hepatitis (12%). This condition is characterized by the development of encephalopathy within 2 weeks of jaundice onset.

The leading cause of cirrhosis is infection with HCV, with alcoholic liver disease closely following. (It is interesting that the lowest rates of cirrhosis in the United States occurred during Prohibition, when

alcohol was made illegal.) The remaining cases are largely a result of cryptogenic causes, such as nonalcoholic fatty liver disease and hepatitis B virus (HBV) infection; other causes are responsible for only 5% of cases **Table 16-7**. Cirrhosis is characterized by irreversible structural changes to the liver that impair its functioning. Once the disease is advanced, the only cure is liver transplantation.

Course of Liver Disease

Inflammation of liver tissue results in damage to a variety of functional cells, including hepatocytes and Kupffer cells. As cells are destroyed, the basic acinar framework involved in blood detoxification and bile production is disrupted. Liver sinusoids and bile drainage canals are inflamed, creating an increased resistance to portal blood flow and bile passage. The increased portal resistance to blood flow can develop into portal hypertension, which has significant effects on the body **Table 16-8**. Splenomegaly can result from the initial blood backup, decreasing the effectiveness of the spleen's metabolic processes. Collateral circulation in the mesentery increases as blood attempts to find a low-resistance route to the vena cava, resulting in an increased risk for esophageal or gastric varices.

TABLE 16-7 Causes of Hepatitis and Cirrhosis

Condition	Cause
Hepatitis	Viral Alcohol abuse Autoimmune disorders Toxins Drugs
Cirrhosis	Hepatitis C Alcoholic liver disease Nonalcoholic fatty liver disease Hepatitis B Miscellaneous

Bilirubin that is normally conjugated by hepatocytes and excreted via bile begins to build up in the blood, resulting in jaundice. Gray feces develop as bilirubin levels decrease in the GI tract. Urine darkens as bilirubin is excreted through the renal system.

The lack of proper nitrogen fixation by damaged hepatocytes can lead to increased concentrations of blood ammonia. The increased circulating ammonia is thought to be the major neurotoxin responsible for **hepatic encephalopathy**, although the overall mechanism is poorly understood. Cognitive changes are seen from the disruption of cerebral function, followed by cerebral edema and coma in 75% to 80% of cases.

In alcoholic liver disease, the liver cells are distended from fat accumulation, disrupting normal function. Daily ingestion of more than 40 to 80 g of ethanol results in excessively reduced nicotinamide adenine dinucleotide (NADH) production from ethanol oxidation in the liver. Elevated hepatocyte NADH concentrations stimulate triglyceride synthesis; these triglycerides are stored within the cell once produced. The continued fat accumulation over the course of 10 to 12 years will result in an enlarged and jaundiced liver.

Cirrhosis develops once hepatocyte damage and destruction become widespread enough to cause local areas of necrotic tissue, such that liver cells attempt to regenerate themselves. During this process, fibroblasts deposit collagen in a disproportionately high rate throughout the extracellular matrix. The

increase in fibrotic tissue distorts the anatomy of the liver, further impairing its function. This permanent disruption of the liver's structure only insinuates further inflammation and portal hypertension.

TABLE 16-8 Effects of Portal Hypertension
Splenomegaly (enlarged spleen)
Esophageal varices
Gastric varices
Increased serum bilirubin • Jaundice • Gray feces • Dark urine
Azygos system overusage
Ascites
Muscle wasting
Hormonal alterations

Signs and Symptoms

Liver Disease

- Jaundice
- Dark urine
- Gray feces
- General malaise
- Fever and chills
- Ascites
- Weight gain
- Frequent bleeding
- Melena, hematochezia
- Chronic shortness of breath
- Hypotension
- Tachycardia
- Hair loss
- Cognitive disorders
- Spider skin
- Hypoglycemia

Differential Diagnosis

Liver Disease

- Acute hepatitis
- Cholestasis
- Acute hepatic failure
- Chronic hepatitis
- Cirrhosis
- Hepatomegaly
- Liver mass
- Jaundice (icterus)
- Increased **aspartate aminotransferase/alanine aminotransferase (AST/ALT) ratio**
- Increased alkaline phosphatase level

Transport Management

Liver Disease

- Manage the airway.
- Provide fluid resuscitation.
- Monitor the ECG.
- Monitor for GI bleeding, renal failure, and encephalopathy.
- Do not administer medications that are metabolized by the liver.
- Administer antiviral and interferon drugs.
- Administer corticosteroids.

Assessment

Signs and Symptoms

General malaise is the most prominent symptom in early acute hepatitis. Improper nutrient metabolism by the inflamed liver causes generalized fatigue. Fever and chills present as a result of the systemic immune response.

As mentioned previously, portal hypertension results in a series of problems, including ascites and weight gain from abdominal fluid retention, as well as frequent bleeding from platelet retention in the spleen. Melena and hematochezia can occur also as a result of induced GI bleeding from portal hypertension. Chronic shortness of breath can develop from impaired diaphragmatic expansion secondary to hepatomegaly, splenomegaly, and ascites. Vasodilation of the splanchnic artery decreases cardiac afterload with the loss of peripheral vascular resistance, causing hypotension and tachycardia. Hair loss is commonly observed as a result of improper metabolism of hormones.

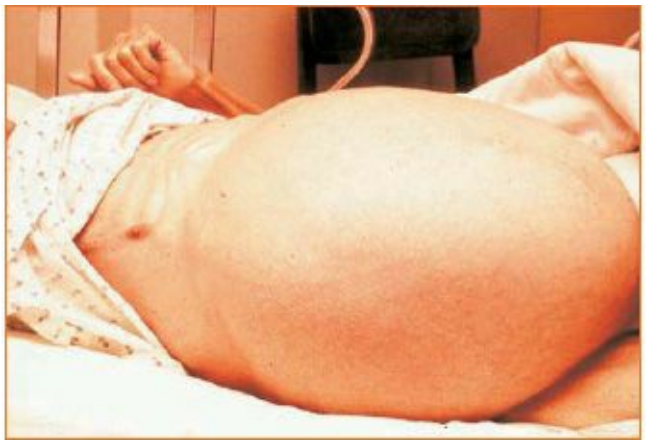


Figure 16-29 Marked ascites in a patient with advanced cirrhosis.

Laboratory Data and Imaging

Liver function is assessed by evaluating the presence of its synthesized products as well as the absence of its cellular enzymes in the blood. It is important to recognize the distinction between tests for liver function and markers of injury. By the time most lab values become abnormal and symptoms have emerged, 60% to 80% hepatocyte destruction has already occurred.

Albumin is a protein synthesized by the liver that assists in maintenance of osmotic balance in the body tissues. A decrease in its production results in the peripheral edema, **ascites** **Figure 16-29**, and pulmonary edema observed in patients with liver failure. Elevated serum levels of bilirubin indicate hepatocyte insufficiency and are a reliable indicator of the severity of liver damage. A prolonged PT time is the result of decreased clotting factor synthesis by the liver, which is also frequently a result of hepatocyte insufficiency secondary to injury.

Release of enzymes specific to hepatocytes into the bloodstream can be detected through laboratory testing and is useful as a marker of the existence of liver disease and its progression. These enzymes include AST, ALT, and gamma-glutamyltransferase (GGTP or GGT). Elevated serum levels of any of the three can suggest hepatic insult, with increases in ALT being the most specific to the liver. GGT is very sensitive to hepatocyte damage, with its level becoming elevated after even one episode of binge drinking. Elevated GGT levels also suggest cholestasis secondary to a biliary obstruction, particularly in conjunction with elevated alkaline phosphatase (AP) levels.

In patients presenting with an altered mental status, a blood glucose test can reveal hypoglycemia from decreased glucose metabolism and glycogen storage secondary to hepatocyte injury. Elevated serum ammonia concentrations from decreased nitrogen fixation in the damaged liver can result in hepatic encephalopathy. Increased levels of BUN and serum creatinine indicate impaired renal function and can suggest fulminant hepatic failure with hepatorenal syndrome **Table 16-9**.

Although no imaging studies are necessary in the diagnosis of hepatitis, they can prove valuable in differentiating hepatitis from similarly presenting diseases. An abdominal CT scan and ultrasound can support a diagnosis of hepatitis through visualization of an enlarged liver. Severity and progress of the disease can be measured through imaging to some extent as well **Figure 16-30**.

TABLE 16-9 Normal vs Abnormal Laboratory Values in Liver Disease

Test	Normal	Abnormal
Albumin	3.8–5.0 g/dL	< 3.8 g/dL
Bilirubin, serum (direct, conjugated)	0.1–0.3 mg/dL	> 0.3 mg/dL

Bilirubin, total	0.3–1.2 mg/dL	> 1.2 mg/dL
Prothrombin time	10-13 s	> 13 s
Alkaline phosphatase	30–120 U/L	> 120 U/L
Serum ammonia	10–80 μg/dL	> 80 μg/dL
Blood glucose	70–110 mg/dL	< 70 mg/dL
Blood urea nitrogen	8–23 mg/dL	> 23 mg/dL
Creatinine, serum	0.6–1.2 mg/dL	> 1.2 mg/dL
Liver Enzymes		
Aspartate aminotransferase	10–30 U/L	Elevated (usually 42 U/L)
Alanine aminotransferase	10–40 U/L	Elevated (usually 48 U/L)
Gamma-glutamyltransferase	2-30 U/L	Elevated (usually 55 U/L)

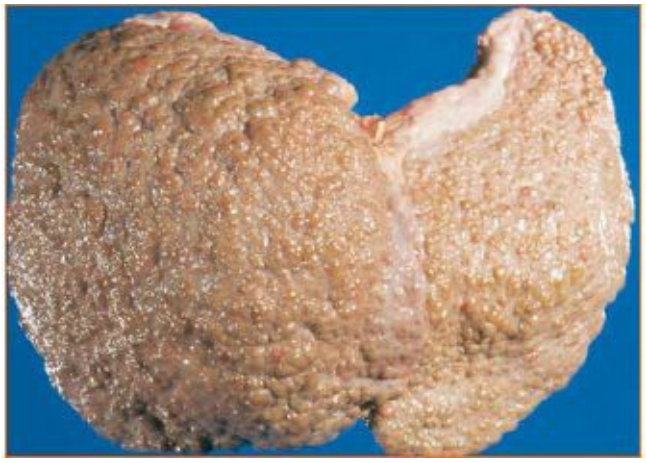


Figure 16-30 Advanced hepatic cirrhosis illustrating elevated nodules of liver tissue surrounded by depressed areas of scar tissue.

Management

Treatment of hepatitis focuses on prevention of further injury with a goal of preventing progression to cirrhosis. Antiviral and interferon drugs are administered in cases of infectious hepatitis to suppress the viral load. Many of these drugs are used in the treatment of human immunodeficiency virus (HIV) infection as well. Corticosteroids are frequently administered to reduce inflammation, and are most effective in the early stages of the disease. Pharmacologic treatment of hepatitis is an active topic of research, and usage of specific medications will vary between institutions.

The bulk of treatment is supportive, mainly concentrating on prevention and correction of any complications. In more severe cases of liver disease, neurologic function can be diminished, requiring airway management. Third spacing secondary to hypoalbuminemia can cause hypotension and is treated with fluid resuscitation. ECG monitoring should be done to observe for arrhythmias from electrolyte imbalances, especially hypokalemia. Patients should be monitored for evidence of GI bleeding, renal failure, and encephalopathy. Avoidance of alcohol and medications metabolized by the liver is mandatory. In many cases, dosages of common medications must be reduced in anticipation of the liver's decreased function.

Prevention of ascites through a low-sodium diet, fluid restriction, and diuretics is often necessary in patients with cirrhosis, keeping an eye toward maintaining blood pressure. **Paracentesis**—"tapping" of the abdomen with a needle—can be used to physically remove ascetic fluid from the abdomen, because its accumulation can sometimes amount to as much as 4 to 6 L/d.

There is no cure for hepatitis or the resultant cirrhosis other than liver transplantation. Liver transplantations have a fairly high success rate, with 1-year survival rates ranging from 85% to 90% and 5-year survival rates exceeding 70%. Unfortunately, there are more cases of cirrhosis than there are available livers, and 12% to 15% of patients die awaiting a donor organ.

Biliary Tract Obstructions

Blockage of the biliary tracts at any point will result in a backup of bile and possibly, depending on the location of the obstruction, pancreatic enzymes. These digestive juices accumulate first in the tracts, and then in the organs from which they originated. The damage caused by this type of obstruction is twofold. Distal to the obstruction, normal digestive processes of the GI system are disrupted, while proximal accessory organs and ducts become distended and inflamed.

Epidemiology and Pathophysiology

Most often, the biliary tract system is obstructed by a migrating gallstone. Gallstones in the gallbladder, **cholelithiasis**, are fairly common, affecting around 20 million Americans each year **Figure 16-31**. They can form one of two ways: through accumulation of excess cholesterol in the bile (cholesterol stones) or through accumulation of excess bilirubin and calcium salts in the bile (pigment stones). Both causes result in the crystallization of bile into stones, which may range in size from small grains to the size of a golf ball. Cholesterol stones account for 75% to 80% of gallstone cases in the United States, with the remainder accounted for by pigment and mixed stones. It is also notable that patients with cirrhosis are at higher risk for pigment stones.

Inside the gallbladder, stones may cause inflammation of the gallbladder itself, known as **cholecystitis**. When the stone is inside the biliary ducts, the condition is termed **choledocholithiasis**. The stone may begin to travel into the cystic duct at any time, lacerating the inner walls as it moves. Frequently, it will lodge in either the cystic duct or the distal common bile duct. If the stone lodges in the common bile duct and fully obstructs the lumen, both bile and pancreatic juice will be unable to pass into the duodenum. As pancreatic enzymes accumulate proximal to the obstruction, they begin to digest the tissues of both the ducts and the pancreas, a condition responsible for almost half of all cases of pancreatitis. As enzymes and bile leak through the compromised duct lining, peritonitis can occur and eventually lead to sepsis and shock.

Figure 16-31 Open gallbladder filled with gallstones composed of cholesterol.

Assessment

Signs and Symptoms

Cholecystitis causes colicky pain in the right upper quadrant of the abdomen. This pain tends to emerge following ingestion of high-fat foods, because fatty chyme stimulates gallbladder contraction.

Patients with choledocholithiasis typically present with fever, jaundice, and right upper quadrant pain, which are collectively referred to as **Charcot's triad**. Nausea and vomiting frequently accompany Charcot's triad, along with clay-colored feces. A patient with this condition will also exhibit a positive **Murphy's sign** (pain in the right upper abdomen on deep inspiration during palpation) when the fingers are pressed under the rib cage.

Laboratory Data and Imaging

The effects of biliary tract obstruction can be observed through elevations of the serum values of blocked secretions from the gallbladder and pancreas. Serum bilirubin levels increase, along with serum amylase and lipase. AP and GGT levels will also be elevated. The PT will increase as vitamin K absorption levels decrease owing to a lack of bile in the GI tract. In infective cases, known as **cholangitis**, white blood cell levels also rise.

Transabdominal ultrasonography can detect stones both in the gallbladder and in the biliary duct system. Although this imaging technique's accuracy is decreased in the common bile duct, distention proximal to the obstruction can usually be detected via this technology.

Signs and Symptoms

Biliary Tract Obstruction

- Charcot's triad
  - Fever
  - Jaundice
  - Right upper quadrant pain
- Nausea and vomiting
- Clay-colored feces
- Murphy's sign: tender right upper quadrant

Differential Diagnosis

Biliary Tract Obstruction

- Appendicitis
- Cholecystitis
- Cholangitis
- Diverticulitis
- Gastritis
- Peptic ulcer disease
- Hepatitis
- Inflammatory bowel disease
- Pancreatitis

- Biliary stricture
- Choledocholithiasis
- Pancreatic tumor
- Choledochal cyst

Transport Management

Biliary Tract Obstruction

- Maintain the ABCs.
- Provide oxygen as required.
- Place an IV.
- Monitor the ECG.
- Avoid administering opiates for pain management.

Endoscopic ultrasonography is an alternative technique that is used when transabdominal ultrasonography yields indeterminate results. This imaging technique uses a probe to detect the presence of stones in the common bile duct from the duodenum. This technique has great accuracy, but is also associated with complications secondary to its invasiveness.

If endoscopy is unsuccessful, a **hepatoiminodiacetic acid (HIDA) scan** can be performed. This test is similar to the technetium-99m–labeled red blood cell scintigraphy used to assess GI bleeding. A radiologically labeled chemical specific for bile incorporation is introduced into the patient. This marker's journey is then observed through radiography. Any obstruction to its expected path can be clearly noted, permitting a confident diagnosis of the obstruction. Also, a CT scan and MRI are useful techniques in the diagnosis of both cholelithiasis and choledocholithiasis.

Endoscopic retrograde cholangiopancreatography and percutaneous transhepatic cholangiography are two methods of cholangiography that have excellent accuracy in detecting choledocholithiasis. Both techniques involve injection of contrast dye into the biliary duct system to observe the obstruction. Unfortunately, both methods are also fairly invasive and carry a high risk of complications. For this reason, they are used only when less invasive techniques are unsuccessful.

Management

Immediate treatment of choledocholithiasis involves pain management once general treatment has been performed. Morphine has traditionally been avoided because it can theoretically cause spasm of the biliary ducts at the ampulla of Vater. Despite this, adequate pain management should not be withheld in the case where this is the only agent available.

Definitive treatment occurs at the hospital and focuses on removal of the stone to relieve the obstruction. Endoscopic retrograde cholangiopancreatography is frequently used to remove the stone after fulfilling its diagnostic role. **Lithotripsy** can break apart larger stones through external vibrations, facilitating removal by endoscopic retrograde cholangiopancreatography or percutaneous transhepatic cholangiography. A sphincterotomy is often performed to allow the stones to pass into the duodenum, where they can be caught with a basket or catheter.

Pancreatic Disease

Epidemiology and Pathophysiology

Inflammation of the pancreas, known as **pancreatitis**, can lead to significant impairment of gastrointestinal physiology depending on its level of severity and etiology. The most common causes are gallstones (45%) and alcohol abuse (35%), with the remainder of cases resulting from idiosyncratic drug reactions, tumors, hypertriglyceridemia, hypercalcemia, and congenital defects **Table 16-10**. The majority of cases are mild, self-resolving in 3 to 4 days with a very low mortality. **Systemic inflammatory response syndrome** develops in 10% to 15% of patients, marking a shift to severe acute pancreatitis with a 15% to 40% mortality rate.

Chronic pancreatitis is characterized by the presence of irreversible anatomic changes to the pancreas, along with some degree of function loss. Long-term alcohol use is the major cause of this condition, although the exact pathological mechanisms are unknown. Chronic pancreatitis has an incidence of about a third of that for acute pancreatitis per year in the United States. Pancreatitis is more likely to develop in men than in women, and African-Americans have an incidence three times that of the national average.

The inflammatory process in pancreatitis comes in response to self-digestion of the pancreatic tissue. Pancreatic enzymes are normally produced as inactive precursors that are activated in the GI lumen. When these enzymes become prematurely activated inside either the pancreas or the ducts that drain the secretions, they immediately begin digesting surrounding tissue. Thanks to the retroperitoneal location of the pancreas, the activated enzymes can easily spread throughout the peritoneum as well as to other retroperitoneal organs. Once digestion reaches pancreatic blood vessels, hemorrhaging occurs. As the disease progresses, tissue necrosis begins to occur. The resulting necrotic tissue is susceptible to bacterial infection owing to its lack of integration into the immune system, such that spontaneous infections of the pancreas or peritoneum frequently occur.

TABLE 16-10 Causes of Pancreatitis

Gallstones
Alcohol abuse
Idiosyncratic drug reactions
Tumors
Hypertriglyceridemia
Hypercalcemia
Congenital defects
Systemic inflammatory response syndrome

Pancreatic pseudocysts are aggregates of cellular debris and extracellular pancreatic enzymes that are freed by the process of tissue necrosis. They occur in about 20% of cases of acute pancreatitis and often rupture, furthering the scope of pathogenesis. Increased or unexpected presence of pancreatic enzymes throughout the body is believed to be the cause of the various multisystem complications observed in pancreatitis, although the exact etiology is unknown. Pulmonary complications may include arterial hypox-emia, atelectasis, pleural effusions, pneumonia, and acute respiratory distress syndrome (ARDS). Fluid shifts and enzyme-induced vasodilation can cause hemodynamic instability and shock. The resultant decrease in organ perfusion can lead to acute renal failure. Clotting can become inhibited as a

result of increased blood concentrations of proteolytic enzymes such as trypsin.

Signs and Symptoms

Pancreatitis

- Upper abdominal pain
- Tenderness and guarding of the abdomen
- Tachycardia
- Nausea, vomiting
- Fever
- Turner's sign (discoloration of the lower back and flank)
- Cullen's sign (periumbilical discoloration)
- Jaundice
- Hemodynamic instability
- Shock

Differential Diagnosis

Pancreatitis

- Cholecystitis
- Cholangitis
- Gastritis
- Hepatitis
- Bowel obstruction
- Pancreatic cancer

Transport Management

Pancreatitis

- Administer supplemental oxygen.
- Place an IV.
- Monitor the ECG.
- Replace fluids in patients with hypovolemia.
- Administer packed red blood cells in the hemorrhaging patient.
- Monitor fluid output.
- Place an NG tube.
- Provide pain management (IV or patient-controlled analgesia devices; avoid opiates).

Assessment

Signs and Symptoms

Upper abdominal pain is the primary symptom of pancreatitis. This pain may radiate throughout the abdomen and to the back depending on the level of pancreatic involvement and can be severe at times. Tenderness and guarding of the abdomen often result as part of the pain response, as does tachycardia. Nausea and vomiting are common owing to the presence of gastric irritation. A fever will be present secondary to the body's immune response against the infectious complications of pancreatitis. Discoloration of the lower back and flank, known as Turner's sign, and periumbilical discoloration, known as Cullen's sign, are both rare and nonspecific findings of the retroperitoneal hemorrhage that may occur in severe pancreatitis. If biliary tract obstruction is the cause of the pancreatitis, jaundice may be observed. In severe hemorrhagic cases, hemodynamic instability and shock may occur.

Laboratory Data and Imaging

As the pancreatic cells are destroyed or the walls of ducts are digested, pancreatic-specific enzymes are released into the bloodstream. Serum amylase levels are the most sensitive indicators and frequently are elevated to more than three times their normal levels in case of pancreatitis. The downside to amylase-level measurement is its short half-life, as levels return to normal within 3 to 4 days. Serum lipase levels will rise and remain high later and longer than amylase levels, for 8 to 12 days after the insults. Although elevated levels of these two enzymes support a diagnosis of pancreatitis **Table 16-11**, they can result from a number of other conditions as well.

Liver function testing for elevated AP, AST, GGT, and serum bilirubin can be indicative of a biliary tract origin of disease. An ALT level greater than 150 U/L is highly suggestive of gallstone disease, a leading cause of pancreatitis. A leukocyte count greater than 16,000/μL indicates the presence of an infection, inflammation, or both. Arterial blood gas levels should be monitored for evidence of hypoxemia so that providers can attempt to preempt the development of severe respiratory complications such as ARDS.

A CT scan with contrast can provide a direct visual observation of pancreatic necrosis, peripancreatic abnormalities, abscesses, and overall size of the pancreas. It has 90% specificity and is invaluable in the determination of disease severity. For the detection of gallstones in the bile duct, magnetic resonance cholangiopancreatography (MRCP) has a 90% accuracy rate. Endoscopic retrograde cholangiopancreatography is a favorable alternative to MRCP in patients with pancreatitis with a suspected biliary origin. Along with supporting a diagnosis, it is amenable to performing simultaneous treatment via sphincterotomy and gallstone removal. MRCP is favored in situations in which the administration of iodinated contrast dye is not desired.

TABLE 16-11 Normal vs Abnormal Laboratory Values for Pancreatitis

Test	Normal	Abnormal
Amylase, serum	27–131 U/L	Elevated (usually > 100 U/L)
Lipase, serum	31–186 U/L	Elevated (usually > 60 U/L)
Aspartate aminotransferase	10–30 U/L	Elevated (usually > 42 U/L)
Alanine aminotransferase	10–40 U/L	Elevated (usually > 48 U/L)
Gamma-glutamyltransferase	2–30 U/L	Elevated (usually > 65 U/L)
Leukocytes (white blood cells)	4,500/μL–11,000/μL	Varies, elevation indicates infection

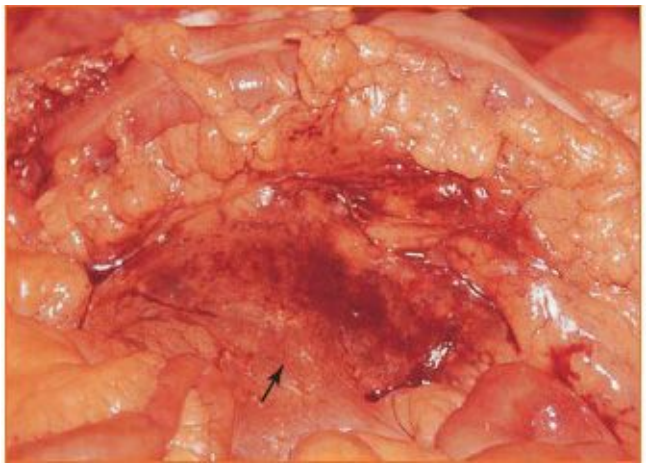


Figure 16-32 Acute pancreatitis. The transverse colon (*upper part* of the photograph) has been elevated to reveal the pancreas (*arrow*) which is inflamed and contains large areas of hemorrhage.

Visualization of the pancreas by abdominal ultrasound is often obstructed by intestinal gas and adipose tissue, such that this imaging modality has a poor success rate in identifying pancreatitis. When combined with endoscopy, it becomes a useful bedside procedure that can assist in the diagnosis of both pancreatitis **Figure 16-32** and gallstones.

Management

General treatment of pancreatitis includes palliative care such as supplemental oxygen administration, IV placement, and ECG monitoring. Fluid replacement is a priority in patients exhibiting signs and symptoms of hypovolemia to prevent decreased renal perfusion. Isotonic crystalloid boluses of 20 mL/kg are recommended, and the administration of packed red blood cells may be required in the hemorrhaging patient. Fluid output should be monitored during hydration therapy and can be facilitated by Foley catheter placement.

NG tube placement can relieve gastric distention. This measure decreases production of secretin, which in turn stimulates pancreatic enzyme secretion. All patients should receive NPO restrictions as well to prevent increased stimulation of pancreatic enzyme synthesis and secretion. In severe cases, TPN may be required.

Pain management is necessary and may take the form of IV narcotics or patient-controlled analgesia devices. Meperidine (Demerol) has traditionally been the preferred analgesic over other opiates because it avoids opiate-induced spasms of the sphincter of Oddi.

Once the patient has entered the hospital, specific treatment of acute pancreatitis focuses on prevention of complications and decreasing pancreatic workload. Prophylactic antibiotic administration to prevent bacterial peritonitis is a controversial but widely used treatment. Peritoneal lavage is used on occasion as well to remove offending substances from the peritoneal space.

Urinary System Conditions

Conditions affecting the urinary system include both acute and chronic renal failure, urinary tract infections, testicular torsion, ovarian torsion, penile fracture, ruptured ovarian cysts, and priapism. This section discusses all of these, except ovarian torsion and ovarian cysts, which are discussed in Chapter 21.

Acute Renal Failure

Epidemiology and Pathophysiology

A reliable flow of blood to the renal vasculature at a consistent rate is required for proper functioning of the nephrons. Significant decreases in renal perfusion pressure disrupt the elimination of waste from the bloodstream by lowering the GFR. When this situation develops in the absence of preexisting renal dysfunction, it is termed **acute renal failure (ARF)**. ARF is a frequently encountered complication in the hospitalized patient, particularly in the intensive care unit, where it is seen in as many as 25% of patients. Disproportionately high mortality rates (70% to 80%) are observed in patients with ARF in the intensive care unit compared with the general patient population (50%). ARF is likely a cause of the multiple comorbidities typically present in the critical care patient.

ARF is classified in terms of its etiology as prerenal, intrarenal, or postrenal. **Table 16-12** summarizes the causes and types of ARF.

Prerenal ARF is the most common cause, implicated in as many as two thirds of cases. This type of ARF is associated with many reversible causes and, if treated promptly and appropriately, can be corrected in up to 90% of cases. Hypovolemia secondary to dehydration or blood loss is often a cause of prerenal ARF, as are congestive heart failure and hepatorenal failure from cirrhosis. A variety of medications, including angiotensin-converting enzyme inhibitors and diuretics, can decrease renal perfusion through systemic vasodilation. Third spacing of fluids from sepsis is another precipitating factor.

TABLE 16-12 Summary of Causes and Types of ARF

Type	Cause
Prerenal	Hypovolemia Dehydration Congestive heart failure Hepatorenal failure Third spacing Sepsis Medications: • ACE inhibitors • Diuretics
Intrarenal	Acute tubular necrosis Contrast dye Interstitial nephritis Glomerulonephritis
	Renal calculi

Postrenal	Blood clots Tumors

Intrarenal ARF is classified as a structural injury to the kidney itself. **Acute tubular necrosis**—the most common cause of intrarenal ARF—is usually a result of renal ischemia or the presence of a toxin. Use of radiologic contrast dye has also emerged as a significant instigator of early ARF. Interstitial nephritis results from adverse reactions to antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), and diuretics.

Glomerulonephritis is another cause of intrarenal ARF, in which glomerular tissue becomes inflamed secondary to an immune response. Glomerulonephritis is a collective term covering about 20 glomerular conditions, with rapidly progressive glomerulonephritis being a severe form that impairs renal function. Glomerulonephritis is characterized by hematuria, initially on a microscopic level but progressing to visible quantities of blood in the urine and accompanied by proteinuria and hypertension. Peripheral edema from fluid buildup secondary to salt retention in afflicted nephrons can lead to congestive heart failure and encephalopathy. Postinfectious causes are frequently implicated in glomerulonephritis development. The most common cause is group A beta-hemolytic *Streptococcus* following pharyngitis infections. Other precipitators of glomerulonephritis include immunoglobulin A nephropathy and lupus.

Postrenal ARF occurs when urine flow distal to the nephrons becomes obstructed. The least frequently observed of the three types of ARF, this condition is associated with bilateral ureter or urethral blockage by renal calculi, blood clots, or lumen-penetrating tumors. Renal calculi, commonly referred to as kidney stones, are a result of salt buildup in the renal pelvis **Figure 16-33**. Most often the calculi consist of calcium-based compounds, although occasionally they are made up of struvite or uric acid. The majority of renal calculi pass through the urinary tract and are excreted without significant complications, although they certainly produce excruciating pain during their travels. A small percentage of calculi become lodged in the lumen of either the ureter or the urethra; these stones can cause life-threatening ARF and infections secondary to the buildup of urine in the kidneys. The urethra and bladder neck are typical sites of obstruction in such cases, as both ureters would require simultaneous blockage to cease all urine flow. Lodged stones can usually be removed through urinary catheterization. (Urinary catheter placement is discussed later in this chapter.)

Figure 16-33 A kidney stone.

One of the earliest external signs of ARF is oliguria, a reduction in urine volume to less than 400 mL/d. A decreased GFR results in a decrease in fluid volume entering the reabsorption tubules. This reduced volume travels slowly through the tubules as a result of a lack of pressure, allowing for elevated reabsorption of fluid and electrolytes—specifically, sodium—to occur. In many cases, the body's compensatory mechanisms for maintaining blood pressure worsen this situation. Any vasoconstrictive effort will further decrease renal perfusion, thereby reducing the GFR. This is especially important to take into account when considering administration of any vasocon-strictive medication to a hypotensive patient with suspected ARF. Eventually, decreased renal perfusion will result in tissue ischemia, injury, and necrosis. When the tubules become necrotic, they lose their ion-exchanging properties, preventing proper

urine formation. Inflammation and extensive shedding of dead tissue will further decrease the flow rate, possibly causing an obstruction.

Assessment

The patient's medical history and family history are important components of the assessment. A family history of renal disease indicates a predisposition to ARF when a precursory disease of ARF is present. The incidence of ARF is increased in patients with diabetes, hypertension, or lupus. The frequency of ARF increases greatly with age as well.

Signs and Symptoms

Signs of ARF include abnormalities in urine production. Oliguria is the most common abnormality, although an increase in urine output can occur if the nephron urine concentration mechanisms become ineffective. Lack of urine production is highly suggestive of an obstruction.

Given that dehydration is often a precipitating factor for ARF, patients commonly present with dizziness, poor skin turgor, thirst, flat neck veins, dry mucous membranes, weight loss, and orthostatic blood pressure changes. If third spacing and fluid retention are the cause of the ARF, the patient may exhibit fever, edema, and ascites. Lower back pain may be present in patients with ARF caused by infection or obstruction.

Laboratory Data and Imaging

The retention of wastes in the bloodstream that results from a decreased GFR provides an easy way to monitor for the existence and severity of ARF. Serum creatine levels will be elevated proportionally to the decrease in GFR. The level of BUN is a less reliable indicator of GFR, however, because it may be altered by numerous other metabolic functions unrelated to renal physiology, although it will also be elevated in ARF. The combined elevation in levels of BUN and serum creatine is known as **azotemia**.

Signs and Symptoms

Acute Renal Failure

- Dizziness
- Poor skin turgor
- Thirst
- Flat neck veins
- Dry mucous membranes
- Weight loss
- Orthostatic blood pressure changes
- Fever
- Edema
- Ascites
- Lower back pain

Differential Diagnosis

Acute Renal Failure

- Azotemia (increased BUN and serum creatine levels)
- Increased or decreased osmolality
- Increased specific gravity
- Increased or decreased fractional excretion of sodium and urine sodium concentration
- Acute tubular necrosis
- Chronic renal failure

Transport Management

Acute Renal Failure

- Ensure and maintain ABCs.
- Place an IV tube.
- Monitor the ECG.
- Maintain perfusing blood pressure through IV fluids.
- Increase the GFR (medication administration and fluid resuscitation).
- Closely monitor serum electrolyte levels.
- If in place, monitor dialysis machinery for potential complications.

TABLE 16-13 Normal vs Abnormal Laboratory Values in Patients with ARF

Test	Normal	Abnormal
BUN	8–23 µg/dL	> 23 µg/dL
Creatine, serum	0.6–1.2 mg/dL	> 1.2 mg/dL
Urinalysis		
• Osmolality	275–295 mOsm/kg	Varies
• Specific gravity	1.003–1.030	Varies
• BUN:creatine	10:1	> 20:1
• Fractional excretion of sodium	1%	Varies
• Urine sodium excretion (over 24 h)	43–217 mEq/24 h	Varies

Abbreviations: ARF, acute renal failure; BUN, blood urea nitrogen.

Urinalysis is critical for differentiating between the various types of ARF. Patients with prerenal ARF exhibit increased osmolality, specific gravity, and BUN:creatine levels, along with a decreased fractional excretion of sodium and urine sodium concentration. Acute tubular necrosis is framed with a nearly opposite set of findings: Urine osmolality is decreased, with an increased urine sodium concentration and fraction excretion of sodium.

Table 16-13 summarizes renal laboratory values for patients with ARF.

Renal ultrasonography is the gold standard in assessing suspected postrenal ARF. It allows for visualization of both renal calculi and the overall renal anatomy. Contrast dye is not used so as to avoid

potential complications from the dye (ie, intrarenal ARF). For this reason, ultrasonography has a distinct advantage over alternative imaging methods in a renal context. A CT scan and MRI are useful for detecting less frequent structural disorders once renal calculi have been ruled out.

Management

Initial treatment of the patient with ARF involves maintaining the ABCs and a perfusing blood pressure. Once these basics are ensured, management focuses on increasing the GFR. This management goal must be factored into the choice of all treatment modalities, but particularly medication administration and fluid resuscitation. Administration of 0.9% normal saline is indicated in dehydrated patients in an effort to treat oliguria. Diuretic administration can increase urine output, potentially increasing the GFR. Serum electrolyte levels must be monitored closely, especially potassium, in an effort to avoid complications from hyperkalemia.

Renal replacement therapy (RRT) is a frequently used treatment for ARF that has not responded to prior less invasive methods of treatment. The modality of choice remains controversial, and options include intermittent hemodialysis (IHD), continuous venovenous hemofiltration, and peritoneal dialysis (PD). IHD is the most commonly used method for both inpatient and outpatient settings. It involves the use of a dialysis machine, filter, extensive water supply, and tubing. The patient's blood is filtered by diffusion through an external semipermeable membrane and then returned to the body. Access to the patient's circulation can be obtained in different ways depending on the duration for which dialysis is required. A non-tunnelled central venous catheter is placed into either the internal jugular or the femoral vein for short-term access, but it carries with it complications such as infection and stenosis. For long-term IHD, an arteriovenous (AV) fistula is the preferred method as a result of increased blood flow, superior long-term patency, and decreased likelihood of thrombosis or infection. To form an AV fistula, a vascular surgeon anastomoses an artery and vein, most commonly in the non-dominant forearm. The typical patient receiving IHD is treated two to three times per week in 4-hour sessions. During continuous venovenous hemofiltration, a crystalloid replacement solution is added to dialysate as a plasma ultrafiltrate is removed. This method is often used when IHD is impossible as a result of severe hemodynamic instability. A convective clearance is used instead of the diffusion used in IHD, and treatment is slow and continuous, being performed over the course of 6 to 12 hours.

PD is infrequently used in the hospital setting although it can be a convenient option for chronic renal failure sufferers in the outpatient setting. This technique involves the insertion of a rubber catheter into the abdomen and the use of the peritoneum as a semipermeable membrane. A dextrose solution is injected into the peritoneal space, where it exchanges across the membrane while removing waste. The most common complication of PD is the increased risk of peritonitis following a site infection.

Chronic Renal Failure

Epidemiology and Pathophysiology

A gradual decrease in renal function over a long time interval is known as **chronic renal failure (CRF)**. The hallmark of CRF is an irreversible destruction of nephrons brought about by the continuous effects of a number of diseases, including diabetes mellitus, hypertension, and chronic infections (such as glomerulonephritis or pyelonephritis). The incidence of CRF in the United States has been steadily increasing over the past 30 years. This disease is diagnosed in more than three times as many blacks as whites. It is 40% more likely to develop in men than in women, and its incidence increases directly with age. Over time, CRF progresses to **end-stage renal disease (ESRD)**, after which dialysis is needed to maintain adequate renal function. The causes of CRF are widespread, although up to 75% of cases can be traced to long-term hypertension, glomerulonephritis, and diabetic neuropathy. A summary of causes is listed in **Table 16-14**.

The degree of severity of renal failure progresses as increasing numbers of nephrons are affected by the disease. When a nephron is compromised, surrounding nephrons are capable of working at overcapacity by increasing their GFRs. This compensatory mechanism permits the kidneys to maintain adequate function up to nephron losses of 70%. CRF is not technically present until the GFR falls below 30 mL/min. A less severe decrease in GFR (30 to 70 mL/min) is more accurately termed chronic renal insufficiency. Significant symptoms of renal failure typically do not emerge until the GFR has decreased to less than 30 mL/min. Symptoms become the most pronounced once the GRF falls below 10 mL/min, at which point the disease is classified as ESRD.

TABLE 16-14 Causes of Chronic Renal Failure
Long-term hypertension
Glomerulonephritis
Diabetic neuropathy
Chronic glomerular disease Chronic infections
Congenital abnormalities
Vascular diseases
Obstructive processes
Collagen diseases
Nephrotoxic agents
Endocrine diseases

Assessment

Signs and Symptoms

The presentation of a patient with CRF varies based on the progression of the disease and the existence of comorbidities. As the GFR decreases, waste retention begins. Many of the symptoms encountered in ARF are then seen, such as azotemia and electrolyte imbalances. Hyperkalemia can result from inadequate potassium excretion and can precipitate cardiac arrhythmias. Elevated BUN and serum creatine levels will be observed, as in ARF. Sodium and water retention may lead to congestive heart failure and edema. As renal erythropoietin synthesis decreases, anemia may develop, resulting in chronic fatigue. Hyperparathyroidism can develop from decreased renal vitamin D metabolism, causing skeletal abnormalities. Patients may exhibit polyuria as a result of the inability of the diseased nephrons to concentrate urine efficiently, a phenomenon known as isosthenuria. Metabolic acidosis frequently occurs in patients with CRF from a combination of acid retention and bicarbonate wasting and can contribute to the symptoms expressed during uremia.

The overall retention of urea and other metabolic waste in the blood leads to uremia, a condition characterized by nausea, vomiting, fatigue, anorexia, weight loss, pruritus, and altered mental status. Patients may present with jaundice if bilirubin retention occurs. Elimination of uric acid through the skin in sweat causes the uremic frost phenomenon, which is characterized by white dust on the skin. In patients with CRF, uremia usually sets in with ESRD and often coincides with the time that dialysis treatment is initiated.

Laboratory Data and Imaging

The results of lab studies in CRF and ESRD are generally parallel to the results seen in ARF. Variations may assist in distinguishing between the various causes of CRF. The most important factor in determining whether the renal failure is acute or chronic is disease duration. Comparing current elevated serum creatinine levels with a patient's readings over the previous months or years is helpful; prolonged elevation is strongly suggestive of chronic disease. If that is not available, signs of metabolic bone disease can also suggest chronic disease. Findings suggestive of this include hyperphosphatemia, hypocalcemia, elevated parathyroid hormone levels, and elevated bone alkaline phosphatase levels. Imaging studies commonly reveal decreased kidney size (< 8.5 cm) as a result of tissue atrophy.

Signs and Symptoms

Chronic Renal Failure

- Azotemia
- Electrolyte imbalances
- Hyperkalemia
- Cardiac arrhythmias
- Congestive heart failure
- Edema
- Anemia
- Chronic fatigue
- Hyperparathyroidism
- Skeletal abnormalities
- Polyuria or anuria
- Metabolic acidosis
- Uremia
- Jaundice
- Uremic frost phenomenon

Differential Diagnosis

Chronic Renal Failure

- Azotemia (increased BUN and serum creatine levels)
- Increased or decreased osmolality
- Increased specific gravity
- Increased or decreased fractional excretion of sodium and urine sodium concentration
- Acute renal failure
- Glomerulonephritis

Transport Management

Chronic Renal Failure

- Identify underlying condition.
- Treat hypertension or diabetes.
- Treat specific conditions (hyperphosphatemia, hypocalcemia, anemia).
- Be alert for and treat any electrolyte abnormalities.
- Monitor the ECG.
- Administer loop diuretics.
- Transport for dialysis.

Management

The best treatment for chronic renal failure is prevention, as there is no definitive cure once it has progressed to clinically detectable signs. Once CRF is recognized, treatment focuses on slowing the progression. Underlying conditions such as hypertension or diabetes must be controlled and managed appropriately. Specific conditions can be treated as needed: erythropoietin for anemia, phosphate binding for hyperphosphatemia, calcium supplements for hypocalcemia, and volume overload with diuretics or dialysis. Electrolyte imbalances can lead to cardiac arrhythmias, and therefore all patients with CRF should have continuous ECG monitoring. In the field, patients with CRF should also receive supplemental oxygen as required.

Peritoneal dialysis and **hemodialysis (HD)** are two options for long-term maintenance of renal function. PD introduces the dialysate solution into the abdominal cavity with a catheter, and the patient's peritoneal membrane acts as the semipermeable membrane for solute separation. HD involves vascular access through either a fistula or an arteriovenous shunt. A fistula, shunt, or arteriovenous graft is a surgically created arterial to venous vessel anastomosis tunneled through the subcutaneous tissue. Solute separation occurs across an artificial semipermeable membrane within a machine. The decision of whether or not to begin renal replacement therapy such as dialysis occurs on a case-by-case basis and does not depend on any specific urea nitrogen or creatinine level. It is typically instated once other treatments have been unsuccessful in alleviating symptoms, such as protein restriction and antihypertensive medications. If symptoms such as anorexia, nausea, encephalitis, pericarditis, or uremia continue even with treatment, the patient should be prepared for dialysis.

For critically ill patients who are unable to tolerate HD or PD but require RRT, a variety of slower, gentler RRTs are available. These include continuous renal replacement therapy, slow continuous ultrafiltration, and a variety of other therapies all provided by dialysis-like machines connected to large-bore venous access cannulas designed to tolerate therapeutic flow rates. These access cannulas can also be used for HD and can be either temporary or tunneled (implanted) dual-lumen catheters. Patients receiving RRT need to be disconnected from their machine and have their catheters flushed to maintain patency during transport. Portable RRT machines able to continue therapy during patient transport are currently not available.

At times the CCTP may encounter dialysis patients who require IV access or even discontinuation of ongoing dialysis as a result of complications during transport. Patients with CRF who are on dialysis typically have poor peripheral vasculature, and obtaining access may be extremely difficult. It is important to ensure that patent venous access is available prior to initiating transport. A fistula, shunt, or arteriovenous graft should never be routinely used for vascular access. An extremity with a fistula should not be used to obtain a blood pressure reading, draw blood, obtain peripheral IV access, or obtain fingerstick blood glucose specimens. If other forms of venous access are lost during transport and a patient's condition deteriorates into extremis (a grave condition) or the patient experiences

cardiopulmonary arrest, the CCTP should consider using a fistula only as a last resort. The likelihood of infection or damage to the fistula is so significant that intraosseous or central venous access (such as external jugular access) should always be attempted before resorting to the use of a fistula.

Complications often arise during dialysis as well; some complications can be treated during dialysis, whereas others may become so severe that dialysis must be discontinued. The most common complication is hypotension, which can usually be treated with small (100- to 250-mL) isotonic saline boluses. Muscle cramping is another frequent complication and is likely associated with rapid volume removal from the patient. Cramps too can be treated with small saline boluses. Anaphylactic reactions to the dialysis machinery are uncommon now, but if a reaction does occur, the dialysis machinery should be immediately removed and the patient treated for anaphylaxis. Severe episodes of hypotension may also necessitate that dialysis be stopped during treatment. If an emergency requires that the dialysis machinery be removed, it should be performed by a provider trained and experienced in operating the equipment in use. If a provider familiar with the equipment is not available, disconnection should be done following the manufacturer's instructions. All medical device manufacturers maintain clinicians on call 24 hours a day to assist in troubleshooting problems. The telephone contact numbers are often prominently displayed on the critical care machinery.

Maintenance doses of many drugs for patients on dialysis may need to be reduced, although the loading doses of drugs that would be given prehospitally do not need to be changed. Certain medications, such as meperidine, and NSAIDs should be avoided though, as should oral antidiabetic agents. The list of medications that need to be reduced in dosage for patients on dialysis is extensive (refer to a publication on the specific topic for a complete list).

Kidney transplantation is the only definitive cure for CRF and has demonstrated increasing success during the past 50 years since its initiation. The 1-year survival rate has been reported to be as high as 95%. Kidney transplantation is the most common transplantation surgery in the United States, accounting for more than 50% of all organ transplantations performed in this country. Unfortunately, the supply of kidneys is lower than the demand for these organs, and many patients remain restricted to dialysis-related therapeutic options.

Urinary Tract Infections

Bacterial flora that live symbiotically in the GI system can cause considerable damage when they are introduced into the urinary system. A **urinary tract infection (UTI)** typically begins in the urethra, because this location is the most proximal to the external environment. The incidence of UTIs in females is higher than in males owing to anatomic differences between the two sexes, such as the shorter urethra in females. More than 20% of females will experience a UTI at some point in their lives. Patients with impaired urination from an obstruction or nervous system disruption are predisposed to UTIs, because urine flow normally cleanses the tracts of pathogens. Patients with bladder catheters are also at a high risk for infection.

Lower UTIs include infections of the prostate, urethra, and bladder, with the latter two infections known as urethritis and cystitis, respectively. Lower UTIs, such as **pyelonephritis**, are more common and less severe than upper UTIs in the kidney. As many as 90% of infections are caused by *Escherichia coli* bacteria, which are normal inhabitants of the GI tract.

Inflammation secondary to the infection causes a burning pain exacerbated by urination, at times accompanied by foul-smelling urine. Pyelonephritis symptoms are more significant and include fever, chills, lower back pain, and nausea with vomiting. Chronic pyelonephritis can develop in patients with underlying renal abnormalities.

The majority of both lower and upper UTIs are treated successfully with antibiotics. On occasion,

surgery may be required to remove an intrarenal or perinephric abscess that forms during the course of pyelonephritis. In more severe cases of pyelonephritis, IV antibiotics may be required, and the CCTP may have to manage the administration during interfacility transports.

Testicular Torsion

Normally, the tunica vaginalis secures the testes inside the scrotum, preventing any excessive movement or rotation. In about 12% of the male population, this attachment is misaligned, allowing some degree of free testicular movement, known as the "bell clapper" deformity. These patients are susceptible to **testicular torsion**, a condition in which one or both of the testes rotate to the point of occluding their blood supply **Figure 16-34**. A rotation of 720°, or two full rotations of the testicle around the axis of the testicular artery and vein, is required to fully tamponade the vessel and cause a resultant buildup of waste products within the testes and scrotal swelling. This condition is extremely painful, and immediate surgical intervention is required to salvage the testis, as necrosis will set in within 12 to 24 hours. The CCTP must be able to recognize the symptoms and initiate rapid transport to an appropriate facility or notify the receiving facility of suspicions upon arrival. Most clinicians prefer that analgesics are not administered prior to diagnosis, although afterwards pain relief should be administered.

Patients will occasionally present with nausea, vomiting, and abdominal pain along with testicular pain. Testicular torsion is most often seen in adolescent males, with the over-whelming majority of cases occurring in males younger than 30 years. Along with developmental abnormality, trauma and exercise are known precursors to testicular torsion.

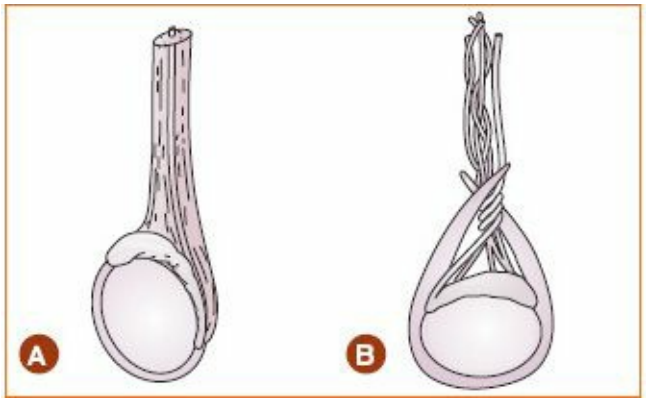


Figure 16-34 **A.** The normal testicle. **B.** Testicular torsion.

The testes are also susceptible to torsion prior to development of the tunica vaginalis. This condition, known as extravaginal torsion, is seen in prenatal males as a result of improper spermatic cord attachment. Typically the testis is unviable if the condition developed in utero, and surgical intervention is not an emergency.

Penile Fracture

During penile erection, the corpora cavernosa and corpus spongiosum are engorged with blood, distending the erectile tissue to the point of elasticity loss. Direct blunt trauma to the penis in this state can result in **penile fracture**. The fracture here is actually a rupture of one or more of the vascular spaces within the penis, mimicking the appearance of a long bone fracture. This event most commonly occurs during sexual intercourse, with a cracking or popping noise being heard at the time of injury, along with the development of moderate to severe pain. The penis will appear swollen and deformed by hematoma formation. Urethral involvement is observed in some cases and should be considered if hematuria exists.

No lab or imaging studies are required for the diagnosis of a penile fracture. Initially, cold

compresses should be placed on the area of injury. Definitive treatment involves reconstructive surgery on the penis. The main treatment goals include restoration of urinary and reproductive functioning. Foley catheter placement is required in cases of urethral injury to allow for proper healing, along with prophylactic antibiotics. In general, penile fracture is a rare condition, but it has a good prognosis following prompt recognition and surgical treatment.

Priapism

Priapism is a prolonged erection of the penis that can be painful and damaging to tissues in the immediate area. Spinal cord transection is commonly cited as the frequent precursor to priapism, but nontraumatic causes exist as well.

The two major classifications of priapism are arterial high-flow and veno-occlusive priapism. Arterial high-flow priapism typically occurs secondary to penile injury, with vessel spasms or clot formation inhibiting blood return. Veno-occlusive priapism, the more painful of the two conditions, is most often seen in patients with sickle cell anemia and certain medication use. Drugs that place men at risk include a number of prescription pharmaceuticals, including psychotropic medications, calcium-channel blockers, and anticoagulants. Intracavernous erectile dysfunction drugs such as papaverine, phentolamine, and prostaglandin E1 are among the most common causes of priapism. There are very few reported cases of priapism from the more popular selective cyclic guanosine ß, 5′-monophosphate inhibitors such as sildenafil citrate (Viagra).

Initial treatment is application of a cold pack followed by administration of either a smooth-muscle relaxant or vasoconstrictive medication. Priapism is infrequently encountered and usually correctible, with the major complication being impotence.

Maintenance Tubes

A variety of tubes may be encountered or placed by the CCPT while caring for a patient with a gastrointestinal or genitourinary condition. The following sections focus on two major types: feeding tubes and drainage tubes **Table 16-15**.

Feeding Tubes

Proper nutrition is necessary for life and must be provided parentally to patients who are extensively incapacitated by either disability or sedation. Enteral nutrition, commonly called tube feeding, can be administered in steps, depending on its expected duration.

Enteral Nutrition

Perhaps the largest obstacle to food intake in the incapacitated patient is the inability to safely and effectively swallow. This can be circumvented by placement of a simple NG tube, which can be used to administer food into the stomach or to remove food. These minimally invasive tubes are often inserted through the nasal orifice with the assistance of the patient in preparation for a short-term event during which temporary enteral feeding is required.

For longer-term situations, a gastrostomy tube (G tube) can be inserted. These tubes are also referred to as **percutaneous endoscopic gastrostomy (PEG) tubes** because of their insertion technique. PEG tubes are placed through a small surgically created opening from the stomach, through the peritoneum, to the abdominal wall **Figure 16-35A**.

Alternatively, a **jejunostomy tube (J tube)** can be introduced directly into the jejunum through a comparable technique, known as a percutaneous endoscopic jejunostomy **Figure 16-35B**. J tubes possess

an advantage over their PEG counterparts in that aspiration risk is largely decreased because food intake occurs distal to the pyloric sphincter. Unfortunately, J tubes are also associated with a higher insertion complication rate owing to the less stable small intestinal wall compared to the thicker stomach lining, which serves to anchor a PEG tube more securely.

TABLE 16-15 GI/GU Maintenance Tubes

Type of Tube	Examples
Feeding tube	NG tube Percutaneous endoscopic gastrostomy tube J tube Percutaneous endoscopic gastrostomy-J tube Total parenteral nutrition
Drainage tube	Straight catheter Foley catheter Suprapubic tube External condom catheter Colostomy Ileostomy Ileoanal anastomosis Kock pouch Ileal conduit urostomy Colon conduit urostomy Jackson-Pratt drain Hemovac drain Davol drain T tube

Abbreviations: GI, gastrointestinal; GU, genitourinary; NG, nasogastric.

A newer procedure known as a PEG-J tube lessens this risk while providing the benefit of decreased aspiration. During this procedure, a PEG tube is placed through the abdominal wall but is then threaded through the duodenum and into the jejunum.

Liquid nutritional products can be administered to the patient through any of these tubes by either a rate-controlled pump or syringe boluses. Proper functioning is confirmed by a lack of pain or resistance while pushing the syringe upon food administration.

Aspiration is the most common complication from a feeding tube that could develop into a life-threatening situation; this problem is easily averted by sitting the patient upright during feeding or vomiting. Bleeding may occur at the insertion site if the tube is pulled on or traumatically removed. Significant hemorrhage from this situation is unlikely, and bleeding is typically self-limited. A third complication involves infection around the insertion site, which may be observed as a reddening or swelling in the immediate vicinity.

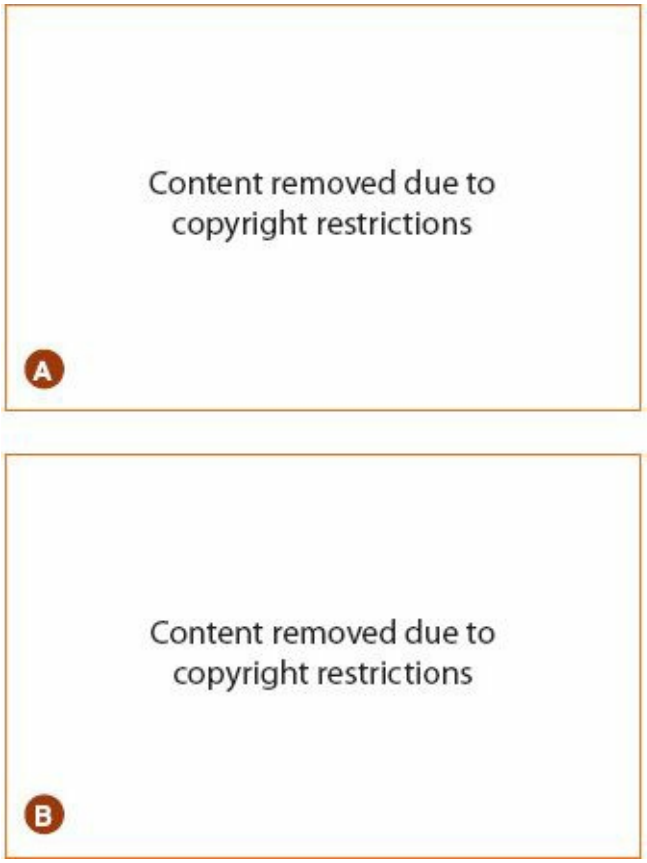


Figure 16-35 **A.** Bard® percutaneous endoscopic gastronomy (PEG) tube. **B.** Bard® jejunostomy (J) tube. © C. R. Bard, Inc.

If the feeding tube becomes clogged, a 30- to 50-mL sterile warm water bolus can be used to clear the blockage. If the tube is removed unexpectedly, the site should be covered with an occlusive dressing until it can be properly replaced. Unmanageable blockage or a removal of the PEG or J tube is not of major concern in the short-term care of the patient, as a brief lapse in feeding can be accounted for later.

Total Parenteral Nutrition

Total parenteral nutrition is an alternative feeding method used when the GI tract is not functioning adequately. During TPN, all required nutrients are administered intravenously to the patient. Typically a central vein is used for administration to prevent osmotic fluid shifts, although some preparations permit peripheral vein usage. Medications are often added to the TPN solution during its creation, but they should not be introduced once that process is complete owing to the potential for contamination. TPN should be used in moderation only when enteral feeding is not an option as a result of the significant GI atrophy that occurs during dormancy.

Patients receiving TPN are at high risk for an air embolus and thrombosis. An air embolus is most likely to occur during line changing or an unexpected disruption. This situation has a high mortality and must be aggressively treated with high-flow oxygen, occlusive dressing placement, and movement of the patient into a left-sided Trendelenburg position. Thrombosis is treated by removing the catheter, followed by anticoagulation and thrombolytic therapy. Continuous monitoring for metabolic abnormalities must be done on the patient receiving TPN, as well as monitoring for electrolyte imbalances that can easily arise.

Management

When transporting a patient with a feeding tube, contact medical direction if the patient has vomited or has diarrhea or a distended abdomen.

Transport Management

Patients With Feeding Tubes

- Examine insertion site for signs of infection, such as redness or swelling.
- Assess for pain at insertion site.
- Assess for any leakage or blockage of tube.
- Keep the patient in a position of comfort.
- Provide supportive care as required.

Drainage Tubes

The digestive system has equally important input and output functions, and the tubes placed in the critically ill patient must achieve both of these purposes. Just as various feeding tubes exist to introduce food to the body that cannot independently consume it, drainage tubes exist to remove waste when it is being retained. Waste products of human metabolism are manifested in urine and feces, which are parenterally removed through Foley catheters and ostomies, respectively.

Urinary Catheters

Types of Urinary Catheters

Different methods of urine collection exist, each with its own advantages and disadvantages.

Straight catheters, often called intermittent catheters, are designed for temporary use and are frequently self-inserted by the patient. They allow for the patient to drain residual bladder contents that could lead to infection. These catheters are associated with less inflammation than catheters intended for longer-term use, but the drainage process must be repeated three or more times per day.

Indwelling catheters, also known as Foley catheters, are used as a more permanent solution to urine retention or incontinence. These pliable rubber catheters are introduced into the bladder via the urethra. A balloon is inflated with 5 mL of saline inside the bladder to prevent inadvertent removal of the catheter. Foley catheters provide complete management of urine for bedridden or incapacitated patients, with minimal upkeep.

When surgery or trauma makes urethral catheter introduction unfavorable, a suprapubic tube may be inserted. This urine collection tube is placed directly into the bladder through the abdominal wall by way of a surgically created stoma.

Male patients have the option of using external condom catheters as well. These catheters are less prone to complications and are a good alternative to diapers.

Each of the four types of urine-collection catheters functions in a similar way; that is, each drains into a bag that allows for clean collection, observation, and measurement of urine output. Two types of collection bags are frequently used—a leg bag that straps to the leg underneath clothes, allowing for greater mobility with a discrete presence, and a down drain bag that hangs on the side of the patient's bed. Larger down drain bags are more commonly used in hospitals and nursing homes. The drains function by gravity, so it is important to ensure they are hung below the patient.

Complications of Urinary Catheters

Common complications of urine collection catheters include clogging, infection, pain, leakage, and dislodgement. Clogging can typically be cleared with a small fluid bolus, although it may indicate dislodgement. Dislodgement is the largest concern during transport. Dislodgement and leakage are corrected by replacing the catheter into the bladder; they are often a result of selecting the improper size

of catheter. Signs of infection—such as pain, irritation, and reddening around the urethral orifice of the insertion site—should be treated promptly with antibiotics.

Transport Management

Patients With Urinary Catheters

- If clogging occurs, clear the catheter with a small fluid bolus.
- If dislodgement or leakage occurs, replace the catheter. A different size may be needed.
- Treat signs of infection with antibiotics.

Management

It would be unlikely for urinary catheter placement to occur during transport, but the procedure is covered here in the event that the CCTP performs it prior to transport. When preparing to place a urinary catheter, gather the following equipment:

- Disposable sterile bladder catheter kit
- Water-soluble lubricant
- Drapes
- Cleansing solution
- Sterile gloves
- Prefilled syringe containing sterile water
- Correct size urinary catheter
- Clamp
- Connecting tubing
- Collecting bag

Indications for urinary catheter placement include drainage of the urinary bladder to precisely measure urine output, incontinence, and acting as an adjunct to diuretic therapy. Contraindications include blood at the meatus (opening) of a trauma patient and a history of urethral stricture (narrowing). When transporting a patient with a urinary catheter by air, air medical considerations include filling the balloon with water. The amount should be equivalent to 10 mL of air.

The steps to place a female urinary catheter are shown in **Skill Drill 16-3** as follows:

1. Prepare equipment.
2. Use BSI precautions.
3. Place the patient in the supine position, with hips flexed and abducted.
4. Using a sterile technique, open the kit and prepare the equipment.
5. Test the balloon on the indwelling catheter by injecting it with sterile water or saline.
6. Position the drapes.
7. Lubricate the catheter.
8. Cleanse the sides of the labia and meatus with a chlorhexidine 2% solution.

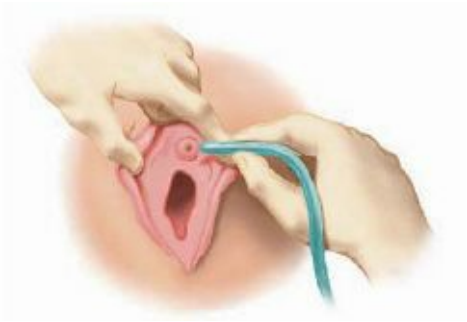
9. Insert the catheter into the meatus gently until urine flow begins **Step 1**.
10. Fill the fluid retention balloon until secure **Step 2**.
11. Connect the open end of the catheter to the drainage tube and collection bag **Step 3**.
12. Secure the catheter to the patient's leg with tape.

The steps to place a male urinary catheter are shown in **Skill Drill 16-4** as follows:

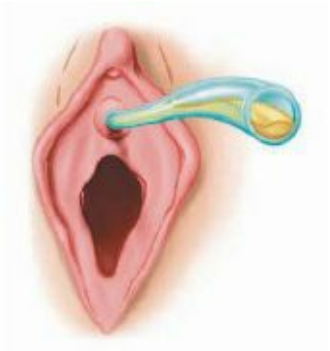
1. Prepare the equipment.
2. Use BSI precautions.
3. Place the patient in the supine position.
4. Using a sterile technique, open the kit and prepare the equipment.
5. Position the drapes.
6. Lubricate the catheter.
7. Cleanse the penis from head to base.
8. Insert the catheter gently until urine flow begins. Once urine is observed, gently advance the catheter an additional 2″ to 3″ **Step 1**.
9. Fill the fluid retention balloon until secure **Step 2**.
10. Connect the open end of the catheter to the drainage tube and collection bag **Step 3**.
11. Secure the catheter to the patient's leg with tape.

Skill Drill 16-3

Placing a Female Urinary Catheter



1 Insert the catheter into the meatus gently until urine flow begins.

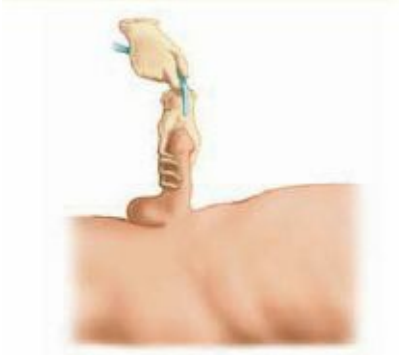


2 Fill the fluid retention balloon until secure.

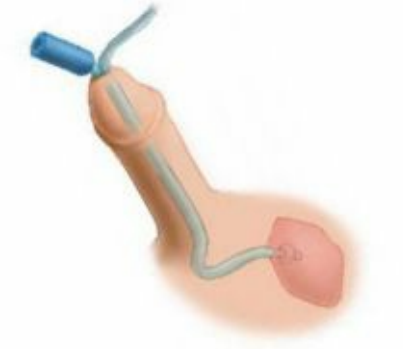
3 Connect the open end of the catheter to the drainage tube and collection bag.

Skill Drill 16-4

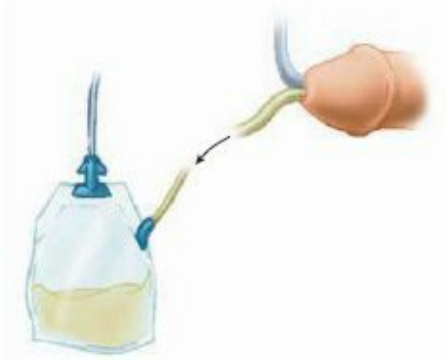
Placing a Male Urinary Catheter



1 Insert the catheter gently until urine flow begins. Once urine is observed, gently advance the catheter an additional 2″ to 3″.



2 Fill the fluid retention balloon until secure.



3 Connect the open end of the catheter to the drainage tube and collection bag.

Ostomies

Types of Ostomies

An **ostomy** is a surgically created opening of the GI tract through the abdomen that allows for waste removal. The ostomy may originate in the ileum of the small intestine (ileostomy) or anywhere in the large intestine (colostomy), depending on which segments of the GI tract must be bypassed. Urostomies may be created as well to drain urine directly from the urinary tract when a distal portion must be avoided as a result of disease or trauma. Indications for an ostomy include bowel injury or disease, bladder injury or disease, and congenital anomalies, including cancer.

Colostomies **Figure 16-36** and ileostomies drain intestinal contents out of the GI tract and into a collection bag. The drainage from a colonoscopy is more solid than that from an ileostomy because the material has progressed farther through the digestive process. The drainage is collected in a bag attached to the opening and strapped to the patient's body. Sometimes a section of small intestine is surgically modified to create an internal collection site called a Kock pouch. In an ileoanal anastomosis, also known as a J pouch, the anus is preserved, allowing it to function in fecal collection. These methods are beneficial to the patient because they allow less visible collection methods and offer more convenient waste disposal management.

Urostomies divert urine away from the bladder by creating a passageway from the ureter to an abdominal stoma through a piece of ileum or large intestine that is surgically separated from the GI tract. When the ileum is used, it is called an ileal conduit; when the large intestine is used, it is called a colon conduit. An external collection pouch is attached to the stoma and secured to the patient's body.

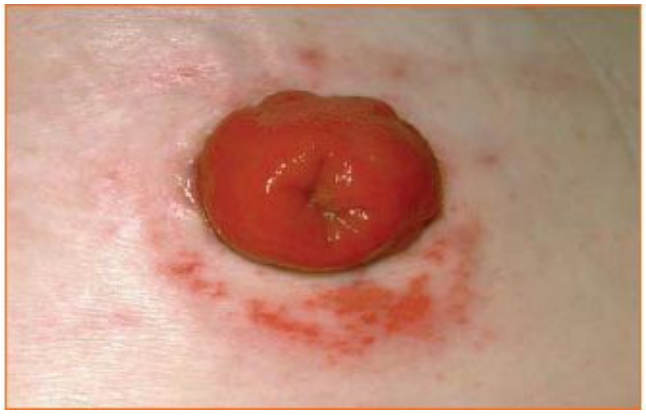


Figure 16-36 A colostomy.

Management

Complications of ostomies include the following possibilities:

- Obstruction
- Constipation (with colostomies)
- Diarrhea
- Dehydration
- Flatulence
- Leakage
- Tissue necrosis
- Detachment
- Prolapse
- Infection

The patient with an ostomy should be monitored for signs of tissue necrosis and infection, which, if detected, may warrant treatments ranging from antibiotic therapy to surgical intervention. Normal assessment findings for a patient with an ostomy include a pain-free insertion site free of redness or swelling. Detachment or prolapse of intestinal tissue from the stoma may require bleeding control and occlusive dressings as well as surgical reconstruction. When assessing the patient with an ostomy, expose the insertion site to examine for redness, swelling, or leakage. If any of these are found, notify the receiving facility upon arrival. Always ask the patient if the ostomy is functioning normally, without either leakage or obstruction.

The next paragraphs discuss two skills: emptying and replacing an ostomy pouch. An ileostomy pouch needs to be emptied 4 to 6 times a day. A colostomy pouch needs to be emptied 1 or more times per day. Laxatives should not be used by patients with ileostomies, and adequate daily fluid replacement (such as 6 to 8 glasses of water) is a necessity. In addition, the patient should not use soaps that contain baby oil, cold cream, or perfumes.

When preparing to empty or replace an ostomy, gather the following equipment:

- Stoma measuring guide or pattern
- Scissors
- New pouch
- Soft wash cloth
- Soap and water
- Razor for shaving hair on abdomen
- Biohazard bag for soiled pouch

Skill Drill 16-5 shows the steps for emptying an ostomy pouch. The pouch should be emptied when it is one third to half full.

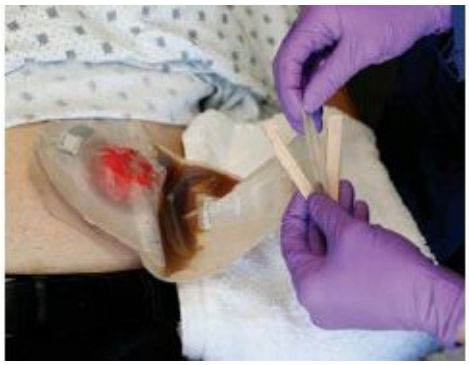
1. Remove the clamp that keeps the pouch closed **Step 1**.
2. If the patient is supine, lift the pouch and position the opening over a waste collection container **Step 2**. If possible, have the patient lie on his or her side with the pouch opening positioned over a waste collection container.

3. Slide your fingers down the outside of the pouch to squeeze the contents out of the pouch.
4. Clean the inside of the pouch opening with a piece of toilet paper.
5. Then rinse out the pouch with room-temperature water using a rubber ear (bulb) syringe.
6. Put the clamp back onto the pouch to close it **Step 3**.

Although ostomy pouches typically only need to be changed every few days, the CCTP may be required to change one under certain circumstances. If there is a leak or rupture of the bag during transport, the pouch must be removed and replaced if the equipment is available. Less emergent reasons to change a pouch include inflammation, pain, or irritation around the insertion site. In such instances, changing the pouch can typically be delayed until arrival at the receiving facility.

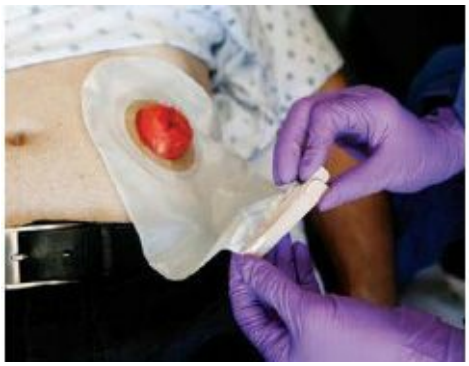
Skill Drill 16-5

Emptying an Ostomy Pouch



1 Remove the clamp that keeps the pouch closed.

2 If patient is supine, lift the pouch and position the opening over a waste collection container.



3 Slide your fingers down the outside of the pouch to squeeze the contents out of the pouch. Clean the inside of the pouch opening with a piece of toilet paper. Then rinse out the pouch with room-temperature water using a rubber ear (bulb) syringe. Put the clamp back onto the pouch to close it.

Skill Drill 16-6 shows the steps for replacing an ostomy pouch.

1. Use BSI precautions.
2. Using the measuring guide or pattern, trace the correct opening onto the skin barrier of the new pouch, then cut out the opening you traced **Step 1**.
3. Take off the paper backing that covers the adhesive on the skin barrier of the new pouch **Step 2**.
4. Lay the pouch near you with the adhesive side up.
5. Empty the pouch the patient is wearing **Step 3** (see Skill Drill 16-5 for step-by-step instructions).
6. Remove the pouch by pushing down on the abdomen with the fingers of one hand and peel off the pouch with the other hand **Step 4**.
7. Remove the clamp from the old pouch.
8. Dispose of the old pouch by placing it into a biohazard bag **Step 5**.
9. Wash thoroughly around the stoma with mild soap and warm water **Step 6**.
10. Rinse and dry with a soft cloth. A small amount of bleeding may be normal.
11. Center the new pouch opening over the stoma **Step 7**.
12. Press the skin barrier wafer onto the abdomen, removing all wrinkles or creases in the wafer **Step 8**.
13. Put the clamp from the old pouch onto the opening of the new pouch **Step 9**.

Specific Drainage Tubes

After major abdominal surgery, a number of drainage tubes are placed to both assist in and evaluate the recovery process. **Jackson-Pratt drains** **Figure 16-37** are inserted to remove postoperative buildups of fluid and blood in the abdominal cavity. Prevention of fluid accumulation greatly decreases the risk of infection. The drain is controlled by the patient, who squeezes a bulb to induce suction. A flexible tube connected to the bulb travels through the surgical incision and into the wound site.

Similar surgical drainage tubes include the Hemovac and Davol drains. Both of these draining devices operate similarly to the Jackson-Pratt tube, with the only difference being bulb design.

During liver transplant surgery, a **T tube** is placed to monitor bile drainage from the gallbladder. After the drainage bag has been removed, the tube may remain in place to facilitate examination of the biliary tract through a T-tube cholangiogram test.

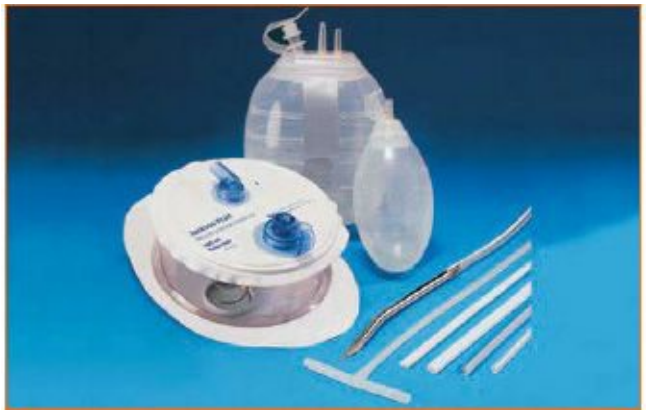


Figure 16-37 A Jackson-Pratt drain (in background) and a Hemovac drain (with blue connectors).

These drainage tubes can frequently be obstructed by blood clots, which can be usually cleared by digitally manipulating the tube. Dislodgement can slow the healing process and greatly increase the likelihood of infection, and the drain may need to be replaced through surgical means. Bleeding may occur from the wound site after dislodgement and should be controlled. The wound site and sutures should be monitored for any signs of infection or improper healing.

In-flight Considerations for Maintenance Tubes

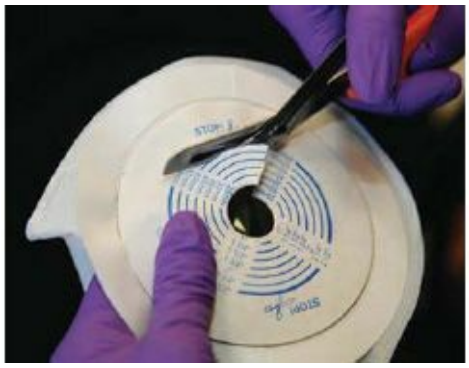
In-flight complications need to be considered for the numerous drainage systems that include closed bags containing air. A decrease in air pressure accompanies increases in altitude. Gas volume increases proportionally to the decrease in pressure, which could lead to distention of drainage devices. Although fixed-wing aircraft typically incorporate pressure-controlling mechanisms, preparations should always be made in the event of equipment failure. Controlling air pressure on rotor-wing aircraft may be more difficult because altitudes may change rapidly and often.

Excess air should be vented from any drainage bag such as a colostomy or urostomy prior to flight in anticipation of changes in air pressure. Further venting may be required during transport, and the CCTP should be sure that the specific device in use can accommodate this manipulation. Similar preparations must be made for Jackson-Pratt and other surgical drainage devices. Be sure to unclamp any feeding or drainage catheters prior to departure, and ensure that they remain unclamped for the duration of travel.

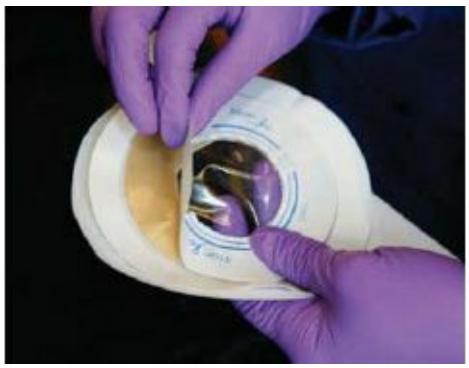
Air transport should be postponed or avoided for any patient with a bowel obstruction if at all possible. The changes in gas pressure will distend the obstructed bowel, leading to significant pain and possible perforation. If transport is unavoidable, measures should be taken to provide for air escape proximal to the obstruction.

Skill Drill 16-6

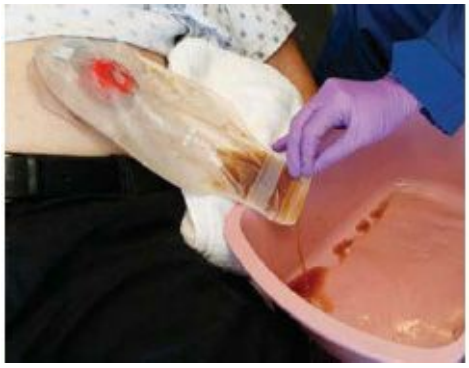
Replacing an Ostomy Pouch



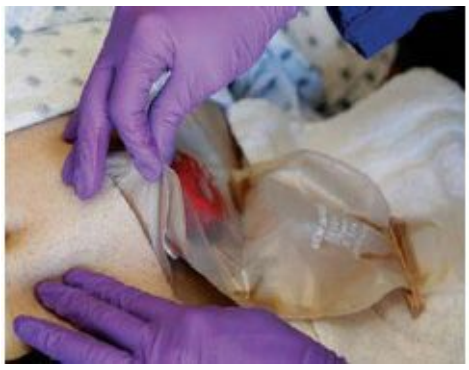
1 Use BSI precautions. Using the measuring guide or pattern, trace the correct opening onto the skin barrier of the new pouch, then cut out the opening you traced.



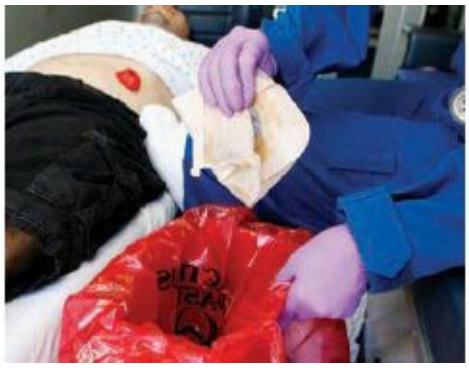
2 Take off the paper backing that covers the adhesive on the skin barrier of the new pouch.



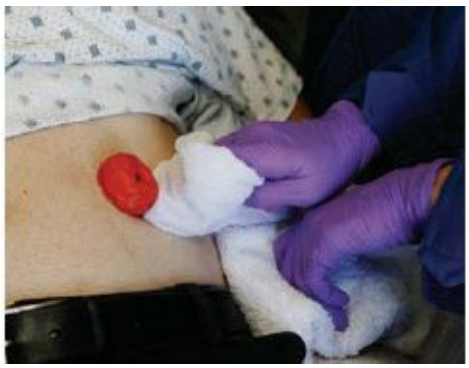
3 Empty the pouch the patient is wearing.



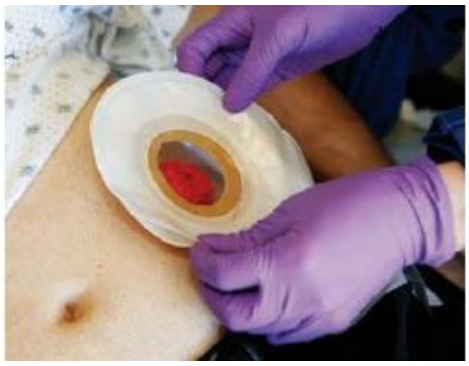
4 Remove the pouch by pushing down on the abdomen with the fingers of one hand and peel off the pouch with the other hand.



5 Remove the clamp from the old pouch. Dispose of the old pouch by placing it into a biohazard bag.

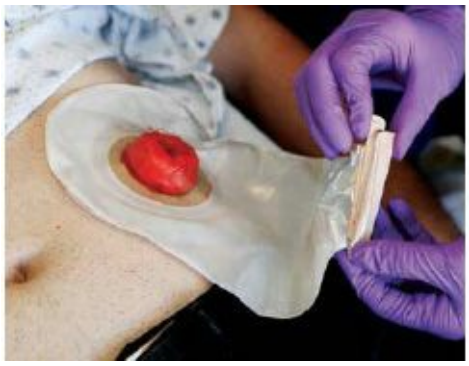


6 Wash thoroughly around the stoma with mild soap and warm water. Rinse and dry with a soft cloth.



7 Center the new pouch opening over the stoma.

8 Press the skin barrier wafer onto the abdomen, removing all wrinkles or creases in the wafer.



9 Put the clamp from the old pouch onto the opening of the new pouch.

Metabolic Regulation of Acid-Base Status

Human bodies demonstrate the amazing ability to adapt and function in extreme environmental conditions. From the peaks of mountains, to great depths in the oceans, in hot, arid deserts, remote arctic locations, and the weightless expanse of space, human activities continue. Despite this incredible ability to adapt to outside environmental changes, human bodies require a carefully controlled internal environment for cells to function, organs to perform, and life to continue.

Significant alterations of oxygen and carbon dioxide levels, electrolyte concentration, temperature, glucose levels, and acid-base status will impair cell, tissue, and organ function, eventually leading to death. CCTPs must manage any of these alterations resulting from illness, injury, or extrinsic factors to save lives and improve chances for patient recovery.

Acid-base status is tightly controlled by numerous organs, receptors, and **buffer** systems. Dysfunction of any of this vast array of cells, tissues, organs, and body systems will disrupt this carefully maintained balance and further compromise cell, tissue, and organ function throughout the body. Although the human body can tolerate large quantities of exogenous acids and bases, widespread damage occurs when the body's capacity to sequester, neutralize, and excrete these substances is exceeded. Metabolic alterations of acid-base status are common among a variety of patient conditions discussed elsewhere in this text, such as trauma, toxicology, respiratory, cardiac, endocrine, neurologic, and environmental emergencies, to name a few. Detailed discussion of respiratory acidosis/respiratory alkalosis can be found in Chapter 6.

Acid-Base Physiology

Hydrogen ions (H^+) are single protons released from a hydrogen atom that become an acid when placed in solution. A base (also basic or alkaline) substance has the ability to accept a free H^+. The relationship of acids to bases within the body is the pH value that represents the concentration of free H^+ present. Because additional basic substances are present, they become bound with free H^+, decreasing the amount of free H^+. The normal extracellular fluid pH ranges from 7.36 to 7.44 or 7.3 to 7.5, depending on the reference source. The pH value is inversely related to the amount of free H^+ present. As the free H^+ concentration increases, the pH value decreases (acidosis). As the free H^+ concentration decreases, the pH value increases (alkalosis). Cell protein activity becomes impaired when the pH falls out of the normal range. Cell death begins to occur when the pH falls below 6.8 or increases above 7.8.

Acids and bases are both absorbed into the body from dietary sources. Additionally, H^+ are produced during various metabolic processes, normally creating 50 to 100 mEq each day. Altered

cellular metabolism, changes in excretion patterns, or exposure to various exogenous substances can prompt wide variations of extracellular fluid pH. Intracellular function is altered when excess H^+ from extracellular fluid enter the cells through a **concentration gradient**. This concentration gradient is the dominant mode of cellular exchange with extracellular fluids.

Intrinsic Regulation of Acid/Base

Three principle mechanisms are used to maintain a physiologic acid-base status within the body. Chemical buffers sequester excess H^+ to maintain a normal pH until they are released to offset an increasing pH when excessive bases are present. The respiratory system controls extracellular PCO_2 concentration, which affects carbonic acid (H_2CO_3) and ultimately H^+ concentration. Finally, the kidneys can either excrete acidic or alkalinic (basic) urine to balance whole-body H^+ concentrations.

Chemical Buffer Systems

The human body uses several buffer systems to mitigate the effects of vast fluctuations or loads with acids and bases. Any substance that can alternately bind or release H^+ depending on outside conditions is a buffer. Buffer systems begin activity immediately.

The **bicarbonate–carbonic acid buffer system** is the principle extracellular buffer system. This system operates primarily in the lungs and kidneys, where carbonic anhydrase is present to stimulate the chemical reaction. Carbon dioxide and water (H_2O) are reversibly converted into H_2CO_3 (carbonic acid), which in turn is reversibly converted into H^+ and HCO_3^- (bicarbonate). The reaction reverses itself when large quantities of H^+ are present, ultimately turning excess H^+ into H_2O and carbon dioxide (CO_2), which can be easily excreted.

$$H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow CO_2 + H_2O$$

The bicarbonate–carbonic acid buffer system is linked to the lungs and kidneys because these two organs can alter the concentrations of CO_2 and HCO_3^-, respectively, thus affecting the concentration of the remaining components and the direction of these reactions **Figure 16-38**. This system allows the body to mitigate the effects of sudden acid or alkali loads without drastic alterations in extracellular (and ultimately intracellular) pH.

The **phosphate buffer system** operates similar to the bicarbonate–carbonic acid buffer system in that it allows only small pH changes following large variations in the presence of free H^+. This system functions primarily in the renal tubules and intracellular fluids to convert strong acids or bases into weak acids or weak bases, allowing only a minimal effect on overall pH. Phosphate buffers work best in areas of the body with a normally lower pH, such as the renal tubules.

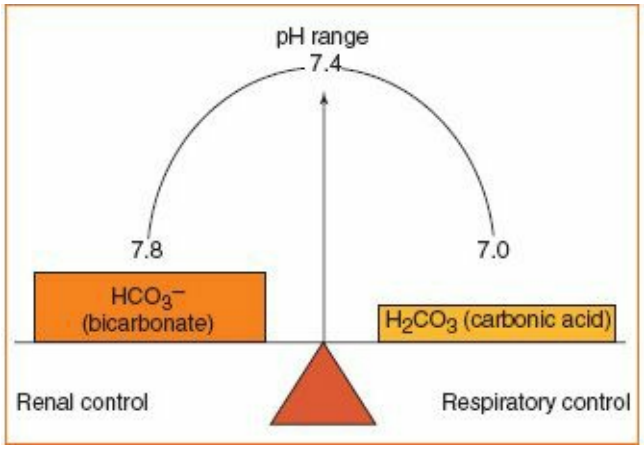


Figure 16-38 The balance of bicarbonate and carbonic acid shifts if either the renal or respiratory system fails, leading to an imbalance.

Hemoglobin, certain bone tissues, and other proteins also act as chemical buffers. Hemoglobin molecules, especially deoxygenated, can absorb a hydrogen ion, and ultimately buffer six times more than other plasma proteins. Venous blood exhibits a slightly lower pH than arterial blood. Other protein buffers take several hours, but provide the majority of total-body buffering capability. Bone buffers also contribute by absorbing and releasing excess H^+ when needed.

Respiratory Influence on pH

Carbon dioxide is a major determinant of acid-base status. Carbon dioxide is produced during normal metabolism and must be continuously excreted. The concentration of CO_2 is directly related to H^+ concentration and inversely related to extracellular fluid pH. In acidic states, alveolar ventilation is automatically increased, resulting in decreased H^+ concentrations (and increased pH) in approximately 3 to 12 minutes. Respiratory adjustment will not completely resolve a primarily metabolic alteration in pH. Respiratory compensation will bridge the delay until renal compensatory mechanisms have a chance to commence, within several hours or up to 7 to 10 days. Any increase in alveolar ventilation above baseline will conversely raise the pH and decrease the available concentration of free H^+. Impaired respiration/ventilation adversely affects acid-base status. A disruption of ventilation for even several minutes has the potential to cause catastrophic acidosis, directly from the retention of CO_2. As mentioned previously, respiratory acidosis and respiratory alkalosis are discussed in depth in Chapter 6.

Renal Influence on pH

The kidneys provide the body's last major line of defense against the catastrophic effects of large quantities of acids or bases. Kidneys excrete either acidic or basic urine depending on the pH of the body. In severe states, kidneys can excrete 500 mEq of H^+ per day, taking 7 to 10 days to reach this capability.

To compensate for a metabolic or respiratory alkalosis, the kidneys allow HCO_3^- to be excreted into the urine. This action decreases the available extracellular fluid HCO_3^-, increases the available free H^+ concentration, and lowers the pH.

In acidic states, the kidneys have three primary mechanisms. Kidneys directly excrete H^+ into the urine when excess H^+ ions are present. Kidneys can reabsorb previously filtered HCO_3^- and return it into circulation, which binds with excess H^+ and corrects all or part of the acidosis. Additionally, kidneys produce additional HCO_3^- to offset excess H^+.

TABLE 16-16 Normal Arterial Blood Gas and Venous Blood Gas Values

Value	Arterial Blood Gas	Venous Blood Gas
pH	7.35–7.45	7.32–7.43
P_{CO_2}	35–45 mm Hg	38–50 mm Hg
P_{O_2}	80–100 mm Hg (adults) 60-70 mm Hg (newborns)	40 mm Hg
HCO_3 (bicarbonate)	21–28 mmol/L (or mEq/L)	23–30 mmol/L

Adapted from: Jacobs DS, DeMott WR, Oxley DK. *Laboratory Test Handbook.* Hudson, OH: Lexi-

Comp; 2001.

Laboratory Analysis

Blood Gas Analysis

The concentration of free H^+ in extracellular fluid (pH) as well as other key variables can be obtained from an arterial blood gas (ABG) or venous blood gas (VBG) sample. Results also include P_{CO_2}, P_{O_2}, and HCO_3^- measurements. Additional values such as base excess, oxygen saturation, carboxyhemoglobin, lactate, and various electrolytes or blood count values are often included in these panel results. These tests are available immediately on many transport vehicles and can be obtained in most, if not all, hospitals. Many outlying clinics, treatment centers, and offices have advanced **point-of-care (POC) testing** capabilities. Normal values are listed in **Table 16-16**.

Interpretation of Blood Gas Sample Results

Arterial blood gas sample results provide the CCTP with valuable information regarding the existence or type of acidosis or alkalosis. First consider the overall pH. A pH below 7.35 is considered an acidosis, regardless of any compensation (discussed later). A pH above 7.45 is considered an alkalosis, again regardless of any compensation.

Next consider the primary source. An acidosis or alkalosis is primary metabolic, primary respiratory, or mixed (combined). Once the primary source of acidosis or alkalosis is determined, the presence of any compensation should also be evaluated. Overcompensation of a primary condition will not occur. Any evidence of overcompensation indicates another separate primary pathology.

Metabolic acidosis occurs when a patient has a decreased pH (< 7.35) and a decreased HCO_3^- level (< 23 mmol/L). An individual may attempt to compensate by increasing respiratory rate and/or depth, thus lowering the level of P_{CO_2}. These efforts may normalize the pH, but will never overcompensate **Figure 16-39**.

Respiratory acidosis occurs when a patient has a decreased pH (< 7.35) and an increased P_{CO_2} level (> 45 mm Hg). In this instance, the respiratory effort/exchange is inadequate to remove enough CO_2 to maintain a normal pH. If this condition persists over time, renal compensation will begin to occur, demonstrating an elevated HCO_3^- level (> 29 mmol/L) **Figure 16-40**.

Metabolic alkalosis occurs when a patient has an elevated pH (> 7.45) and an elevated HCO_3^- level (> 29 mmol/L.). The patient's P_{CO_2} level may increase in compensation because the alkalosis inhibits respiratory drive **Figure 16-41**.

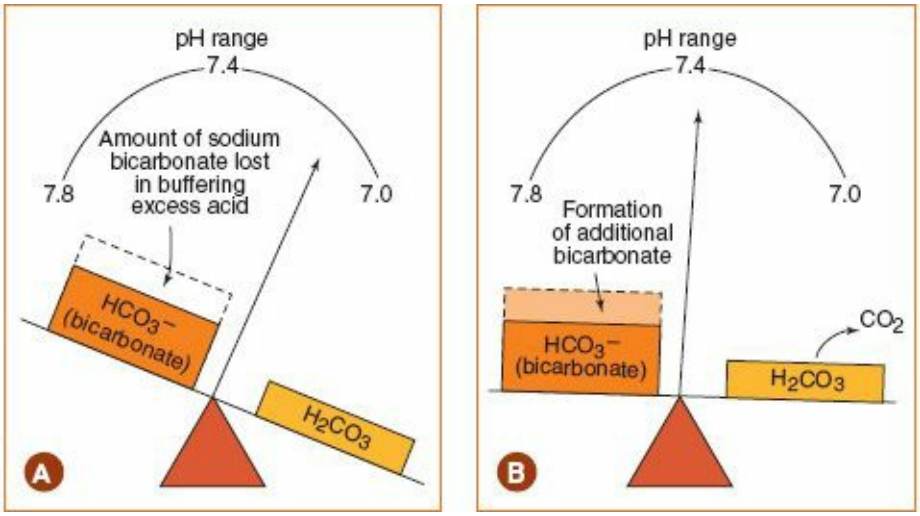


Figure 16-39 **A.** Derangement of acid-base balance in metabolic acidosis. **B.** Compensation by reduction of carbonic acid and formation of additional bicarbonate.

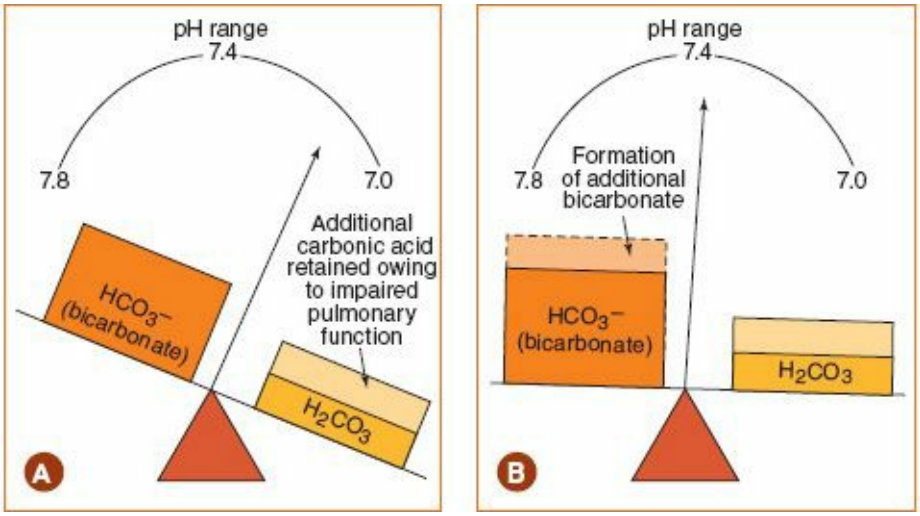


Figure 16-40 **A.** Derangement of acid-base balance in respiratory acidosis. **B.** Compensation by formation of additional bicarbonate.

Respiratory alkalosis occurs when a patient has an increased pH (> 7.45) and a decreased P_{CO_2} level (< 35 mm Hg). This is usually the result of some type of alveolar hyperventilation. Over time, renal compensation mechanisms promote the retention of H^+ and the excretion of HCO_3^- **Figure 16-42**. HCO_3^- values will be lower if this problem persists for several hours to several days or longer.

Mixed acidosis involves a low pH (< 7.35) as well as an elevated P_{CO_2} level (> 45 mm Hg) and low HCO_3^- level (< 23 mmol/L). This occurs when both respiratory and metabolic acidosis are present at the same time in the same patient. Severe trauma, cardiogenic shock, or a drug overdose are common situations in which this may occur.

Mixed alkalosis involves an elevated pH (> 7.45), a low P_{CO_2} level (< 35 mm Hg), and an elevated HCO_3^- level (> 29 mmol/L). This may occur when two seemingly unrelated medical issues manifest at the same time in the same patient. For example, a patient with chronic respiratory alkalosis who experiences a gastrointestinal emergency may have respiratory alkalosis combined with metabolic alkalosis and therefore demonstrate mixed alkalosis.

A patient with one of the primary conditions previously described may present to the CCTP as uncompensated, partially compensated, or well compensated, but not overcompensated unless two pathologic conditions are simultaneously present. Practice is essential for developing comfort in the identification of complex acid-base disorders.

VBG analysis has limitations compared to an ABG determination in certain situations. Both pH and HCO_3^- levels correlate well between venous and arterial blood gas measurements. The Pa_{O_2} level does not correlate well and should not be inferred from the result of a VBG analysis. A VBG analysis can be used as a screening tool for an elevated P_{CO_2} level. The P_{CO_2} values generally correlate well between ABG and VBG analyses, although the relationship becomes less accurate as the patient's perfusion or hemodynamic status deteriorates.

Indirect Measurement of Acid-Base Status

Total blood carbon dioxide, which can be written as either T_{CO_2} or HCO_3^- in clinical practice, provides

an indication of an acid-base abnormality on routine blood work. This test is often included on the basic chemistry panel (Chem-7, SMA-7) and may provide the necessary information to determine a patient's acid-base abnormality. The normal plasma value is 23 to 29 mmol/L.

An elevated TCO_2 value may indicate either a chronic respiratory acidosis with CO_2 retention or a metabolic alkalosis. Low TCO_2 values may indicate either a metabolic acidosis or respiratory alkalosis. In either instance, further evaluation is warranted to determine the cause. This particular test should only be used for screening purposes for the presence of a metabolic acid-base alteration, which can be present as the primary problem or be evidence of compensation for a respiratory alteration. The TCO_2 value provides limited information by itself, but may prompt an attentive clinician to the presence of a significant acid-base disorder. A blood gas with pH measurement remains the definitive test.

Evaluation of Anion Gap

When an acidosis is suspected, its origin can be examined using the anion gap (AG). Acidosis may be present with or without an AG, but this will help exclude or identify possible specific causes of the acidosis. The AG is a calculated value using the sodium (Na), chloride (Cl), and HCO_3^- results. Many laboratory instruments automatically perform this calculation when communicating electrolyte panel results. The formula is:

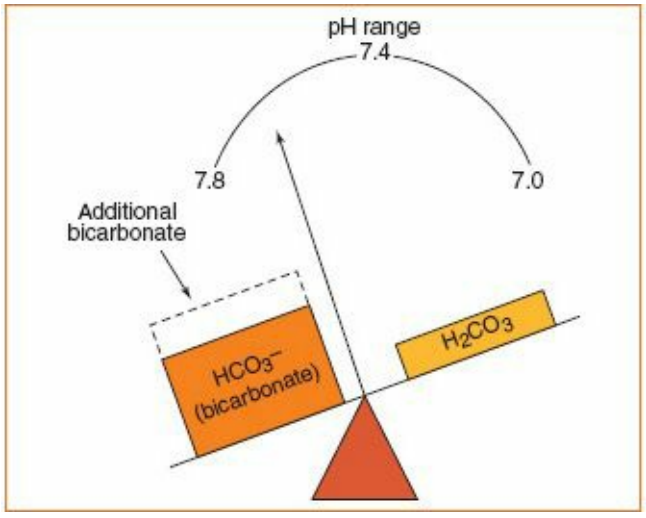


Figure 16-41 Derangement of acid-base balance in metabolic alkalosis. The body's compensatory mechanisms are ineffective.

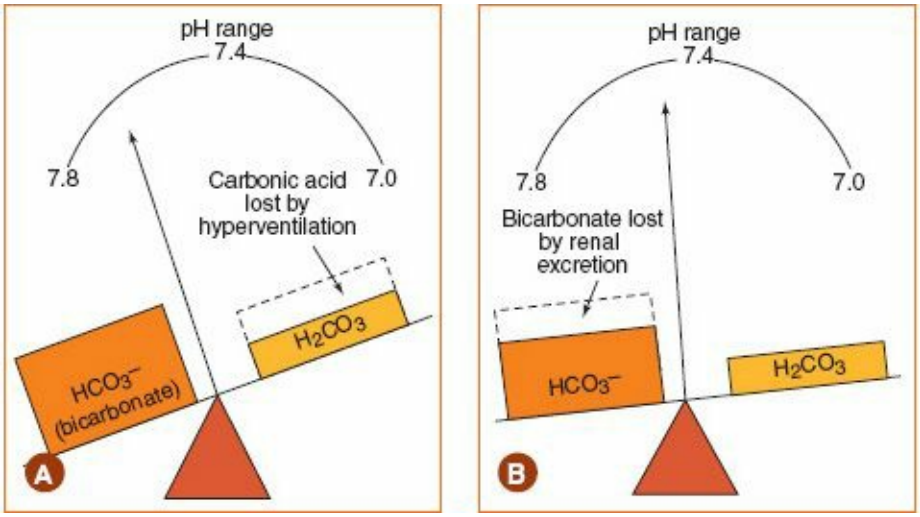


Figure 16-42 **A.** Derangement of acid-base balance in respiratory alkalosis. **B.** Compensation by

excretion of bicarbonate.

$$AG = Na - (Cl + Hco_3^-)$$

The normal AG is 8 to 12 mEq/L. An elevated AG usually represents the presence of lactic acid that is elevated as a result of trauma, shock, seizures, hypoxia, or numerous toxic substances. Toxic substances, including methanol, ethylene glycol, salicylates, and valproic acid, cause an elevated AG without lactic acid. **Alcoholic ketoacidosis** and **diabetic ketoacidosis** also cause an elevated AG acidosis as a result of ketones, without increased lactic acid. Serum lactate levels greater than 5.0 mmol/L indicate **lactic acidosis**.

An acidosis with a normal AG is a **hyperchloremic acidosis**. Relative losses in HCO_3^- are corrected mathematically with an increase in chloride concentrations. Hyperchloremic acidosis occurs from excessive chloride loading, pathologic retention of H^+, or unusual HCO_3^- losses.

Metabolic Alkalosis

When metabolic conditions create an excess of HCO_3^- accompanied by a pH of greater than 7.45 on an ABG sample, the disorder is termed metabolic alkalosis. This disorder is further described as "chloride resistant" or "chloride responsive," depending on the urine chloride concentration. Metabolic alkalosis results from either H^+ losses or gains of HCO_3^-, relative or actual. Relative gains of HCO_3^- occur when there is a disproportional loss of chloride, usually accompanying a sodium loss that alters electroneutrality leading to renal HCO_3^- retention. This disorder is the most common acid-base disturbance in hospitalized adults.

Vomiting or gastric suction is a common cause for metabolic alkalosis. The use of certain diuretics can cause metabolic alkalosis when they prompt the excretion of excess chloride ions in the urine. Various endocrine conditions, including primary aldosteronism, Bartter syndrome, Liddle syndrome, primary reninism, and hyperglucocorticoidism, are implicated as well.

Prehospital providers risk causing metabolic alkalosis when a patient with a previously compensated respiratory acidosis from hypercapnea or chronic obstructive pulmonary disease with an elevated P_{CO_2} level is placed on a ventilator and aggressively ventilated. Prior to ventilation, the chronically elevated P_{CO_2} level is compensated for with enhanced HCO_3^- retention by the kidneys to maintain a neutral pH. Following aggressive ventilation, the CO_2 level normalizes, creating a relative HCO_3^- excess, allowing a metabolic alkalosis to predominate.

The excessive administration of sodium HCO_3^- may cause an occasional, transient metabolic alkalosis. The extent of the injury is usually limited in most situations. The kidneys quickly begin excreting the excess HCO_3^-, resolving the misadventure. This mechanism may be impaired in patients with renal disease, causing more serious complications, including cell dysfunction or death.

Clinical Features

Patients with metabolic alkalosis demonstrate many significant clinical effects, often related to concomitant electrolyte abnormalities. Elevated blood pH increases hemoglobin affinity for oxygen molecules, inhibiting tissue oxygen extraction and leading to tissue hypoxia. Compensatory hypoventilation and hypercarbia further undermine tissue oxygenation. Expect P_{CO_2} levels to increase 0.7 mm Hg for every 1 mmol/L increase in HCO_3^-. Seizures, altered mental status, refractory and lethal arrhythmias, and weakness may occur. Alkalosis will decrease ionized calcium levels and sequester

potassium within cells (causing a transient hypokalemia), each causing additional symptoms.

Treatment

The treatment of metabolic alkalosis is directed at the identification and correction of the particular cause in a specific patient. Urgent treatment should occur when the HCO_3^- level exceeds 40 mmol/L or the pH exceeds 7.55. Many patients experiencing metabolic alkalosis have potentially massive hypovolemia from gastrointestinal- or renal-related fluid volume losses. These patients require volume resuscitation and electrolyte repletion with careful monitoring. Potassium, sodium, calcium, and chloride repletion are often required.

Specific management includes surgical management for pyloric stenosis, which causes protracted vomiting and massive GI fluid and H^+ losses. Other GI fluid and H^+ losses are controlled with treatment of emesis or the discontinuation of gastric suction devices. Diuretic discontinuation (may be temporary) or changing the type of diuretic is indicated when renal chloride losses are the cause of the alkalosis.

Several pharmacologic options exist for the treatment of severe metabolic alkalosis. Hydrochloric acid can be given IV to replete H^+ and correct chloride losses. Potassium chloride will replete potassium and also correct chloride losses. Acetazolamide (Diamox) is a diuretic and carbonic anhydrase inhibitor that can correct any fluid volume excess and promote the excretion of HCO_3^- in the urine. Chloride-responsive alkalosis is generally treated with the previously described medications. Chloride-resistant alkalosis often benefits from potassium chloride repletion, but also requires correction of the underlying condition to resolve the alkalosis. Certain endocrine disorders have specific treatment requirements that necessitate consultation with medical direction.

Metabolic Acidosis

Metabolic acidosis is demonstrated by a decreased serum pH (< 7.35) accompanied by a decreased HCO_3^- concentration (< 23 mmol/L). This disorder occurs through three possible mechanisms:

1. Kidneys may be unable to excrete enough of the 50 to 100 mEq of H^+ produced or absorbed each day.
2. An increased amount of H^+ is present because of exogenous loading or altered metabolism.
3. The kidneys or GI tract excrete too much HCO_3^-, causing an imbalance.

A myriad of situations cause metabolic acidosis. Cellular starvation states such as diabetic ketoacidosis, alcoholic ketoacidosis, and profound malnutrition each accumulate acids from altered metabolism and demonstrate an increased AG acidosis. GI HCO_3^- losses occur from diarrhea, fistulas, or lower GI suctioning, causing metabolic acidosis. Many toxic chemicals produce either an AG or non-AG metabolic acidosis following exposure or absorption.

Excess accumulation or impaired excretion of lactate leads to acidosis and an elevated AG. Lactic acidosis can be a result of exposure to various toxic substances, such as ethylene glycol, ethanol, epinephrine, cocaine, iron, propofol, salicylates, valproic acid, metformin (Glucophage), and numerous others. It can occur from inadequate tissue perfusion in various shock states, dysfunction of certain organs, nutritional deficiency, infection, malignancy, or diabetes. Other hereditary metabolic disorders, such as glucose-6-phosphatase deficiency, pyruvate carboxylase deficiency, oxidative phosphorylation deficiency, and methylmalonic aciduria, also cause lactic acidosis.

Renal Metabolic Acidosis

Kidney dysfunction is the cause of metabolic acidosis through a variety of mechanisms. Uremic states

from the accumulation of cellular waste products directly cause a metabolic acidosis. Inadequate renal ammonium (NH_4) production prevents excretion of sufficient H^+ leading to acidosis.

Type 1 (distal) **renal tubular acidosis (RTA)** impairs the kidney's ability to excrete H^+. This state is often characterized by plasma HCO_3^- levels of less than 15 mmol/L, hypokalemia, increased urine calcium levels, and kidney stones. Urine pH remains greater than 5.5 despite acidosis. This condition is relatively rare and may be hereditary, autoimmune, or linked to other diseases or chemicals.

Type 2 (proximal) RTA involves impaired HCO_3^- reabsorption translating into increased renal HCO_3^- excretion. It is characterized by an initial elevated urine pH until plasma HCO_3^- levels drop, then the urine pH drops significantly. Glucose, protein, and phosphate are increasingly excreted in the urine in addition to HCO_3^-. This condition is also rare. Causes included heredity, numerous medications, and a variety of medical conditions.

Type 4 (generalized or hyperkalemic) RTA results from impaired liberation of renin by the kidneys or altered synthesis, excretion, or response to aldosterone by the kidneys or adrenal glands. In addition to hyperkalemia, there is often an elevated urine sodium level (> 40 mmol/L) and hyperchloremia. Type 4 RTA is the most common manifestation of RTA. It is linked with toxicity to numerous medications and genetic disorders, and as a consequence to diabetes, HIV, systemic lupus erythematosus, and sickle cell anemia. Types 1, 2, and 4 RTA present with a metabolic acidosis without an elevated AG.

Finally, Type 3 RTA is a rare classification, with characteristics of Type 1 and Type 2, and is not clinically relevant.

Clinical Features

Metabolic acidosis is rarely a clinically isolated occurrence. Underlying pathologic conditions causing acidosis will predominantly shape the clinical status of a particular patient. Kussmaul respirations are characteristic of metabolic acidosis because a decreasing pH will stimulate respiratory centers, causing an increase in alveolar ventilation. Hypotension and hypovolemia are also characteristic of severe metabolic acidosis, demonstrating the progression of widespread cell, tissue, organ, and body system dysfunction. In response to acidosis, potassium ions shift out of the cell, moving excess H^+ into the cell, resulting in a pseudohyperkalemia even when there is an actual normal or decreased whole-body potassium presence.

Treatment

CCTPs should resist the temptation to treat every metabolic acidosis with IV sodium HCO_3^-. Certain conditions, such as RTA, hyperchloremic acidosis, and isolated GI HCO_3^- losses, are amenable to treatment with IV sodium HCO_3^-, but other causes of metabolic acidosis require a more specific approach. Sodium HCO_3^- is not generally indicated even in these circumstances unless the pH has fallen below 7.2.

Lactic acidosis requires correction of cellular metabolism not accomplished with sodium HCO_3^-. Improving cellular metabolism will cease production of excess H^+ and allow normal acid-base control mechanisms to restore a physiologic pH. HCO_3^- is ineffective in the treatment of lactic acidosis and is not recommended. Optimal tissue oxygen delivery and adequate perfusion are the primary goals of lactic acidosis treatment along with more specific management of potential underlying causes.

The various types of RTA are treated with agents such as sodium HCO_3^- or sodium citrate often over long periods of time. Calcium, potassium, and other electrolytes require careful monitoring and often repletion, depending on the particular type of RTA. Type 2 RTA often requires potassium repletion. Type

4 RTA often requires volume expansion, potassium wasting diuretics, or even mineralocorticoid replacement therapy in select situations.

Sodium HCO_3^- is not recommended for the treatment of diabetic ketoacidosis and has been shown to cause cerebral edema when administered to children with this condition. Diabetic ketoacidosis is best managed with fluid volume restoration, IV insulin, and electrolyte (especially potassium and sodium) correction.

Alcoholic ketoacidosis, another cellular starvation acidosis, is also not treated with IV sodium HCO_3^-, unless there is a profound, life-threatening acidosis (pH > 7.1). Alcoholic ketoacidosis is treated with IV fluids and carbohydrates to improve nutritional status and allow normal metabolic processes to resume. Potassium chloride in 5% dextrose and sodium chloride injection D_5NS and thiamine (to correct deficiencies and prevent Wernicke's encephalopathy) are generally the only medications needed for treatment.

Other causes of metabolic acidosis, such as uremia or exposure to toxic chemicals, require specialty consultation and often complex treatment. Critical care providers should seek medical consultation when transporting patients with these conditions.

Diagnosis and management of patients with metabolic alterations of acid-base status can challenge CCTPs. Clinical suspicion, focused investigation, and optimal treatment of these disorders will improve patient outcomes and minimize the adverse impact of many serious medical conditions. Providers should consider the potential and consequences of metabolic acid-base alterations during every critical care transport.

Flight Considerations

Critical care providers must take precautions or modify techniques when performing air-medical patient transport. Unpressurized or underpressurized aircraft place GI/GU patients at increased risk for complications related to transport. Careful assessment and simple interventions eliminate or minimize many adverse effects.

GU complications specifically related to air-medical transport are rare. Prolonged exposure to altitude will alter urinary pH. Pneumaturia (presence of gas within the bladder) can occur following a urinary tract infection with certain microorganisms, after manipulation of the genitourinary tract, and when a fistula develops between the digestive tract and bladder (causing bowel contents to contaminate the normally sterile bladder environment). Pneumaturia requires more specific interventions, often including an indwelling urine (Foley) catheter for bladder and urinary tract decompression. Unless contraindications are present, a catheter will eliminate the need for patients to void during transport, permit assessment of fluid volume status, and prevent complications associated with urinary retention. GI complications during air medical transport are much more common. Sequestered gases due to abdominal surgery, bowel obstruction, and abdominal trauma will significantly expand as cabin altitude increases. Ascending from sea level to 9,000′ will cause any sequestered gas to expand by 50%. In severe situations, this gas expansion can lead to respiratory compromise, severe discomfort, syncope, and vasovagal episodes.

Patients with any of the previously mentioned conditions should have an NG or OG tube placed prior to any increase in cabin altitude. Once these tubes are in place, they should be either vented to ambient air or connected to suction for transport. Colostomy patients should have their devices frequently monitored for excessive amounts of gas and either emptied or changed when this occurs. If at all possible, patients who have undergone abdominal surgery should wait 24 to 48 hours following surgery before being transported by air.

CCTPs should take certain steps to minimize personal GI symptoms associated with air medical transport. Drinking carbonated beverages, swallowing large amounts of air while eating, and consuming gas-forming foods all contribute to GI distress at altitude.

Summary

The GI and GU systems are complex pathways through the body that support critical functions—the digestion of food and the elimination of wastes, respectively. They withstand constant usage, leading to inevitable wear and tear. The many GI/GU pathologies that can occur require prompt recognition and treatment to preserve function. Because of the interactions of these systems with accessory organs and other systems, local problems can quickly evolve into systemic ones. Through awareness of the possible diseases, history taking, and proper assessment, many complications can be prevented or corrected at an early stage.

The CCTP is the connection between the patient and the tertiary care required. The continuity of care provided is a fundamental piece of the treatment and recovery of the critically ill patient with a GI/GU condition. When a problem arises during transport, it is the responsibility of the CCTP to correct it and prevent its recurrence. Being equipped with this knowledge provides the CCTP and the patient with the greatest likelihood for a safe, uneventful transfer.

Case Study

YOU AND YOUR CRITICAL CARE TEAM ARE DISPATCHED to the scene of an adolescent who is complaining of severe abdominal pain. Upon your arrival at the residence, you find a 17-year-old female lying on the bathroom floor. The patient's mother reports that her daughter has been complaining of stomach and shoulder pain for the past four days. At first she thought that her daughter was exaggerating because she did not want to go to school, but she became concerned when her daughter collapsed on the bathroom floor. You question the mother further and find out that the daughter had a "spell" yesterday morning, but the mother attributed it to her daughter taking diet pills and not eating enough. The patient denies having had a fever or vomiting. You question the patient further and are informed that she had a regular menstrual period last month and is due to start any day now. She denies being sexually active and states that she does not take contraceptives.

You perform your physical exam, which reveals a well-nourished female who appears to be in acute distress. The patient is lethargic, but will respond to your questions. Her pupils are equal round at 4 mm and reactive to light. Her airway is patent and clear, as evidenced by verbal communication. Breath sounds are present and clear to auscultation. Her respiration rate is 26 breaths/min and shallow. S1 and S2 heart tones are auscultated, and no murmurs, gallops, or friction rubs are noted. The radial pulse is 115 beats/min and strong. Her initial blood pressure is 92/40 mm Hg. Her skin is pale and diaphoretic. The abdomen is positive for rebound tenderness and is slightly distended. The patient has a positive Kehr's sign.

Your partner brings the stretcher to the bathroom doorway in preparation to load the patient in the hallway because the bathroom is very narrow. You and your partner prepare to assist the patient in moving to the stretcher. As the patient attempts to stand with assistance, she loses consciousness and is assisted to the ground by you and your partner. The patient regains consciousness shortly afterwards. Your critical care team initiates two large-bore IVs, places the patient on oxygen via nonrebreathing mask at 15 L/min, and prepares to transport the patient to the closest emergency department with an ETA of 15 minutes.

The patient's condition continues to deteriorate en route. Upon arrival at the emergency department,

the patient's condition has worsened; her blood pressure has dropped to 70/40 mm Hg, her pulse rate is 125 beats/min, and she is only responding to tactile stimulation. Blood and urine were collected and sent to the laboratory. An emergency transvaginal sonogram was requested.

The lab values are as follows:

- Urinalysis
  - Color, dark yellow
  - Appearance, clear
  - Specific gravity, 1.030
  - pH 5.3
  - Protein, none
  - Glucose, none
  - Ketones, none
  - Bilirubin, none
  - Urobilinogen, normal
  - Hemoglobin, none
  - Red blood cells, none
  - Nitrite, none
  - White blood cells, none
- Hematology tests
  - White blood cells, 7,725/μL
  - Red blood cells, 3.5×10^6/μL
  - Hemoglobin count, 7.5 g/dL
  - Hematocrit value, 22.5%
  - Platelets, 145,000/μL
- Electrolytes
  - Sodium, 138 mEq/L
  - Potassium, 4.6 mEq/L
  - Chloride, 114 mEq/L
  - Calcium (ionized), 9.9 mg/dL
- Arterial blood gas analysis
  - pH, 7.01
  - P_{CO_2}, 68 mm Hg
  - P_{O_2}, 38 mm Hg
  - Base excess, 13 mmol/L
  - HCO_3^-, 11.7%
  - O_2 saturation, 59%
  - O_2, 15 L/min 100%
- Other test
  - Human chorionic gonadotropin, 1,500 mIU/mL

- Transvaginal sonogram
  - The results of the sonogram revealed a right complex mass measuring 3 to 4 cm, minimal free fluid in the adnexa, and an empty sac. The findings were discussed with the radiologist, who stated that the appendix could not be well visualized.

1. What is the patient's diagnosis?
2. What treatment should be initiated?
3. What other diagnosis should be considered in this patient?

Analysis

Pregnant patients who present with an acute abdomen are considered a surgical emergency and require an appropriate diagnosis to avoid the risk of maternal and fetal mortality. Ectopic pregnancy and appendicitis are two possible causes of acute abdomen in a pregnant patient. CCTPs should not become complacent and look for a single diagnosis, but be observant for the possibility of concurrent causes of abdominal pain in the female patient, particularly during pregnancy.

The patient was transfused with 3 units of packed red blood cells and a culdocentesis was performed (aspiration of fluid from the space between the uterus and rectum), which revealed non-clotting blood in the cul-de-sac. A preoperative diagnosis of acute abdomen was made, and the patient was taken emergently to surgery for a laparotomy, which revealed a ruptured right fallopian tube. A saplingectomy was performed (removal of the fallopian tube), the blood and clots were aspirated from the abdominal cavity, and bleeding was localized and controlled. The patient was transferred to the recovery room postoperatively with stable vital signs and later transferred to the OB/GYN floor for further follow-up.

The patient made a full recovery from the ectopic pregnancy, and was discharged 2 days later. She was instructed to return for a follow-up in 1 week for a serum human chorionic gonadotropin test to confirm a decrease in human chorionic gonadotropin levels.

Prep Kit

Ready for Review

- Homeostasis depends on a number of metabolic processes involving the GI and GU systems, but these processes can be the target of a number of pathologic conditions. Assessment and management of GI and GU pathologies is inherently difficult because the signs and symptoms that accompany these may be obscure.
- The GI system consists of a specifically arranged network of organs and ducts devoted to the digestion of food and the extraction of its nutritional content.
- The alimentary canal contains numerous specialized cells and tissues. Four layers are seen almost continuously from the esophagus to the rectum: the mucosa, the submucosa, the muscularis externa, and the serosa.
- The alimentary canal maintains a chemical environment that is beneficial for the digestion of food, without altering the environment of the rest of the body.
- The alimentary canal consists of the mouth, pharynx, esophagus, stomach, small intestine, and large intestine; food travels through these structures and finally exits the body via the rectum.
- The renal system provides a path for waste to leave the body.

- The kidneys continuously filter blood, manage volume, maintain appropriate balances between acids and bases, and discard toxins and excesses by producing urine. The remainder of the urinary system—the ureters, urinary bladder, and urethra—provides for transport and storage of urine during its journey out of the body.
- The purpose of the male reproductive system is to generate sperm and provide a means for its delivery to a fertilizable egg in the female partner.
- The female reproductive system produces and develops eggs. In addition, the uterus of the female provides a home for a developing embryo during gestation.
- The common symptoms of GI abnormalities include abdominal pain, tenderness, and bleeding.
- GI bleeding can be classified into one of two categories based on the location of the bleeding: upper GI bleeding and lower GI bleeding.
- Endoscopy is the preferred method of locating the bleeding source.
- Peptic ulcers are the leading cause of upper GI bleeding, accounting for more than 50% of acute cases.
- Stress-related erosive syndrome is a frequent cause of peptic ulcers in critically ill patients, with an incidence of nearly 100%.
- Ulcers associated with severe burns are termed Curling's ulcers, and those accompanying episodes of increased intracranial pressure are Cushing's ulcers.
- Inflammation of the gastric mucosa, termed gastritis, is a common precursor to upper GI bleeding.
- Acute gastritis is characterized by a rapid onset of mucosal inflammation frequently accompanied by mild to severe upper GI bleeding.
- Autoimmune disorders targeting the GI system can cause chronic inflammation of mucosal lining and can also inhibit certain metabolic pathways crucial to digestion.
- Reactive gastritis is a chronic mucosal edema that results from recurring contact of the mucosa with antagonistic substances such as bile, pancreatic juice, or NSAIDs.
- Radiation exposure causes varying forms of gastritis depending on the amount of radiation involved.
- An esophageal varix results from the swelling of esophageal veins into the lumen of the esophagus.
- When portal hypertension occurs, venous blood will use alternate pathways for its return to the vena cava via the azygos system.
- In cirrhosis, buildup of fatty acids, along with chronic destruction of liver tissue and fibrosis, obstructs blood flow.
- Mallory-Weiss syndrome involves a longitudinal laceration of the esophageal mucosa as a result of repeated significant changes in local pressure, such as those seen in repeated vomiting.
- Lower GI bleeding is frequently the result of an exacerbation of a chronic condition.
- Diverticulosis is a common disease of the lower GI in the elderly.
- Diverticulitis, an inflammation of the diverticula, is frequently a result of infection from invading intestinal bacteria and is the most common cause of lower GI bleeding.
- Angiodysplasia is a malformation of submucosal blood vessels in the GI tract.
- Inflammatory bowel disease is a collective term covering two idiopathic colon pathologies that are together responsible for about 10% of the cases of lower GI bleeding: ulcerative colitis and Crohn's disease.
- Ulcerative colitis is an inflammation of the rectal mucosal and submucosal tissues.

- Crohn's disease is a less organized inflammation of the GI tract in which all layers of the mucosa may be affected.
- Radiation colitis occurs in 75% of patients who have received radiation doses of 40 Gy or more to the abdomen or pelvis.
- Symptoms of GI bleeding include melena, diffuse abdominal pain, emesis containing hematemesis, fever, complaints of frequent dizziness and syncope, diarrhea, dehydration, and cardiac arrhythmias.
- Symptoms of significant GI bleeding include hemodynamic instability, shock, large amounts of bright red blood in vomit, hematochezia, hypotension, tachycardia, diaphoresis, altered mental status, and a decreased level of consciousness.
- Fluctuations in the hematocrit value over time indicate active bleeding.
- Pressure ulcers can arise from prolonged pressure on body tissues, are commonly seen in patients confined to a bed, and are classified on a scale of stages 1 through 4.
- Endoscopy is capable of visualizing more than 90% of the upper GI tract with a 90% to 95% diagnosis rate.
- Colonoscopy allows visualization of the entire rectum and large intestine, up to the ileocecal junction.
- For an angiography, dye injected into blood vessels will leak into the lumen of the GI tract at the site of the bleeding.
- During acute GI bleeding, airway compromise occurs primarily by either of two ways: physical obstruction or mechanical obstruction.
- Any patient with acute GI bleeding should be designated NPO (nothing by mouth), and blood samples should be drawn concurrently with IV placement.
- Along with treating the immediate concerns of GI bleeding, other factors such as incontinence, diarrhea, and constipation need to be addressed.
- Treatments for endoscopic and pharmacologic refractory variceal hemorrhage include balloon inflation and shunt placement.
- The introduction of a transjugular intrahepatic portosystemic shunt into the systemic venous circulation results in a significant decrease in hepatic portal pressure and, therefore, a significant decrease in the pressure in the esophageal collateral veins.
- A mechanical obstruction is the result of a physical blockage of the intestinal lumen and can be classified into one of three categories: extrinsic, intrinsic, or intraluminal.
- Ileus is the lack of movement of the GI contents through the intestines in the absence of a mechanical obstruction.
- In a patient with an intestinal obstruction, the abdomen is tender and distended on palpation, typically with diffuse pain.
- Plain radiography can diagnose an intestinal obstruction by detecting abnormally high air volumes proximal to the obstruction site. A CT scan can be used to diagnose a bowel obstruction with exceptional specificity.
- After the stabilization of any life threats to the patient's airway, breathing, or cardiac functioning, initial treatment focuses on fluid resuscitation.
- An NG or OG tube may already be in place upon the CCTP's first encounter with the patient, particularly during interfacility transports.
- In general, an insult to the liver is either infectious or noninfectious. If the insult resolves within 6

months without any permanent function deficit, it is termed acute. When symptoms persist longer, chronic liver disease has developed.

- Hepatitis, an inflammation of the liver, is the physiological result of any liver disease.
- Fulminant hepatic failure is the result of a sudden significant insult to the liver.
- The leading cause of cirrhosis is infection with the hepatitis C virus (HCV), with alcoholic liver disease closely following.
- Inflammation of liver tissue results in damage to a variety of functional cells, including hepatocytes and Kupffer cells.
- Bilirubin that is normally conjugated by hepatocytes and excreted via bile begins to build up in the blood, resulting in jaundice.
- Symptoms of early acute hepatitis include general malaise, generalized fatigue, and fever and chills. Other symptoms include portal hypertension, melena, hematochezia, chronic shortness of breath, hypotension, tachycardia, and hair loss.
- Liver function is assessed by evaluating the presence of its synthesized products as well as the absence of its cellular enzymes in the blood. It is important to recognize the distinction between tests for liver function and markers of injury.
- Treatment of hepatitis focuses on prevention of further injury with a goal of preventing progression to cirrhosis.
- Blockage of the biliary tracts at any point will result in a backup of bile and, possibly, depending on the location of the obstruction, pancreatic enzymes.
- Gallstones in the gallbladder, cholelithiasis, are a fairly common condition that affects around 20 million Americans per year.
- Cholecystitis causes colicky pain in the right upper quadrant of the abdomen. Patients typically present with Charcot's triad, nausea and vomiting, clay-colored feces, and a positive Murphy's sign.
- Imaging and detecting techniques include transabdominal ultrasonography, endoscopic ultrasonography, hepatoiminodiacetic acid scanning, endoscopic retrograde cholangiopancreatography, and percutaneous transhepatic cholangiography.
- Immediate treatment of choledocholithiasis involves pain management once general treatment has been performed. Opiates should be avoided, because they can cause spasms of the biliary ducts at the ampulla of Vader.
- Inflammation of the pancreas, known as pancreatitis, can lead to significant impairment of gastrointestinal physiology depending on its level of severity and etiology.
- Upper abdominal pain is the primary symptom of pancreatitis. Symptoms include tenderness and guarding of the abdomen, tachycardia, nausea and vomiting, and a fever. Turner's sign, Cullen's sign, jaundice, hemodynamic instability, and shock may result from severe pancreatitis.
- Although elevated levels of serum amylase and serum lipase support a diagnosis of pancreatitis, they can result from a number of other conditions as well.
- A CT scan with contrast dye can provide a direct visual observation of pancreatic necrosis, peripancreatic abnormalities, abscesses, and overall size of the pancreas.
- General treatment of pancreatitis includes palliative care such as supplemental oxygen administration, IV placement, and ECG monitoring.
- Pain management is necessary and may take the form of IV narcotics or patient-controlled analgesia

devices.

- Significant decreases in renal perfusion pressure disrupt the elimination of waste from the bloodstream by lowering the glomerular filtration rate (GFR). When this situation develops in the absence of preexisting renal dysfunction, it is termed acute renal failure (ARF).
- Symptoms include dizziness, poor skin turgor, thirst, flat neck veins, dry mucous membranes, weight loss, orthostatic blood pressure changes, fever, edema, ascites, and lower back pain.
- A family history of renal disease indicates a predisposition to ARF when a precursory disease of ARF is present. The incidence of ARF is increased in patients with diabetes, hypertension, or lupus.
- The retention of wastes in the bloodstream that results from a decreased GFR provides an easy way to monitor for the existence and severity of ARF. Renal ultrasonography is the gold standard in assessing suspected postrenal ARF.
- Initial treatment of the patient with ARF involves maintaining the ABCs and a perfusing blood pressure. Once these basic elements are ensured, management focuses on increasing the GFR.
- A gradual decrease in renal function over a long time interval is known as chronic renal failure (CRF).
- Symptoms include azotemia, electrolyte imbalances, hyperkalemia, cardiac arrhythmias, congestive heart failure, edema, anemia, chronic fatigue, hyperparathyroidism, skeletal abnormalities, polyuria or anuria, metabolic acidosis, uremia, jaundice, and uremic frost phenomenon.
- The results of lab studies on CRF and ESRD are generally parallel to the results seen for ARF. The most important factor in determining whether the renal failure is acute or chronic is disease duration.
- The best treatment for chronic renal failure is prevention, as there is no definitive cure once it has progressed to clinically detectable signs. Once CRF is recognized, treatment focuses on slowing the progression.
- Bacterial flora that live symbiotically in the GI system can cause considerable damage when they are introduced into the urinary system. A urinary tract infection typically begins in the urethra, as this location is the most proximal to the external environment.
- In about 12% of the male population, the tunica vaginalis attachment is misaligned, allowing some degree of free testicular movement, known as the “bell clapper” deformity. These patients are susceptible to testicular torsion, a condition in which one or both of the testes rotate to the point of occluding their blood supply.
- Direct blunt trauma to the penis during penile erection can result in penile fracture. The fracture here is actually a rupture of one or more of the vascular spaces within the penis, mimicking the appearance of a long bone fracture.
- Priapism is a prolonged erection of the penis that can be painful and damaging to tissues in the immediate area. The two major classifications of priapism are arterial high-flow and veno-occlusive priapism.
- A simple NG tube can be used to administer food into the stomach or to remove food. For longer-term situations, a gastrostomy tube (G tube) or percutaneous endoscopic gastrostomy (PEG) tubes can be inserted
- Total parenteral nutrition is an alternative feeding method used when the GI tract is not functioning adequately. In TPN, all required nutrients are administered IV to the patient.
- When transporting a patient with a feeding tube, contact medical direction if the patient has vomiting, abdominal distention, or diarrhea.

- Straight catheters, often called intermittent catheters, are designed for temporary use and are frequently self-inserted by the patient.
- Indwelling catheters, also known as Foley catheters, provide a complete management of urine for bedridden or incapacitated patients, with minimal upkeep.
- When surgery or trauma makes urethral catheter introduction unfavorable, a suprapubic tube can be placed directly into the bladder through the abdominal wall by way of a surgically created stoma.
- Common complications of urine collection catheters include clogging, infection, pain, leakage, and dislodgement. Dislodgement is the largest concern during transport. Any signs of infection should be treated with antibiotics immediately.
- An ostomy is a surgically created opening of the GI tract through the abdomen that allows for waste removal.
- The patient with an ostomy should be monitored for signs of tissue necrosis and infection, which, if detected, may warrant treatments ranging from antibiotic therapy to surgical intervention.
- Jackson-Pratt drains are inserted to remove postoperative buildups of fluid and blood in the abdominal cavity. During liver transplant surgery, a T tube is placed to monitor bile drainage from the gallbladder.
- In-flight complications need to be considered for the numerous drainage systems that include closed bags containing air. Excess air should be vented from any drainage bag such as a colostomy or urostomy prior to flight in anticipation of changes in air pressure.
- Be sure to unclamp any feeding or drainage catheters prior to departure, and ensure that they remain unclamped for the duration of travel.
- Air transport should be postponed or avoided for any patient with a bowel obstruction if possible.
- Significant alterations of oxygen and carbon dioxide levels, electrolyte concentration, temperature, glucose levels, and acid-base status will impair cell, tissue, and organ function, eventually leading to death.
- The relationship of acids to bases within the body is the pH value, which represents the concentration of free hydrogen ions (H^+) present.
- The bicarbonate–carbonic acid buffer system is the principle extracellular buffer system that operates primarily in the lungs and kidneys, where carbonic anhydrase is present to stimulate the chemical reaction.
- The phosphate buffer system operates similar to the bicarbonate–carbonic acid buffer system in that it allows only small pH changes following large variations in the presence of free H^+.
- The concentration of free H^+ in extracellular fluid (pH) as well as other key variables can be obtained from an arterial blood gas (ABG) or venous blood gas (VBG) sample.
- Results from an ABG sample provide the CCTP with valuable information regarding the existence or type of acidosis or alkalosis.
- Total blood CO_2, which can be written as either TCO_2 or HCO_3^- in clinical practice, provides an indication of an acid-base abnormality on routine blood work.
- When an acidosis is suspected, its origin can be examined using the anion gap.
- When metabolic conditions create an excess of bicarbonate (HCO_3^-) accompanied by a pH greater than 7.45 on an ABG sample, the disorder is termed metabolic alkalosis.

- Patients with a metabolic alkalosis demonstrate many significant clinical effects, often related to concomitant electrolyte abnormalities.
- Specific management of metabolic alkalosis includes surgical management for pyloric stenosis, which causes protracted vomiting and massive GI fluid and H^+ losses.
- Metabolic acidosis is demonstrated by a decreased serum pH (< 7.35) accompanied by a decreased HCO_3^- concentration (< 23 mmol/L).
- Kidney dysfunction is the cause of metabolic acidosis through a variety of mechanisms.
- Metabolic acidosis is rarely a clinically isolated occurrence. Underlying pathologic conditions causing the acidosis predominantly shape the clinical status of a particular patient.
- CCTPs should resist the temptation to treat every case of metabolic acidosis with IV sodium HCO_3^-. Providers should consider the potential and consequences of metabolic acid-base alterations during every critical care transport.
- Critical care providers must take certain precautions or modify techniques when performing air-medical patient transport.
- Pregnant patients who present with an acute abdomen are considered a surgical emergency and require an appropriate diagnosis to avoid the risk of maternal and fetal mortality.

Vital Vocabulary

acute gastritis A type of gastritis characterized by a rapid onset of mucosal inflammation, frequently accompanied by mild to severe upper GI bleeding.

acute renal failure (ARF) Decreased renal function in the absence of preexisting renal disease. Classified into three categories: prerenal, intrarenal, and postrenal.

acute tubular necrosis Damage to the tubules of the nephron, preventing proper ion and fluid exchanges in the kidneys.

adhesions Bands of connective tissue that can distort the normal GI anatomy. They are the result of improper healing or scar tissue growth following surgery.

alcoholic ketoacidosis A type of acidosis characterized by a buildup of ketones in the blood and caused by a large intake of alcohol.

angiodysplasia Deformed submucosal blood vessels in the GI tract that are prone to bleeding.

angiography Radiologic observation of dye injected into the bloodstream for leakage.

ascites A buildup of fluid in the abdominal cavity that can impede lung expansion and put pressure on other organs.

aspartate aminotransferase/alanine aminotransferase (AST/ALT) ratio A ratio used as an indicator of liver damage; this ratio can determine if the elevation is due to a liver injury or injury elsewhere in the body.

azotemia A combined elevation of blood urea nitrogen and serum creatine levels often seen in acute renal failure.

azygos system A network of blood vessels that connects the superior and inferior vena cava; it also drains a portion of the esophageal venous blood.

bicarbonate–carbonic acid buffer system The principal extracellular buffer system, which operates

primarily in the lungs and kidneys, and by which CO_2 and H_2O are reversibly converted into H_2CO_3 (carbonic acid), which in turn is reversibly converted into H^+ and HCO_3^- (bicarbonate).

bilirubin A waste product of heme formed during erythrocyte metabolism. It is moved to the small intestine within bile and then converted into urobilinogen, resulting in brown pigmented feces.

buffer Any substance that can alternately bind or release H^+ depending on outside conditions.

Charcot's triad Fever, jaundice, and right upper quadrant abdominal pain suggestive of choledocholithiasis.

cholangitis An infective form of biliary tract obstruction in which white blood cell levels rise.

cholecystitis Inflammation of the gallbladder.

choledocholithiasis Gallstones in the biliary tract system, which put the person at high risk for developing a biliary tract obstruction.

cholelithiasis Gallstones in the gallbladder.

chronic renal failure (CRF) A gradual decrease in renal function resulting from irreversible damage to the nephrons. It is characterized by a glomerular filtration rate (GFR) of less than 30 mL/min, and will eventually progress to end-stage renal disease.

cirrhosis Irreversible structural changes to the liver that impair its proper functioning.

colonoscopy Similar to endoscopy, but used for the lower GI tract.

concentration gradient The natural tendency for substances to flow from an area of higher concentration to an area of lower concentration, within or outside the cell.

creatinine The metabolized form of creatine that is eliminated through the urine. The rate of creatinine clearance is calculated as follows: [(140 — age) × weight in kilograms], divided by [plasma creatinine level × 72].

Crohn's disease An inflammation of the GI tract in which all layers of the mucosa may be affected. It results in scattered ulcerations and fibroses throughout the large and small intestines.

diabetic ketoacidosis A form of acidosis in uncontrolled diabetes in which certain acids accumulate when insulin is not available.

diverticula Small pouches of tissue that develop as outcroppings of the large intestine, typically in the descending colon and sigmoid colon.

diverticulitis Inflammation of diverticula.

diverticulosis Development of diverticula that may then become inflamed or bleed.

end-stage renal disease (ESRD) A loss of proper kidney functioning, with renal replacement therapy becoming a requirement for survival.

endoscopy The method of choice for visualization of the GI tract; it allows for observation, diagnosis, and treatment of an insult to the GI tract.

enteroclysis Infusion of barium contrast dye into the GI tract to observe for obstruction.

esophageal varix Swelling of esophageal veins into the lumen of the esophagus.

fulminant hepatic failure A sudden and significant insult to the liver characterized by encephalopathy and a mortality rate of up to 90%.

gastritis Inflammation of the gastric mucosa that occurs when the equilibrium of offensive and defensive

mechanisms for the lining is altered.

glomerular filtration rate (GFR) A benchmark for comparison of nephron function, based on the amount of fluid filtered by the glomerulus per minute.

Helicobacter pylori A bacterium that causes most peptic ulcers.

hematemesis Coffee-ground emesis containing partially digested blood.

hematochezia Stool streaked with bright red blood, originating from the lower GI tract.

hemodialysis (HD) Use of vascular access through a fistula or AV shunt to access the blood directly. Solute separation occurs across an artificial semi-permeable membrane within the dialysis machine.

hepatic encephalopathy Encephalopathy secondary to hepatic disease.

hepatitis Inflammation of liver cells that can impede proper functioning of the liver and lead to chronic conditions such as cirrhosis.

hepatitis C virus (HCV) The virus that is the most common cause of cirrhosis and is especially pathogenic in causing hepatitis.

hepatoiminodiacetic acid (HIDA) scan Administration of a radiologically labeled chemical specific for bile incorporation to a patient; the path taken by the marker is observed through radiography to ascertain whether an obstruction is present.

hernia A protrusion of an organ from its tissue lining.

homeostasis Stability in the body's internal environment.

hyperchloremic acidosis A form of metabolic acidosis in which low bicarbonate (base) concentration occurs along with an increase in chloride concentration.

ileus A lack of movement of GI contents in the absence of an obstruction.

inflammatory bowel disease (IBD) Term covering two colon inflammation pathologies: ulcerative colitis and Crohn's disease.

intussusception A prolapse of the intestine into an adjacent segment.

Jackson-Pratt drain A surgical drain used to remove fluid buildup from the wound site during the postoperative healing process.

jejunostomy tube (J tube) A feeding tube placed through the abdominal wall into the jejunum.

lactic acidosis A form of acidosis caused by an excess accumulation or impaired excretion of lactate, leading to an elevated anion gap; can result from exposure to various toxic substances, inadequate tissue perfusion in various shock states, dysfunction of certain organs, nutritional deficiency, infection, malignancy, diabetes, or hereditary metabolic disorders.

ligament of Treitz A small ligament supporting the small intestine at the junction between the duodenum and jejunum. It serves as the dividing point between the upper and lower GI tract.

lithotripsy Use of external vibrations to break up gallstones.

Mallory-Weiss syndrome Longitudinal laceration of the esophagus, often following severe vomiting.

mass A tumor, aneurysm, or abscess.

melena Black, tarry stool containing partially digested blood, with bleeding originating from the upper GI tract.

metabolic acidosis A pathologic condition (blood pH < 7.35) resulting from the accumulation of acids in

the body caused by any number of systems in the body, including the gastrointestinal system, or major organ failure.

metabolic alkalosis A pathologic condition (blood pH > 7.45) resulting from the accumulation of bases in the body caused by any number of systems in the body, including the gastrointestinal system, or major organ failure.

Minnesota esophagogastric tamponade tube A tube that is placed to stop bleeding of esophageal varices. It is similar to the Sengstaken-Blakemore tube, but has a built-in suction catheter.

mixed acidosis A pathologic condition in which there is a low pH (< 7.35), an elevated P_{CO_2} level (> 45 mm Hg), and a low bicarbonate level (< 23 mmol/L), which occurs when there is both a respiratory and a metabolic cause present at the same time.

mixed alkalosis A pathologic condition in which there is an elevated pH (> 7.45), a low P_{CO_2} level (< 35 mm Hg), and an elevated bicarbonate level (> 29 mmol/L), which occurs when there is both a respiratory and a metabolic cause present at the same time.

mucosa The outermost layer of the alimentary canal. It consists of three sublayers: surface epithelium, lamina propria, and muscularis mucosae.

Murphy's sign Pain in the right upper abdomen on deep inspiration during palpation.

muscularis externa The third tissue layer of the alimentary canal. It contains two levels in most places: the circular layer and the longitudinal layer.

ostomy A surgically created opening through which feces can be voided in the absence of some or all of the large intestine or rectum.

pancreatitis Inflammation of the pancreas leading to tissue destruction, improper functioning of the pancreas, and chronic disease.

paracentesis A "tapping" of the abdomen with a needle to draw off ascites.

penile fracture A rupture of one of the blood-containing sacs in the penis, resulting in deformity and possible loss of function.

peptic ulcer An erosion of the mucosal lining of the GI tract.

percutaneous endoscopic gastrostomy (PEG) tube A feeding tube that is placed through the abdominal wall into the stomach.

peristalsis A general term applicable to any concerted and directional movement in the body such as the movements that push food or urine through the body.

peritoneal dialysis Use of a catheter to introduce dialysate solution into the abdomen using the peritoneum as a natural semipermeable membrane to separate solutes.

phosphate buffer system The buffer system that functions in the renal tubules and intracellular fluids to convert strong acids or bases into weak acids or bases so that there is only a minimal effect on overall pH.

point-of-care (POC) testing Laboratory testing that is performed at the patient's beside, so that results can be quickly obtained and can be considered while decisions are still being made about patient care.

portal hypertension An increase in vascular resistance through the hepatic portal system. It can cause high venous pressure in gastric and esophageal veins, leading to varices, among other problems.

pressure ulcer A sore on the skin arising from prolonged pressure, classified in four stages, with stage 4 being the most severe with tissue necrosis and muscle and bone involvement. All stages are painful and

prone to infection; commonly seen in patients confined to a bed.

priapism Prolonged, painful erection of the penis.

pyelonephritis An infection of the kidney, typically the result of a urinary tract infection that traveled up the ureter.

reactive gastritis A chronic mucosal edema that results from recurring contact of the mucosa with antagonistic substances such as bile, pancreatic juice, or NSAIDs.

renal tubular acidosis (RTA) A form of metabolic acidosis caused by dysfunction of the kidneys or renal system; presents without an elevated anion gap; characterized into Type 1, 2, or 4.

respiratory acidosis A pathologic condition (blood pH < 7.35) resulting from the accumulation of acids in the body caused by a breathing problem or insufficient function of the respiratory system.

respiratory alkalosis A pathologic condition (blood pH > 7.45) resulting from the accumulation of bases in the body caused by a breathing problem or insufficient function of the respiratory system.

scintigraphy An imaging technology that is similar to angiography, except that the red blood cells themselves are radiologically labeled to allow greater specificity.

Sengstaken-Blakemore tube A tube with an inflatable balloon at its end that is inserted into the GI tract and inflated to tamponade bleeding.

serosa A protective layer of connective tissue over most of the alimentary canal; also called the visceral peritoneum.

stress-related erosive syndrome A condition in which small, diffuse peptic ulcers appear in critically ill patients in the intensive care unit, including Cushing's ulcers from head injuries and Curling's ulcers from severe burns.

submucosa The layer of connective tissue below the mucosa. Blood vessels, lymph, and nerves reside here.

systemic inflammatory response syndrome An immune response to acute pancreatitis that significantly worsens the patient's prognosis by expanding the scope of tissue damage.

testicular torsion Twisting of a testicle about the spermatic cord to the point of ischemia and possibly necrosis.

third spacing An abnormal increase in the amount of vascular fluid that flows into the intestinal lumen; it can lead to dehydration, electrolyte imbalances, and hypovolemia.

total parenteral nutrition (TPN) The IV administration of all necessary nutrients in a patient whose GI tract does not function.

transjugular intrahepatic portosystemic shunt The placement of a shunt in the abdomen that bypasses much of the hepatic portal system, intended to decrease portal hypertension and its effects.

T tube A T-shaped tube used to drain bile from the gallbladder.

type A gastritis A form of gastritis in which the intrinsic factor secreted by mucosal parietal cells is attacked by autoantibodies, ultimately resulting in pernicious anemia.

type B gastritis A form of gastritis caused by *Helicobacter pylori* and in which the degree of inflammation varies in severity.

ulcerative colitis An inflammation of the rectal mucosal and submucosal tissues.

urinary tract infection (UTI) A common type of infection of the urinary tract that can progress to major

conditions if not treated.

volvulus A twisting of the intestine onto itself, usually causing strangulation.

Zollinger-Ellison syndrome A condition in which a tumor in the GI tract secretes large amounts of acids.

Bibliography

Adams K. Anatomy and physiology of the renal system. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Adams K. Patient assessment: renal system. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Adams K. Patient management: renal system. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Amend W Jr, Vincenti F. Chronic renal failure and dialysis. In: Tanagho E, McAninch J, eds. *Smith's General Urology*, 16th ed. New York, NY: McGraw-Hill Co; 2004.

Amend W, Vincenti F. Oliguria: acute renal failure. In: Tanagho E, McAninch J, eds. *Smith's General Urology*, 16th ed. New York, NY: McGraw-Hill Co; 2004.

Campbell N, Reece J. Regulating the internal environment. In: *Biology*, 6th ed. Upper Saddle River, NJ: Pearson Education; 2002.

Casiday R, Frey R. Blood, sweat, and buffers: pH regulation during exercise. Washington University, Department of Chemistry Web site. Available at: http://www.chemistry.wustl.edu/~edudev/LabTutorials/Buffer/Buffer.html. Accessed September 12, 2008.

Chong SJ, Lim KB, Tan YM, Chow PKH, Yip SKH. December 12, 2004. Case report: atypical presentations of emphysematous cystitis. Available at: http://www.rcsed.ac.uk/journal/svol32/3020009.html. Retrieved November 25, 2008.

Clive DM. Metabolic acidosis and metabolic alkalosis. In: Irwin RS, Rippe JM, eds. *Manual of Intensive Care Medicine*, 4th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2006: 341–346.

Coleman J. Patient assessment: gastrointestinal system. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*. 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Day D, Jass J, Price AB, Shepherd NA, Sloan JM, Talbot NJ, et al. *Morson and Dawson's Gastrointestinal Pathophysiology*, 4th ed. Oxford, England: Blackwell Science Ltd; 2003.

Diepenbrock N. *Quick Reference to Critical Care*, 2nd ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2004.

Gehr T, Schoolwerth A. Clinical assessment of renal function. In: Fink M, Abraham E, Vincent J, Kochanek P, eds. *Textbook of Critical Care*, 5th ed. Amsterdam, The Netherlands: Elsevier, Inc; 2005.

Gould B. *Pathophysiology for the Health-Related Professions*, 3rd ed. Philadelphia, PA: W.B. Saunders Co; 2006.

Guyton AC, Hall JE. Regulation of acid-base balance. In: *Textbook of Medical Physiology*, 11th ed. Philadelphia, PA: Elsevier Saunders; 2006:383–401.

Hinkle C. Renal system. In: Chulay M, Burns S, eds. *AACN Essentials of Critical Care Nursing*. New York, NY: McGraw-Hill Co; 2006.

Hoffman RS, Nelson LS, Howland MA, Lewin NA, Flomenbaum NE, Goldfrank LR, eds. *Goldfrank's Manual of Toxicologic Emergencies*. New York, NY: McGraw-Hill Co; 2007:152.

Holcombe D, Feeley N. Renal failure. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Holleran RS, ed. *Air and Surface Patient Transport Principles and Practice*. St Louis, MO: Mosby; 2003.

Holzman N, Schirmer C, Nasraway S. Gastrointestinal hemorrhage. In: Fink M, Abraham E, Vincent J, Kochanek P, eds. *Textbook of Critical Care*, 5th ed. Amsterdam, The Netherlands; Elsevier, Inc; 2005.

Huang LH. August 13, 2008. Alkalosis, metabolic. Available at: http://www.emedicine.com/ped/TOPIC69.HTM. Accessed August 25, 2008.

Irwin R, Rippe J, Curley F, Heard S. *Procedures and Techniques in Intensive Care Medicine* , 3rd ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2003.

Jacobs DS, DeMott WR, Oxley DK. *Laboratory Test Handbook*. Hudson, OH: Lexi-Comp; 2001.

Krumberger J, Parrish C, Krenitsky J. Gastrointestinal system. In: Chulay M, Burns S, eds. *AACN Essentials of Critical Care Nursing*. McGraw-Hill Co; 2006.

Lanas A, Perez-Aisa MA, Feu F, et al. A nationwide study of mortality associated with hospital admission due to severe gastrointestinal events and those associated with nonsteroidal antiinflammatory drug use. *Am J Gastroenterol*. 2005;100(8):1685–1693.

Lipsett P. Acute pancreatitis. In: Fink M, Abraham E, Vincent J, Kochanek P, eds. *Textbook of Critical Care*, 5th ed. Amsterdam, The Netherlands: Elsevier, Inc; 2005.

Madigan M, Martinko J, Parker J. *Helicobacter pylori* and gastric ulcers. In: *Brock Biology of Microorganisms*. 12th ed. Upper Saddle River, NJ: Pearson Education; 2003.

Marieb E, Hoehn K. *Human Anatomy and Physiology*, 7th ed. San Francisco, CA: Benjamin/Cummings Publishing; 2009.

Merck Manual Professional Edition. Renal tubular acidosis (RTA). Available at: http://www.merck.com/mme/sec17/ch237/ch237f.html. Accessed September 27, 2008.

Molitoris B. Acute renal failure. In: Molitoris B, ed. *Critical Care Nephrology*. London, England: Remedica; 2005

Nicks BA, McGinnis HD, Megarbane B. Lactic Acidosis. April 9, 2008. Available at: http://emedicine.medscape.com/article/768159-overview. Accessed January 27, 2009.

O'Brien MC. July 2, 2007. Alcoholic ketoacidosis. Available at: http://www.emedicine.com/emerg/topic21.htm. Accessed September 28, 2008.

Okuda K, Mitchell D, Itai Y, Ariyama J. *Hepatobiliary Diseases: Pathophysiology and Imaging*. Oxford, England: Blackwell Science, Ltd; 2001.

Olson KR. Comprehensive evaluation and treatment. In: Olson KR, ed. *Poisoning and Drug Overdose*, 5th ed. New York, NY: Lange Medical; 2007:5–30.

Poole B, Schrier R. Acute renal failure. In: Fink M, Abraham E, Vincent J, Kochanek P, eds. *Textbook of*

Critical Care, 5th ed. Amsterdam, The Netherlands: Elsevier, Inc; 2005.

Raghavan M, Linden P. Fulminant hepatic failure, including acetaminophen toxicity. In: Fink M, Abraham E, Vincent J, Kochanek P. *Textbook of Critical Care*, 5th ed. Amsterdam, The Netherlands: Elsevier, Inc; 2005.

Rucker DW. February 12, 2008. Diabetic ketoacidosis. Available at: http://www.emedicine.com/emerg/topic135.htm. Accessed September 28, 2008.

Sherman SC, Schindlbeck M. When is venous blood gas analysis enough? *Emerg Med*. 2006;38(12):44–48.

Stavile KL. September 22, 2005. Metabolic acidosis. Available at: http://www.emedicine.com/emerg/topic313.htm. Accessed August 13, 2008.

Steele A, Sabol V. Anatomy and physiology of the gastrointestinal system. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Steele A, Sabol V. Common gastrointestinal disorders. In: Morton P, Fontaine D, Hudak C, Gallo B, eds. *Critical Care Nursing: A Holistic Approach*, 8th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

Tanagho E. Anatomy of the genitourinary tract. In: Tanagho E, McAninch J, eds. *Smith's General Urology*, 16th ed. New York, NY: McGraw-Hill Co; 2004.

Worthley LI. Acid-base balance and disorders. In: Bersten AD, Soni N, eds. *Oh's Intensive Care Manual*, 5th ed. New York, NY: Butterworth-Heinemann; 2003.


19

Endocrine Emergencies

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Objectives

1. Describe the anatomic structures of the endocrine system and their physiology (p 694–696).
2. Discuss the pathophysiology, assessment, and critical care transport management of conditions related to diabetes, including hypoglycemia, hyperglycemia, diabetic ketoacidosis, and hyperosmolar hyperglycemic nonketotic syndrome (p 696–701).
3. Discuss the pathophysiology, assessment, and critical care transport management of pituitary disorders, including central diabetes insipidus, pituitary lesions, acromegaly, and gigantism (p 701–702).
4. Discuss the pathophysiology, assessment, and critical care transport management of adrenal abnormalities, including adrenal insufficiency, Addison's disease, Cushing's disease, pheochromocytoma, aldosteronism, and amyloidosis (p 702–706).
5. Discuss the pathophysiology, assessment, and critical care transport management of thyroid abnormalities, including hyperthyroidism, hypothyroidism, myxedema coma, thyrotoxicosis, Hashimoto's disease, and thyroid cancers (p 706–708).
6. Discuss the pathophysiology, assessment, and critical care transport management of lipid disorders, including metabolic syndrome (p 709).

Introduction

Patients with an endocrine disorder will often present with a multitude of signs and symptoms that require a thorough knowledge of history of present illness or injury, the patient's medical history, and a detailed patient assessment. The CCTP will need to consider disorders that involve the endocrine system when assessing a patient's condition. For example, adrenal insufficiency after cardiac surgery can easily be overlooked while other symptoms are present during the unstable postoperative period. If unrecognized, this condition may cause serious morbidity and can be fatal.

Anatomy and Physiology

As a CCTP student, you are likely already familiar with the components of the endocrine system, which are reviewed in **Figure 17-1. Figure 17-2** provides a review of substances secreted by the pituitary gland and their destinations in the body.

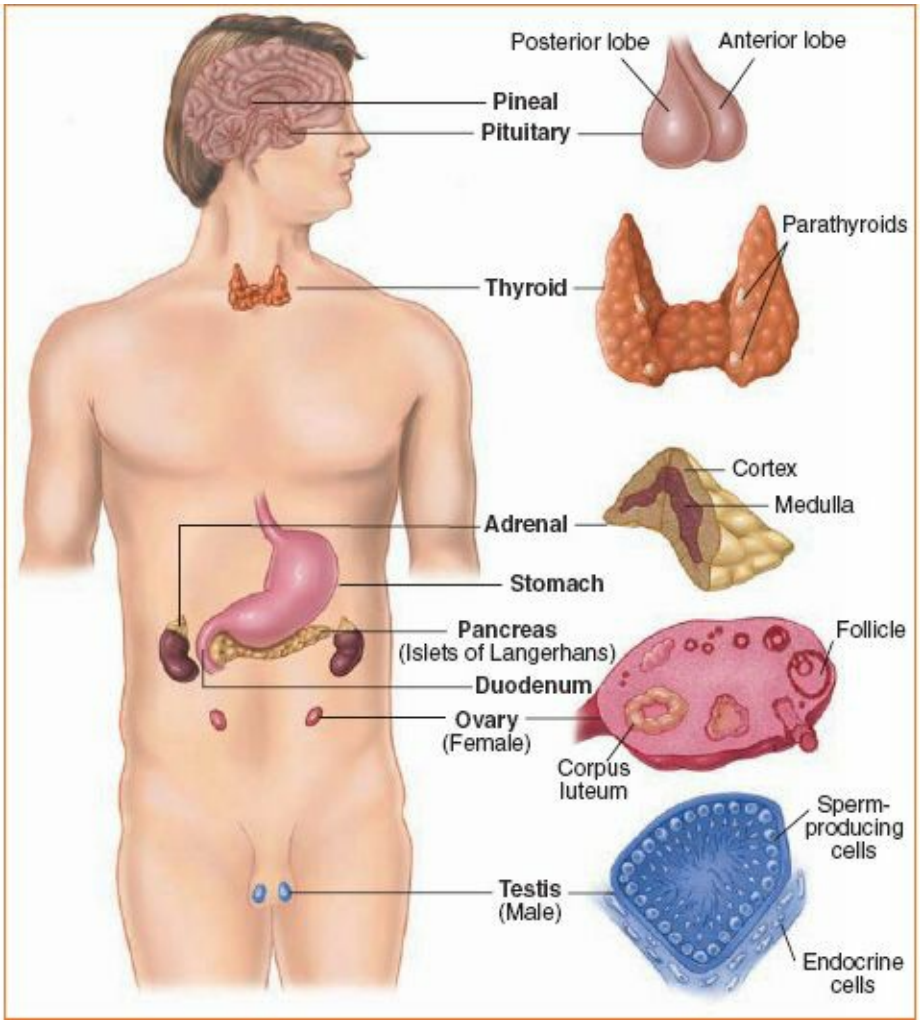

Figure 17-1 The endocrine system uses various glands to deliver chemical messages to organ systems throughout the body.

The Thyroid

The largest gland in the neck, situated in the anterior part of the neck below the skin and muscle layers, is the thyroid gland. It takes the shape of a butterfly, with the left and right thyroid lobes wrapping around the trachea. The function of the thyroid gland is to regulate the body's metabolism by secreting thyroxine when the body's metabolic rate decreases. Thyroxine, the body's major metabolic hormone, stimulates energy production in cells, which increases the rate at which cells consume oxygen and use carbohydrates, fats, and proteins. When the body gets cold, for example, the increased cellular metabolism creates heat. Iodine is an important component of thyroxine. Without the proper level of dietary iodine intake, thyroxine cannot be produced, and a person's physical and mental growth is diminished. The thyroid gland also secretes calcitonin, which helps maintain normal calcium levels in the blood. This hormone is secreted directly into the bloodstream when the thyroid detects high levels of calcium. Calcitonin travels to the bones, where it stimulates the bone-building cells to absorb the excess calcium. It also stimulates the kidneys to absorb and excrete excess calcium.

The location of the thyroid gland, the largest gland in the neck, is important. It is very vascular and if inadvertently nicked during an emergency cricothyrotomy, it will hemorrhage profusely.

The Adrenal Glands

Consisting of two parts, the adrenal glands produce hormones. The outer part is called the adrenal cortex, and the inner part is called the adrenal medulla **Figure 17-3**. Corticosteroids are hormones that are

created in the adrenal cortex. Corticosteroids regulate metabolism, the immune system, and sexual function; they also maintain the balance of salt and water within the body. The adrenal medulla produces catecholamines (the hormones epinephrine and norepinephrine). Catecholamines are involved in regulating autonomic functions, such as increasing the heart rate, respiratory rate, and blood pressure as needed when responding to stress.

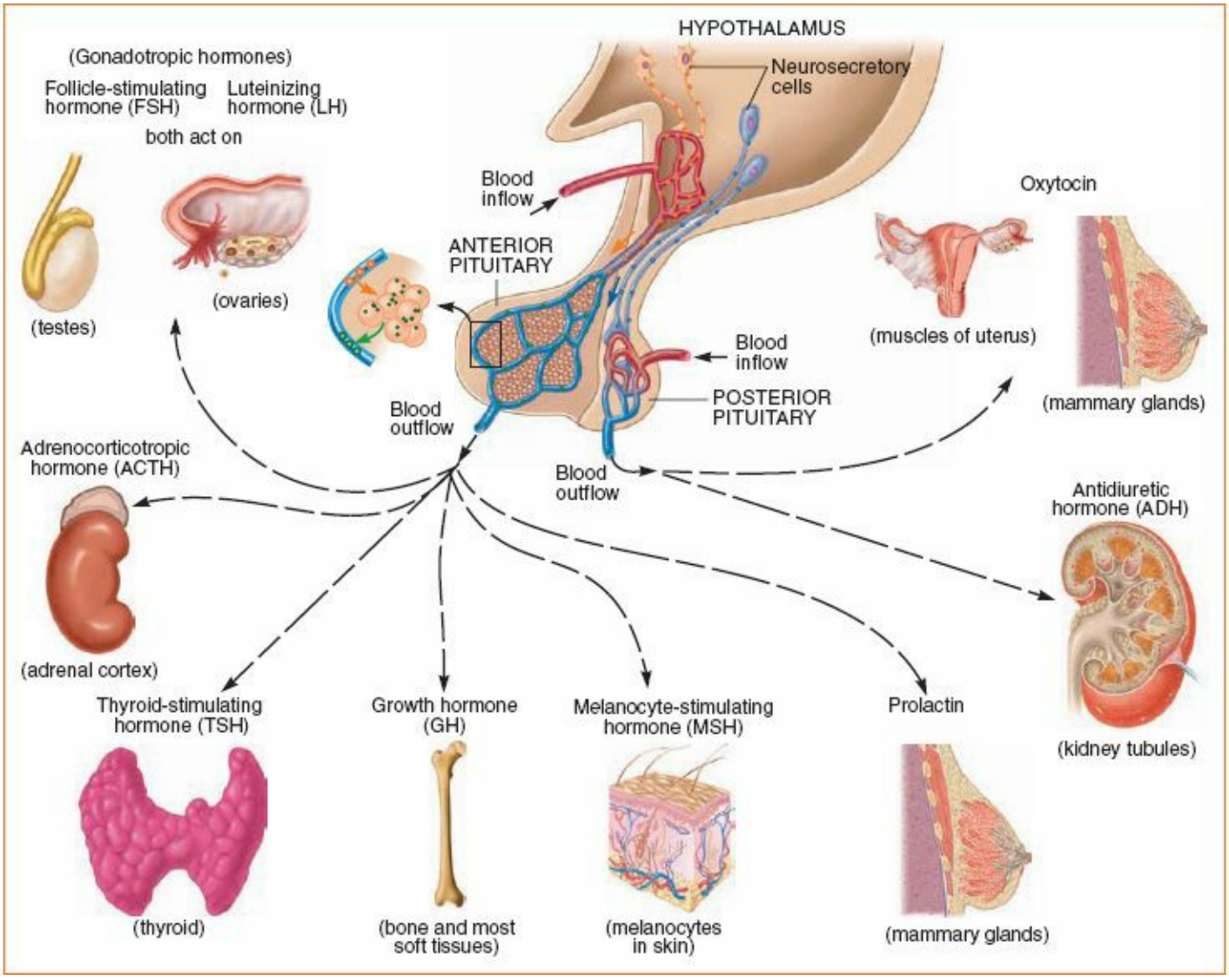

Figure 17-2 The pituitary gland secretes hormones from its two regions: the anterior pituitary lobe and the posterior pituitary lobe.

During times of stress, the hypothalamus secretes a hormone that stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH). ACTH targets the adrenal cortex, resulting in cortisol (a glucocorticoid) secretion. Cortisol stimulates most body cells to increase their energy production in response to increased stressors.

If the body experiences a drop in blood pressure or volume, a decrease in sodium level, or an increase in potassium level, the adrenal cortex is stimulated to secrete aldosterone (a mineralocorticoid). Aldosterone stimulates the kidneys to reabsorb sodium from the urine and excrete potassium by altering the osmotic gradient in the blood; this action increases both blood volume and blood pressure. Aldosterone also reduces the amount of salt and water lost through the sweat and salivary glands.

The body's reaction to physical or emotional stress is referred to as the "fight-or-flight" response. Following stimulation from the hypothalamus, the adrenal medulla secretes small amounts of norepinephrine and large amounts of epinephrine. Norepinephrine raises blood pressure by causing blood

vessels and skeletal muscles to constrict. Epinephrine stimulates sympathetic nervous system receptors throughout the body. In addition, epinephrine stimulates the liver to convert glycogen to glucose for use as energy in the cells. The action of both hormones results in increased levels of oxygen and glucose in the blood and faster circulation of blood to the brain, heart, and muscles. The body is then able to respond to the short-term emergency situation.

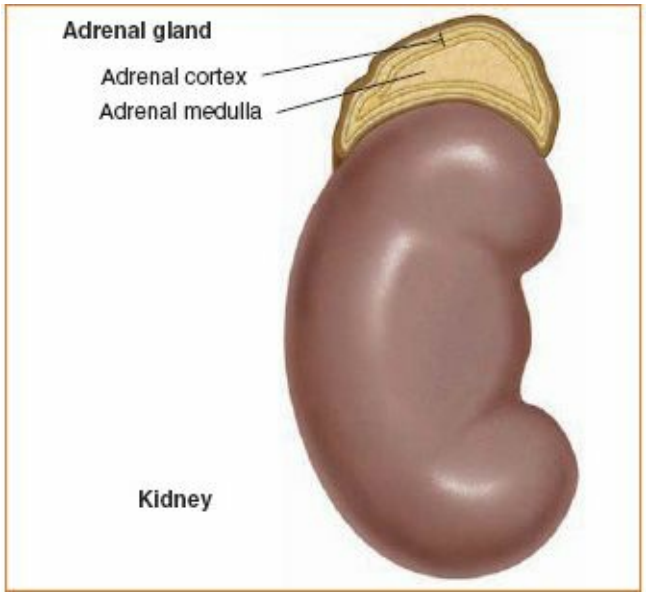

Figure 17-3 The adrenal glands, which sit on top of the kidney, consist of two parts: the adrenal cortex and the adrenal medulla.

Aldosterone

Aldosterone is the principal steroid hormone (mineralocorticoid family) that is produced in the zona glomerulosa (outer zone) of the adrenal cortex and acts primarily at the distal renal convoluted tube. When aldosterone is produced, its function is to increase the reabsorption of sodium and water and release potassium into the kidneys, thereby increasing circulating blood volume and blood pressure. Aldosterone is vital for the management of sodium in the kidneys, salivary glands, sweat glands, and colon.

Abnormal overproduction of aldosterone, resulting from either primary or secondary disorders, is common in the general population and is an important cause of morbidity and mortality.

Pancreas

The pancreas is a digestive gland that is considered an endocrine gland and an exocrine gland. It secretes digestive enzymes into the duodenum through the pancreatic duct. The exocrine component is responsible for the secretion of the digestive enzymes. The endocrine component comprises the islets of Langerhans. These cell groups within the pancreas act like “an organ within an organ.” The main hormones they secrete—glucagon and insulin—are responsible for the regulation of blood glucose levels **Figure 17-4**.

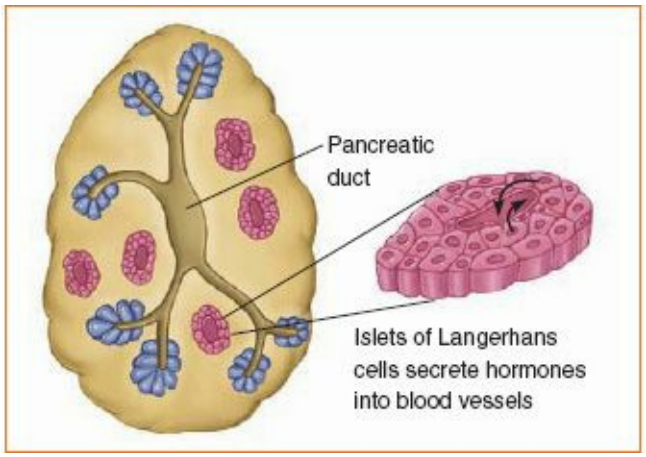

Figure 17-4 The islets of Langerhans secrete hormones into blood vessels.

When the body's blood glucose level falls, such as between meals, the hormone glucagon is secreted to raise the glucose level and bring the body's energy back to normal. When it enters the bloodstream, glucagon stimulates the liver to convert glycogen (a storage starch form of the sugar glucose made up of thousands of glucose units). The glucose is secreted into the bloodstream, where cells can use it for energy.

Insulin is responsible for the removal of glucose from the blood for storage as glycogen, fats, and protein. When blood glucose levels are elevated, the islets of Langerhans secrete insulin, which is carried by the bloodstream to the cells. The cells then take in more glucose to produce energy. Insulin also stimulates the liver to take in more glucose and store it as glycogen for later use by the body. Insulin is the only hormone that decreases the blood glucose levels. Insulin is essential in order for glucose to enter and nourish the cells. Once the blood glucose levels have returned to normal, the islets of Langerhans discontinue the secretion of insulin.

Diabetes

The term diabetes refers to a metabolic disorder in which the ability of the pancreas to metabolize simple carbohydrates (glucose) is impaired because of the lack of insulin or inadequate production of insulin. As mentioned, the pancreas is the primary blood glucose regulator. Digestive enzyme production and insulin and glucagon production take place in the islets of Langerhans. Beta cells in the islets of Langerhans produce insulin and amylin (65% to 80% of the islet cells), alpha cells release glucagon (15% to 20%), and delta cells produce somatostatin (3% to 10%). Insulin and glucagon are the hormones responsible for keeping the blood glucose level within normal ranges of 70 to 110 mg/dL. Diabetes, characterized by the passage of large quantities of urine containing glucose, can cause significant thirst and deterioration of body function.

Glucose that is not used immediately for energy is stored in the liver and muscles as glycogen, or converted by adipose tissue into fat. The liver releases glucose back into the bloodstream when the levels decrease and removes glucose when the levels are too elevated. Only glycogen in the liver can be converted back to glucose; the glycogen stored in muscle can be used only by the muscle for energy.

Insulin is secreted by the beta cells of the pancreas in response to blood glucose levels. After a meal, blood glucose levels rise, stimulating the pancreas to release insulin. The more glucose in the blood, the more insulin the pancreas releases. Insulin transports glucose into the cells via facilitated diffusion, where it is converted into energy. It also has an effect on many cells that play a part in absorbing the glucose, causing the blood glucose levels to return to normal.

Glucagon, which is secreted by the alpha cells, has the opposite effect. When blood glucose levels

are low, the pancreas releases more glucagon. The presence of glucagon stimulates the release of the glucose that is stored in liver cells, making energy available to the tissues between meals.

Diabetes mellitus results from an impairment of the body's ability to produce or use insulin. There are two major types of diabetes mellitus: type 1 and type 2.

Pathophysiology

Type 1 diabetes generally affects children and has historically been referred to as juvenile diabetes, although now patients are developing type 1 diabetes in adulthood as well. Type 1 diabetes has a hereditary predisposition. It is now believed, however, that environmental factors may be part of the cause—for example, an infection that triggers an autoimmune disorder (ie, antibodies destroy the islets of Langerhans).

In type 1 diabetes, most patients do not produce insulin. They require daily injections of supplementary, synthetic insulin throughout their lives to control blood glucose. In addition to daily replacement of insulin through injections or continuous infusion pump, and careful balancing of dietary intake of carbohydrates and simple sugars, physical activity must be in place to achieve good glucose control to avoid extreme highs and lows. This can be a particular challenge in young children, performance athletes, alcoholics, and patients with multiple medical problems.

The most common form of diabetes is **type 2 diabetes** (sometimes called adult-onset diabetes), in which blood glucose levels are elevated. Approximately 90% of all diabetics in the United States have type 2 diabetes, which typically develops later in life, usually when the patient is middle-aged, although the disease is becoming more common in younger people.

In many people with type 2 diabetes, the pancreas actually produces enough insulin; however, for reasons not fully understood, the body cannot effectively use it. This condition is known as **insulin resistance**. One possible explanation is that the insulin receptor cells located on the target cells have changed in some way and are no longer able to receive the insulin when it arrives at the target cell. Type 2 diabetes can also be caused by a deficiency in insulin production.

Hypoglycemia

Hypoglycemia in patients with diabetes is often the result of having taken too much medication, not having eaten enough food, unusual or unexpected physical activity, or high levels of alcohol consumption. The tissues of the central nervous system (including the brain) depend entirely on glucose as their source of energy, which is unlike other tissues, which can usually metabolize fat or protein in addition to glucose. If the blood glucose level drops dramatically, the brain is starved, and if hypoglycemia persists, cerebral dysfunction progresses very quickly to permanent brain damage.

Signs and Symptoms

Hypoglycemia

- Headache
- Altered mental status
- Memory loss
- Blurred or double vision
- Lack of coordination
- Slurred speech
- Stroke-like symptoms

- Seizure
- Hypertension
- Tachycardia
- Diaphoresis
- Chest pain
- Upper abdominal pain
- Tremor
- Anxiety
- Hostility
- Paranoia
- Hunger
- Coma

Normal blood glucose is approximately 70 to 110 mg/dL; hypoglycemia occurs when blood glucose drops to 45 mg/dL or less. Hypoglycemia can develop rapidly, from minutes to a few hours. It should be suspected in any patient with diabetes who presents with bizarre behavior, neurologic signs, or coma.

Of course, diabetics are not the only persons who are prone to episodes of hypoglycemia. Persons who are alcoholics, those who have ingested poisons or overdosed with drugs (notably aspirin), and those with certain cancers, liver disease, kidney disease, profound sepsis, and other conditions may also experience hypoglycemia.

Hyperglycemia and Diabetic Ketoacidosis

Hyperglycemia is one of the classic symptoms of diabetes mellitus and occurs when blood glucose levels exceed the normal range (70 to 110 mg/dL). In patients with a known history of hyperglycemia, physicians typically try to keep the glucose levels at less than 160 mg/dL. Hyperglycemia can be caused by excessive food intake, insufficient insulin dosages, infection or illness, injury, surgery, and emotional stress. Onset may be rapid (within minutes) or gradual (hours to days), depending on the cause. For example, excessive food intake may cause blood glucose levels to rise quickly, whereas an infection or illness will result in hyperglycemia over the course of several days. If left untreated, hyperglycemia in a patient with type 1 diabetes will progress to **diabetic ketoacidosis (DKA)**, a life-threatening condition that occurs when certain acids accumulate in the body because insulin is not available. Patients with this condition tend to be young—teenagers and young adults. In DKA, the deficiency of insulin prevents cells from taking up the extra glucose. Because the body cannot use glucose, it turns instead to other sources of energy—principally, fat. The metabolism of fat generates acids and ketones as waste products. (The ketones give the characteristic fruity odor to the breath of a patient with DKA.) Because glucose must be excreted in the urine in solution, the body loses excessive amounts of water and electrolytes (sodium and potassium). This may lead to disturbances in water balance and acid-base balance.

Special Populations

Pediatric patients with DKA can pose a greater management challenge than adult patients with DKA. Children are more susceptible to rapid shifts in fluid volume and osmolarity, and as such are at much greater risk for cerebral edema as a result of treatment. Although fluid administration is the mainstay of therapy for both children and adults with DKA, a more judicious approach must be used with the pediatric patient. The CCTP must meticulously monitor the inputs and outputs of the patient, and fluids

must be given in 20-mL/kg boluses with careful reassessment after each bolus. Most experts recommend a maximum of three boluses followed by one to one-and-one-half times maintenance fluids using either normal saline or one half normal saline based on age and electrolyte status. Additionally, insulin infusion should be between 0.05 and 0.1 U/kg/h, with a goal of lowering the glucose at a rate of about 50 mg/dL/h.

Meanwhile, glucose continues to accumulate in the blood. As the blood glucose level rises, the patient undergoes massive osmotic diuresis (passing large amounts of urine because of the high solute concentration of the blood). This diuresis, together with vomiting and the rapid, deep respirations will cause progressive dehydration and acidosis, ultimately leading to shock, coma, and death.

These processes usually progress slowly, during a period of 12 to 48 hours, with the patient's level of consciousness deteriorating only gradually.

Signs and Symptoms

Diabetic Ketoacidosis

- Fruity-smelling breath
- Polydipsia
- Polyuria
- Polyphagia
- Nausea and vomiting
- Abdominal pain
- Deep, rapid (Kussmaul) respirations
- Dehydration
- Hypotension
- Tachycardia
- Warm, dry skin and mucous membranes
- Confusion
- Gradual deterioration of consciousness

Hyperosmolar Hyperglycemic Nonketotic Syndrome

Hyperosmolar hyperglycemic nonketotic syndrome (HHNS) is a metabolic derangement that occurs principally in patients with type 2 diabetes. This condition is characterized by hyperglycemia, hyperosmolarity, and an absence of significant ketones. HHNS often develops in patients with diabetes who have some secondary illness that leads to reduced fluid intake. Although infection (in particular, pneumonia and urinary tract infection) is the most common cause, many other conditions can cause altered mentation or dehydration. In most cases, the secondary illness is not identified. Hyperglycemia and hyperosmolarity lead to osmotic diuresis and an osmotic shift of fluid to the intravascular space, resulting in further intracellular dehydration. Unlike patients with DKA, patients with HHNS do not experience ketoacidosis. Although most patients diagnosed as having HHNS have a known history of diabetes (usually type 2), approximately 30% do not have a prior diagnosis of diabetes. The stress response to any acute illness tends to increase hormones that favor elevated glucose levels; cortisol, catecholamines (epinephrine and norepinephrine), glucagon, and many others.

Signs and Symptoms

Hyperosmolar Hyperglycemic Nonketotic Syndrome

- Severe dehydration
- Drowsiness
- Lethargy
- Delirium
- Coma
- Focal or generalized seizures
- Visual disturbances
- Hemiparesis
- Sensory deficits
- Acute myocardial infarction

Assessment

Patients with diabetes mellitus may present with varied physical findings and vital signs on examination, depending on the current blood glucose level. The patient assessment, the patient's history of present illness or injury, and a thorough review of the patient's medical history will aid in determining if a diabetes-related problem is influencing the patient's vital signs and symptoms.

The assessment of the patient with hypoglycemia may reveal headache, altered mental status, memory loss, lack of coordination, strokelike symptoms, and possibly seizures. Hypertension, tachycardia, diaphoresis, chest pain, upper abdominal pain, and anxiety may be present as well because of the release of epinephrine from stimulation of the sympathetic nervous system.

It is important to rule out the possibility of hypoglycemia in patients presenting with signs of intoxication, including slurred speech, lack of coordination, paranoia, hostility, and aggressiveness. These signs may be the direct result of cerebral hypoxia, rather than alcohol or drugs.

Hyperglycemia and DKA typically present with the three Ps: polyuria, polydipsia, and polyphagia. **Polyuria** is a result of osmotic diuresis, **polydipsia** is caused by dehydration, and **polyphagia** is believed to be caused by the body's inefficient use of nutrients. Patients may also experience abdominal pain, which results in nausea and vomiting, thus increasing the degree of dehydration, causing hypotension; tachycardia; warm, dry skin; and dry mucous membranes. The two hallmark signs of DKA are deep, rapid (Kussmaul) respirations, which are the body's attempt to compensate for the resultant acidosis by "blowing off" carbon dioxide, and the fruity odor of ketones on the patient's breath.

Hyperosmolar HHNS presents similarly to DKA; however, it does not produce the two hallmark signs of DKA—Kussmaul respirations and fruity breath odors. Patients with HHNS typically present with severe dehydration and focal or global neurologic deficits, such as drowsiness and lethargy, delirium and coma, focal or generalized seizures, visual disturbances, hemiparesis, and sensory deficits. In addition, an acute myocardial infarction is frequently associated with HHNS. **Table 17-1** compares DKA with HHNS.

Laboratory Assessment

Testing for hypoglycemia or hyperglycemia is one of the limited testing procedures that a CCTP will be able to perform during transport, allowing for easy administration and/or titration of existing medications as a result of the biochemical analysis.

Because glucose is one of the three major energy sources for the body, it is important to routinely monitor the patient's blood glucose level during transport. Most commonly, this is accomplished via a

portable blood glucose monitor (glucometer) **Figure 17-5**, similar to the one the patient may use at home. The portable blood glucose monitor measures the glucose level in whole blood using either capillary or venous samples. Blood glucose testing performed in a laboratory measures the blood glucose level in the plasma, which is typically about 10 mg/dL higher than that in a whole blood sample.

It is important to read and understand the operator's manual prior to use because of variabilities in glucometer manufacturer specifications. Some glucometers will read "Lo" when a glucose reading below 20 mg/dL is detected, whereas others will display "Lo" when a reading below 30 mg/dL is detected. Inversely, the same is true with a "Hi" reading; some glucometers read "Hi" at 550 mg/dL, and some read "Hi" at 600 mg/dL, so it is important to know both the upper and lower ranges of the glucometer.

The normal lab value for glucose in whole blood in nonfasting adults and children is 70 to 110 mg/dL; in a neonate, blood glucose levels should be maintained above 65 mg/dL.

Arterial or venous blood gas monitoring should also be used in patients with DKA. Monitoring commonly reveals metabolic acidosis with a pH below 7.30, a bicarbonate level below 15 mEq/L, and a $Paco_2$ level below 30 mm Hg.

Laboratory testing for diabetics may also include a urine ketone test, which results in either a positive or a negative reading, depending on whether it detects ketones in the urine. Generally blood chemistries and blood testing for ketones is indicated to detect elevations in BUN and creatinine that may result from significant dehydration, detect electrolyte abnormalities (which can be profound), and to calculate anion gap which, when calculated in the presence of serum ketones, is used to guide ongoing insulin infusion therapy. The patient's creatinine and blood urea nitrogen levels may be elevated as well because the patient is dehydrated.

TABLE 17-1 Diabetic Ketoacidosis vs Hyperosmolar Hyperglycemic Nonketotic Syndrome

	Diabetic Ketoacidosis	**Hyperosmolar Hyperglycemic Nonketotic Syndrome**
Onset	Hours to days	Days to weeks
Cardiovascular	Tachycardia Hypotension	Tachycardia Hypotension
Respiratory	Kussmaul-type pattern Fruity odor	Normal to tachypnea Normal odor
Integumentary	Warm, dry Nonpyretic	Warm, dry Normal to pyrexia
Gastrointestinal	Polyphagia Polydipsia Polyuria	Polyphagia Polydipsia Polyuria
Urinalysis	Glucose present Ketones present	Glucose present Ketones absent
Blood chemistry	pH: < 7.30 Bicarbonate: < 15 mEq/L Glucose: > 300 mg/dL Sodium: normal Carbon dioxide: elevated	pH: normal Bicarbonate: normal Glucose: > 600 mg/dL Sodium: > 145 mEq/L Carbon dioxide: normal
	20-mL/kg bolus of isotonic crystalloid,	20-mL/kg bolus of isotonic crystalloid,

Treatment	repeated as needed IV insulin Continuous insulin infusion	repeated as needed IV insulin

Figure 17-5 A portable glucometer used in the critical care transport environment.

A diabetic patient may also have had a hemoglobin A1c, or glycohemoglobin, test completed prior to transport, although this test is typically not performed during an acute emergency. As a result of prolonged hyperglycemia, the red blood cells become saturated with glucose. A measurement is made using a percentage of saturation in an attempt to estimate the patient's blood glucose level during the previous 3 to 4 months. Typically, nondiabetic patients will have a hemoglobin A1c level between 4% and 6%. In diabetic patients, a measurement below 7.5% indicates good control, a measurement of 7.6% to 8.9% indicates fair control, and a measurement of 9.0% or more indicates poor control or no control of diabetes.

Management

Patients presenting with hypoglycemia should have the condition corrected prior to initiating transport. Standard treatment for hypoglycemia in adults is 50% dextrose; and in pediatric patients, 25% dextrose. However, the CCTP may be called on to transport a patient who remains hypoglycemic as a result of an intentional or unintentional insulin overdose. In these cases, a continuous dextrose infusion may be required. This may be accomplished by adding 50 g of 50% dextrose (two amps of D_{50}) to 1,000 mL of normal saline (making $D_{10}NS$) and titrating the infusion to the patient's blood glucose level.

As a result of profound dehydration in the hyperglycemic patient (with DKA and HHNS), fluid boluses with an isotonic saline solution should be initiated via an IV, intraosseous, or central line at a volume of 20 mL/kg, repeated as needed in an effort to stabilize the intravascular volume. After the intravascular volume has been normalized, short-acting insulin, such as a human insulin isophane suspension (Humulin N), should be administered at a dosage of 2 to 12 U IV, followed by a continuous insulin infusion of 2 to 10 U/h, in an attempt to decrease the blood glucose level to approximately 180 mg/dL. As the glucose is brought under control, the body begins to process the buildup of ketones that are the hallmark of DKA. The insulin infusion to treat DKA should be continued until there are no longer ketones present in the serum. For the CCTP, this may mean adding glucose to maintenance fluids and/or periodic administration of D_{50} in order to maintain glucose levels in the normal range while the insulin infusion is continued. In general, insulin drips should *not* be titrated based on glucose results in patients with DKA. During the hyperglycemic episode, the patient may become hypokalemic as well. An IV infusion of potassium should be initiated if the patient's serum potassium level is below 4.8 mEq/L via

laboratory studies. This infusion is typically accomplished by adding 40 mEq of potassium to 1,000 mL of normal saline and infusing at 5 to 10 mEq/h.

Patients being treated for hyperglycemic problems should have continuous cardiac monitoring for potential cardiac arrhythmias and hypokalemia. As with all patients the CCTP will encounter, definitive airway management and ventilatory assistance may be required. If there is a possibility of aspiration because of a decreased level of consciousness, insertion of a nasogastric tube should be considered. If urine output is desired during transport, an indwelling (Foley) catheter should be inserted as well.

Recent controversy has arisen with regard to the control of glucose levels in critically ill patients. Historically, continuous insulin infusions have been used in the intensive care unit to maintain an intensive control (81 to 108 mg/dL) of the glucose level as a means of reducing morbidity and mortality. In the March 26, 2009, issue of the *New England Journal of Medicine*, the Normoglycemia in Intensive Care Evaluation—Survival Using Glucose Algorithm Regulation (NICE-SUGAR) study found that by utilizing a conventional control (below 180 mg/dL) of glucose, there was an increased likelihood of morbidity and mortality if intensive controls were utilized, as opposed to conventional controls.

Transport Management

Hypoglycemia

- Test for hypoglycemia/hyperglycemia.
- Administer a continuous dextrose infusion, if required, and titrate the infusion to the patient's blood glucose level.

Numerous studies in the cardiac surgery patient subset demonstrate decreased infections and better survival when intensive controls are utilized, and it would stand to reason that these values could be applied to the rest of the critically ill patient population (noncardiac surgery patients); however, as a result of the NICE-SUGAR study, this seems untrue. This is not to say that insulin infusions are unhelpful in noncardiac surgery patients, only that an as-of-yet unknown target value should be utilized. The NICE-SUGAR study sought to determine optimal glucose levels to improve outcomes in critically ill patients and found only that intensive controls were less than optimal. This should not be interpreted as a call for abandoning good glucose management practices for critically ill patients.

CCTPs can expect to see continuous insulin infusions in almost any patient admitted to an intensive care unit who subsequently requires transfer to another hospital. When insulin infusions are used in critically ill patients, there is an appreciable decrease in both mortality and morbidity. The controversy is as follows: (1) whether the increased survival results from the insulin infusion or the control of the blood glucose level and (2) what is the optimal blood glucose target for improved outcomes in critically ill patients.

To add to the controversy, most institutions have their own protocols for insulin infusions. All are very different, and the incidences of hypoglycemia between the various protocols vary widely. Research has shown that hypoglycemic episodes increase morbidity and mortality and may negate the benefits of the insulin infusion in some patient populations.

Nevertheless, the CCTP should understand that insulin infusion is a routine part of care, requiring vigilance in monitoring during transport, including frequent (hourly or more often) checks of blood glucose levels using a blood glucometer, and may require adjustments in the insulin infusion rate to maintain the blood glucose targets prescribed for that patient.

Transport Management

Hyperglycemia (DKA and HHNS)

- Replenish fluids via an IV, an intraosseous, or a central line.
- Administer short-acting insulin, such as Humulin, followed by a continuous insulin infusion.
- Administer sodium bicarbonate for acidosis.
- Administer an IV infusion of potassium if the patient becomes hypokalemic.
- Provide continuous cardiac monitoring.
- Provide airway management and ventilatory assistance if required.
- Insert an indwelling (Foley) catheter if urine output is desired.

TABLE 17-2 Common Medications Used to Treat Diabetes Mellitus

Medication Class	Examples
Sulfonylureas	Acetohexamide (Dymelor) Chlorpropamide (Diabinese) Tolbutamide (Orinase) Tolazamide (Tolinase) Glipizide (Glucotrol) Gliclazide (Glipizide) Glibenclamide (Glyburide) Gliquidone (Glurenorm) Glimepiride (Amaryl)
Biguanides	Metformin (Glucophage)
Thiazolidinediones	Rosiglitazone (Avandia) Pioglitazone (Actos)
Alpha-glucosidase inhibitors	Acarbose (Precose) Miglitol (Glyset)
Meglitinides	Repaglinide (Prandin) Nateglinide (Starlix)
Dipeptidyl peptidase IV (DPP-IV) inhibitors	Vildagliptin (Galvus) Sitagliptin (Januvia) Saxagliptin (Onglyza) Linagliptin (Ondero)
Carbohydrates	50% Dextrose in water 25% Dextrose in water 10% Dextrose in water
Pancreatic hormones	Glucagon (GlucaGen) Insulin Premixed insulin – Humulin 70/30

	– Novolin 70/30 – NovoLog 70/30 – Humulin 50/50 – Humalog mix 75/25
Rapid-acting insulin	Humalog mix NovoLog
Short-acting insulin	Humulin Novolin
Intermediate-acting insulin	Neutral Protamine Hagedorn Lente
Long-acting insulin	Ultralente Insulin glargine (Lantus)

Table 17-2 lists common medications used to treat diabetes mellitus.

Pituitary Disorders

When a patient is experiencing a pituitary disorder, the presenting signs and symptoms commonly mirror other disease processes, requiring you to obtain a thorough medical history and conduct a physical exam in order to narrow down the condition to a potential pituitary problem. Failure to recognize these disorders, although they are seldom life threatening, could have an impact on your patient's well being.

Pathophysiology

The pituitary gland, commonly referred to as the "master gland," secretes hormones that regulate the secretions of other endocrine glands. Located at the base of the brain, approximately the size of a grape, it is attached to the hypothalamus by a very thin piece of tissue. This gland is divided into the following two lobes: (1) the anterior pituitary, which produces and secretes six hormones (growth hormone, thyroid-stimulating hormone [TSH], ACTH, and three gonadotropic hormones); and (2) the posterior pituitary, which secretes two hormones (antidiuretic hormone and oxytocin) but does not produce them.

Central Diabetes Insipidus

Central diabetes insipidus occurs when the posterior part of the pituitary gland is damaged, which causes a lack of production of the antidiuretic hormone vasopressin. The damage may be caused by traumatic head injuries, neurosurgery, or genetic abnormalities. Because of the lack of antidiuretic hormone, patients frequently experience unregulated fluid losses, which leads to dehydration.

Signs and Symptoms

Central Diabetes Insipidus

- Polyuria
- Polydipsia
- Dehydration

Pituitary Lesions

Pituitary lesions can be classified into the following two types: functioning adenomas and nonfunctioning adenomas. **Nonfunctioning adenomas**, which account for 40% of pituitary lesions, are tumors that do not secrete hormones. With **functioning adenomas**, which make up the remaining 60% of pituitary tumors, overproduction of hormones can occur depending on the type of hormone the adenomas produce:

- ACTH-secreting adenomas, which are responsible for Cushing's disease
- Growth hormone-secreting adenomas, which are responsible for acromegaly and gigantism
- Prolactin-secreting adenomas, which are responsible for gynecologic problems

Acromegaly and Gigantism

Acromegaly is a syndrome that results from excessive growth hormone, secreted by the pituitary gland *after* the epiphyseal plate has closed. Commonly affecting middle-aged adults, acromegaly can result in disfigurement and premature death if left untreated. Because of the slow progression of the syndrome, definitive diagnosis often goes undiagnosed for many years, until enlargement appears in the hands, feet, and face. Acromegaly is often associated with gigantism.

Signs and Symptoms

Acromegaly and Gigantism

- Abnormally fast growth of bones, muscles, and internal organs

TABLE 17-3 Common Medications Used to Treat Acromegaly and Gigantism

Medication Class	Examples
Dopaminergic agonist	Bromocriptine (Parlodel)
Somatostatin analog	Octreotide (Sandostatin) Lanreotide (Somatuline)
Growth hormone receptor antagonist	Pegvisomant (Somavert)

Gigantism, on the other hand, results from excessive growth hormone secreted by the pituitary gland *before* the epiphyseal plate has closed.

Regardless of the onset (childhood or adulthood), both of these conditions are characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate. Acromegaly and gigantism are almost always caused by a benign pituitary tumor.

Assessment

The assessment of patients presenting with acromegaly or gigantism will follow the same assessment steps as other patients, ensuring that a detailed medical history and answers to questions about weight gain as well as excessive and rapid growth are obtained.

Laboratory Assessment

Patients presenting with acromegaly and gigantism typically present with elevated creatinine levels (> 1.2 mg/dL). Because acromegaly and gigantism are a result of excessive growth hormone production, growth hormone levels will be elevated (> 18 ng/mL).

Management

There are currently two medications that are used to treat acromegaly and gigantism. Bromocriptine (Parlodel), which is a dopaminergic agonist, is used to decrease growth hormone secretion. Octreotide (Sandostatin) and lanreotide (Somatuline), synthetic forms of the hormone somatostatin, stop growth hormone production entirely. A recent development of acromegaly and gigantism is the use of pegvisomant (Somavert), which is a growth hormone receptor antagonist. Pegvisomant is able to control acrogmegaly and gigantism in virtually all patients because of the blocking action of the endogenous growth hormone molecules.

Table 17-3 lists common medications used to treat acromegaly and gigantism.

Adrenal Abnormalities

There are many abnormalities of the adrenal glands. Some are hereditary (familial adrenal hypoplasia syndromes), others are steroid resistance and hypersensitivity abnormalities such as glucocorticoid resistance syndromes and states, and there are genetic disorders such as adrenal insufficiency as a result of x-linked adrenoleukodystrophy.

Pathophysiology

Conditions associated with **pseudo-Cushing's states** are a mixed group of disorders that are related to increased cortisol production. The physiologic conditions that cause increased cortisol production include surgery-associated stress, severe illness, emotional stress, intense aerobic exercise, and caloric restriction. Nonphysiologic conditions that may elevate cortisol production include chronic alcoholism and alcohol withdrawal syndrome, poorly controlled diabetes mellitus, and obesity.

Endocrine hypertension is a result of hormonal disorders that cause clinically significant hypertension. The most common causes of endocrine hypertension are excess production of mineralocorticoids, such as aldosterone; catecholamines, such as epinephrine, norepinephrine, and dopamine; and glucocorticoids, such as cortisol. The cause of endocrine hypertension often relates to excess hormone produced by a tumor. Endocrine hypertension can be treated with surgery or antihypertensive therapy.

Adrenal Insufficiency

Underproduction of cortisol and aldosterone caused by decreased functioning of the adrenal cortex is called **adrenal insufficiency (AI)**. When at least 90% of the adrenal cortex has been damaged, AI occurs. A function of cortisol is to help the body respond to stress, including stress caused by surgery, illness, or infection. The hormone also aids in maintaining normal blood pressure and cardiovascular functions and regulating the metabolism of proteins, carbohydrates, and fats. AI may affect the adrenal gland (idiopathic adrenal insufficiency) or other glands (polyendocrine deficiency syndrome).

Primary causes of adrenal insufficiency include:

- Autoimmune disorders (such as with Hashimoto's disease)
- Illness or increased stress Genetic disorders
- Renal injuries
- Radiation therapy
- Surgery Infections
- Pituitary and hypothalamic lesions

Men and women of all ages are equally affected by AI. Causes of permanent AI include Addison's

disease, congenital adrenal hyperplasia, and complete surgical removal of the pituitary gland or the adrenal glands. Temporary AI can be caused by physical stress, infections, surgery, or failure to take corrective medication as required.

In the patient with chronic AI, signs and symptoms of **Addisonian crisis** may appear suddenly as a result of an increased period of stress, trauma, surgery, or severe infection. The primary clinical manifestation of Addisonian crisis is shock. Other symptoms include weakness, altered mental status, hyperthermia, and severe pain in the lower back, legs, or abdomen. Severe vomiting and diarrhea may precipitate dehydration in these patients. The ACTH stimulation test is the most specific test for diagnosing AI. The test measures blood cortisol levels (normal range, 10 to 60 ng/L). Undiagnosed, AI is a potentially fatal disease that usually results in death from hypotension or cardiac arrhythmias as a result of hyperkalemia. Acute AI may present in postsurgical or trauma patients. One study showed that as many as 5% of general surgical patients were affected by postoperative AI.

Signs and Symptoms

Addisonian Crisis

- Shock
- Weakness
- Altered mental status
- Hyperthermia
- Severe pain in the lower back, legs, or abdomen
- Severe vomiting and diarrhea
- Dehydration

Patients with moderate or severe traumatic brain injury present with some form of AI in approximately 50% of injury cases. Patients with traumatic brain injury receiving high-dose pentobarbital or propofol as well as patients with vasopressors used to manage lower blood pressures should be carefully monitored for AI.

Addison's Disease

Addison's disease is a chronic hormonal or endocrine disorder that occurs in patients of all age groups, caused by a deficiency in cortisol and/or aldosterone. Addison's disease is characterized by weakness, fatigue, hypotension, unexplained weight loss, and darkening of the skin **Figure 17-6**. Although other AI conditions are acute, Addison's disease is chronic.

Most cases of Addison's disease are caused by the slow destruction of the adrenal cortex. Addison's disease affects about 1 in 100,000 people.

Treatment of Addison's disease is the same as that of acute AI conditions. Prognosis is good for well-managed adult-onset Addison's disease, with a normal average mortality rate; however, recent studies show that young patients with Addison's disease are at risk for premature death. In patients diagnosed at a young age, Addison's disease is still a potentially lethal condition, with excess mortality in acute adrenal failure, infection, and sudden death.

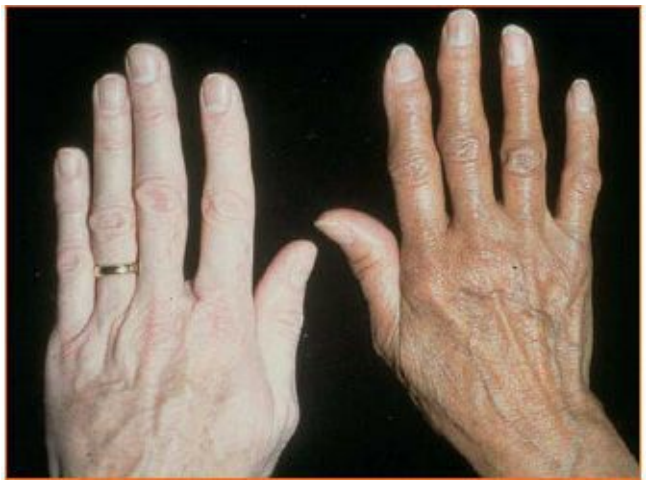

Figure 17-6 The hand of a patient with Addison's disease (right) compared with the hand of a healthy subject (left).

Signs and Symptoms

Addison's Disease

- Weakness
- Fatigue
- Hypotension
- Unexplained weight loss
- Darkening of the skin

Cushing's Syndrome

Cushing's syndrome, also known as hypercortisolism, is caused by prolonged exposure to elevated levels of cortisol in the body. The syndrome is relatively rare and affects persons between 20 and 50 years old. Risk factors for Cushing's syndrome are obesity, poorly controlled type 2 diabetes, and hypertension. Patients taking glucocorticoids, such as prednisone for inflammatory diseases, are also at risk for developing Cushing's syndrome.

Regardless of the cause, excess cortisol causes characteristic changes in many body systems. The metabolism of carbohydrates, protein, and fat is disturbed, causing hyperglycemia. Protein synthesis is impaired so that body proteins are broken down, which leads to loss of muscle fibers and muscle weakness. Bones become weaker and more susceptible to fracture. Other common signs and symptoms include the following:

- Weakness and fatigue
- Depression and mood swings
- Increased thirst and urination
- Weight gain, especially on the abdomen, face ("moon face") **Figure 17-7**, neck, and upper back ("buffalo hump") **Figure 17-8**
- Thinning of the skin, with easy bruising and pink or purple stretch marks (striae) on the abdomen, thighs, breasts, and shoulders

Signs and Symptoms

Cushing's Syndrome

- Hyperglycemia
- Weakness and fatigue
- Depression and mood swings
- Increased thirst and urination
- Weight gain (especially on the abdomen, face, neck, and upper back)
- Thinning of the skin; easy bruising and pink or purple stretch marks on the abdomen, thighs, breasts, and shoulders
- Increased acne and facial hair growth; in women, scalp hair loss and cessation of menstrual periods
- Darkening of skin on the neck
- Obesity and poor growth in height in children

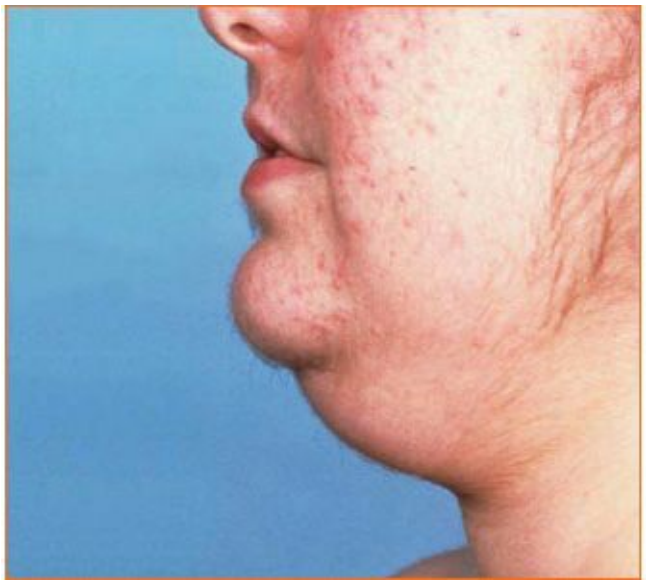

Figure 17-7 Weight gain in the face as a result of Cushing's syndrome.

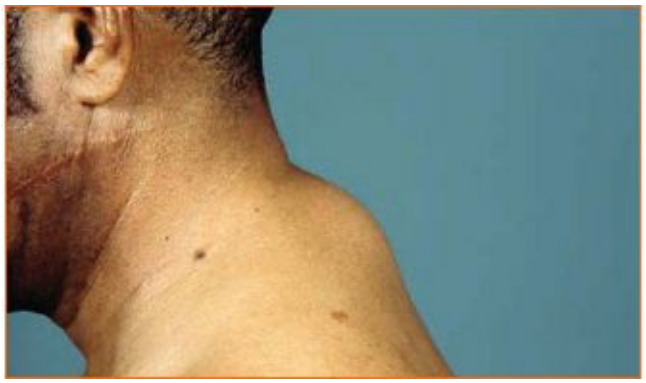

Figure 17-8 Weight gain in the upper part of the back as a result of Cushing's syndrome.

- In women increased acne, facial hair growth, scalp hair loss, and cessation of menstrual periods
- Darkening of skin (acanthosis) on the neck
- Obesity and poor growth in height in children

Pheochromocytoma

A **pheochromocytoma** is a catecholamine-producing benign tumor of chromaffin cells, located in the center of the adrenal gland, which can occur either sporadically or chronically as a result of genetic risk factors. The catecholamines produced include epinephrine, norepinephrine, and occasionally dopamine. This catecholamine release causes stimulation of alpha-adrenergic receptors, resulting in hypertension, increased cardiac contractility, glycogenolysis, gluconeogenesis, and intestinal relaxation. Stimulation of the beta-adrenergic receptors results in an increase in heart rate and contractility. Although the majority of the pheochromocytomas are found in the adrenal glands, pheochromocytoma can be found anywhere there is chromaffin tissue. Of pheochromocytomas, 97% are in the abdomen, 2% are in the thorax, and the remaining 1% are in other body areas.

Investigation of sustained hypertension leads to a diagnosis of pheochromocytomas in 0.05% to 0.2% of patients. Pheochromocytoma can be corrected in about 90% of cases if identified. If left untreated, fatal events such as catecholamine-induced malignant hypertension, heart failure, myocardial infarction, stroke, or ventricular arrhythmias occur.

Definitive treatment of pheochromocytoma is surgery to remove the tumor. Patients with pheochromocytoma should be treated with appropriate preoperative medical management to block the effects of released catecholamines.

Patients can experience a hypertensive crisis as a result of the rapid increase in catecholamines released from the tumor. Patients may present with a hypertensive crisis in varying ways, such as severe headaches or diaphoresis, visual disturbances, palpitations, epistaxis, acute myocardial infarction, congestive heart failure, or cerebrovascular accidents. Antihypertensive therapy must begin immediately. Treatment of a hypertensive crisis as a result of pheochromocytoma should include phentolamine. The dosage is an IV bolus of 2.5 to 5 mg at 1 mg/min. Because of the short half-time of phentolamine, this dose can be repeated every 5 minutes until hypertension is adequately controlled (decreasing the systolic blood pressure by no more than 20% to 25%). A continuous infusion of phentolamine (100 mg of phentolamine in 500 mL of 5% dextrose in water) titrated to the patient's blood pressure may be preferred to bolus dosing.

Some patients remain hypertensive immediately following surgery, although the majority present with hypotension that requires treatment with fluids. Hypoglycemia is another complication after surgery that is prevented by infusion of 5% dextrose started immediately after tumor removal and continuing for several hours thereafter. Post-operative hypoglycemia is transient, whereas hypotension may continue for several days or more after surgery.

Signs and Symptoms

Pheochromocytoma

- Hypertension
- Increased heart rate
- Severe headache
- Diaphoresis
- Visual disturbances
- Palpitations
- Chest pain
- Upper abdominal pain
- Epistaxis

- Anxiety
- Acute myocardial infarction
- Congestive heart failure
- Cerebrovascular accident

Transport Management

Pheochromocytoma

- Administer an IV bolus or a continuous infusion of phentolamine.

The long-term prognosis of patients after pheochromocytoma excision is excellent; however, nearly half of these patients will experience resultant life-long hypertension.

Aldosteronism

Aldosteronism is a syndrome of high blood pressure and low blood potassium levels caused by an excess of aldosterone. There are two main types of aldosteronism, primary and secondary.

Primary aldosteronism is usually caused by a tumor of a single adrenal gland that overproduces aldosterone. This is also known as Conn's syndrome. More than 95% of the cases are benign. Rarely, however, these tumors may be malignant. **Secondary aldosteronism** results from other conditions not associated with the adrenal gland. It can be caused by obstructive renal artery disease, renal vasoconstriction, and edematous disorders, and occurs as a result of reduced renal blood flow. The reduced blood flow stimulates hypersecretion of aldosterone.

Aldosteronism is suspected in patients with hypertension and low blood potassium levels, because aldosterone's normal function is to increase sodium and fluid in the bloodstream and potassium excretion in the kidney. Elevated aldosterone levels can be measured via blood or urine chemistry. A special blood test called plasma renin activity is measured to distinguish between primary aldosteronism (low plasma renin activity) and secondary aldosteronism (high plasma renin activity). Once blood chemistry testing is completed, a computed tomo-graphic (CT) scan of the abdomen may be performed to confirm the location of the disease. Surgery is the treatment for aldosteronism if the primary cause is a single adenoma. When the cause does not lie within the adrenal gland, treatment is to correct the underlying condition that led to the elevated aldosterone levels.

Signs and Symptoms

Aldosteronism

- Hypertension
- Low blood potassium levels
- Alkalosis
- Muscular weakness
- Polyuria
- Polydipsia
- Edema
- Heart failure

- Hepatic cirrhosis

Amyloidosis

Amyloidosis is a group of diseases that result from abnormal deposits of the protein amyloid in various tissues of the body. This protein can be deposited in a localized area or it may affect tissues throughout the body in a systemic fashion. Systemic amyloidosis can cause serious changes in virtually any organ of the body and can cause the affected organs to fail.

Signs and Symptoms

Amyloidosis

- Pedal edema
- Weight loss
- Dyspnea
- Weakness
- Diarrhea
- Fatigue
- Cardiac arrhythmias

Primary amyloidosis is the most common form of amyloidosis and affects the heart, kidneys, tongue, nerves, and intestines. Primary amyloidosis is associated with multiple myeloma, a form of bone marrow cancer, in a minority of cases. Primary amyloidosis is classified as an apolipoprotein.

Secondary amyloidosis occurs as a result of another illness, such as multiple myeloma, chronic infections, or chronic inflammatory diseases. Secondary amyloidosis is classified as an amyloid A protein. The disease primarily affects the kidneys, spleen, liver, and lymph nodes, although other organs may be involved. Treatment of the underlying disease may help stop this form of amyloidosis. There is no known cure for amyloidosis. Therapies are directed at managing symptoms and limiting the production of amyloid protein.

Assessment

Patients with adrenal abnormalities may present with varied physical findings and vital signs on examination. The patient assessment, history of present illness or injury, and thorough review of medical history will aid in determining whether adrenal abnormalities are influencing the patient's vital signs and symptoms. It is important to emphasize the need to be aware of the possibility of adrenal complications to aid in the correct diagnosis. Treatment of adrenal abnormalities could easily escape recognition and, thus, contribute to needless increased morbidity and mortality.

In addition to the medical history and physical exam, a diagnosis of Cushing's syndrome or Cushing's disease requires laboratory studies demonstrating high levels of blood cortisol.

The assessment of the patient with pheochromocytoma may reveal hypertension and other symptoms associated with catecholamine release, such as tachycardia, diaphoresis, chest pain, upper abdominal pain, and anxiety.

Primary aldosteronism typically presents with hypokalemia, alkalosis, muscular weakness, polyuria, polydipsia, and hypertension.

Secondary aldosteronism is commonly associated with edematous states, heart failure, hepatic cirrhosis, and malignant hypertension.

Signs and symptoms of amyloidosis depend on the organ system affected and include pedal edema, weight loss, dypsnea, weakness, diarrhea, fatigue, and cardiac arrhythmias.

Laboratory Assessment

Laboratory testing is required to definitively diagnose these underlying health factors and will vary with the disease process.

TABLE 17-4 Common Medications Used to Treat Adrenal Conditions

Medication Class	Examples
Adrenal Insufficiency Synthetic glucocorticoids	Fludrocortisone acetate (Florinef), taken once a day
Cushing's Syndrome Synthetic cortisol	Ketoconazole (Nizoral), mitotane (Lysodren), metyrapone (Metopirone), prednisone, hydrocortisone, dexamethasone (Dosages depend on the cause of the syndrome and the treatment plan).
Pheochromocytoma Antihypertensives	Phentolamine: IV bolus of 2.5 to 5 mg at 1 mg/min; sodium nitroprusside (preparation similar to phentolamine) at 0.5 to 10.0 μg/kg/min (stop if no results are seen after 10 min)
Aldosteronism	Treatment for primary aldosteronism depends on the underlying cause
Amyloidosis	Treatment is directed at managing symptoms and limiting the production of amyloid protein; it includes melphalan (Alkeran or Alkeran IV), a chemotherapy agent, and dexamethasone; new drugs being tested for use in amyloidosis include bortezomib (Velcade), thalidomide (Thalomid), and lenalidomide (Revlimid)

The ACTH stimulation test, along with the cortisol test, is the most specific combination of tests for diagnosing Addison's disease. In these tests, a synthetic form of ACTH is injected, then blood and urine cortisol are measured before and after the injection is given. A healthy patient will exhibit a rise in blood and urine cortisol levels after an injection of ACTH, but patients with adrenal insufficiency will have less of an increase, or no increase. The normal range for cortisol levels in adults is 5 to 25 μg/dL. The normal range for aldosterone levels in adults is 2 to 9 ng/dL.

No increase in blood and urine cortisol levels indicates that the pituitary gland is the origin of the condition, whereas a delayed increase indicates that the hypothalamus is the origin of the condition. More specifically, patients with primary adrenal insufficiency have high ACTH levels but do not produce cortisol. Patients with secondary adrenal insufficiency have decreased cortisol responses but absent or delayed ACTH responses.

Unlike adults, children are more likely to have absolute adrenal insufficiency, defined by a basal cortisol level of less than 18 μg/dL and a peak ACTH-stimulated cortisol concentration of less than 18 μg/dL. Patients at risk of inadequate cortisol or aldosterone production in the setting of shock include children with purpura fulminans and Waterhouse-Friderichsen syndrome, children who previously received corticosteroid therapies for chronic illness, and children with pituitary or adrenal insufficiency.

Management

Treatment of adrenal diseases usually involves replacing, or substituting, the hormones that the adrenal glands are not making and varies depending on the cause of the condition and the clinical presentation and severity of symptoms.

The initial treatment of an acute adrenal crisis is the correction of hypovolemic shock via fluid resuscitation. Large volumes of an isotonic crystalloid (500 to 1,000 mL) solution should be given by IV or intraosseous routes. For a patient diagnosed with glucocorticoid deficiency, IV dexamethasone sodium phosphate or hydrocortisone sodium succinate may be administered. Dexamethasone has a long duration of action and does not interfere with serum or urinary steroid testing; therefore, it is preferable.

Table 17-4 lists common medications used to treat adrenal conditions. Although treatment of the patient with pituitary disorders in the acute setting is rare, the CCTP should appreciate the impact that these disease processes have on the patient's day-today life, and should keep a high index of suspicion when confronted with the patient who may be presenting with these signs and symptoms.

Thyroid Abnormalities

As a CCTP, you will seldom be called upon to transport a patient with a life-threatening condition as a result of a thyroid abnormality. However, with a high index of suspicion and available laboratory studies, you should be in a position to appreciate the long-term outlook of the patient's care in relation to your care of the patient's concurrent medical problems.

Pathophysiology

The thyroid gland, as a primary controller of the body's metabolism, produces the hormones T3 (triiodothyronine) and T4 (thyroxine). These hormones cause an increase in organ function in all organs to which they are exposed.

Hypothyroidism

Hypothyroidism is caused by a deficiency of T3 and T4, both of which are secreted by the thyroid gland. Because of the decrease in the production, the body's organ function slows, causing the patient to feel a decrease in body temperature, to have gradual weight gain, and to have an increased risk for acute MI and CVA. In the critical care patient, a good history will help with the diagnosis of hypothyroidism.

Signs and Symptoms

Hypothyroidism

- Decrease in body temperature
- Gradual weight gain
- Lethargy
- Constipation
- Muscle aches and weakness
- Joint pain, stiffness, or swelling
- Puffy face
- Pale, dry skin
- Brittle fingernails and hair
- Hoarse voice

- Depression

Hyperthyroidism

Hyperthyroidism is caused by an increased production of T3 and T4. Because of the increase in the body's metabolism as a result, patients with hyperthyroidism experience an increase in body temperature and gradual weight loss, even if they are eating the same amounts of food. Other side effects of hyperthyroidism are increased and/or irregular heart rate, sweating, and irritability.

Signs and Symptoms

Hyperthyroidism

- Increase in body temperature
- Gradual weight loss
- Increased and/or irregular heart rate
- Hypertension
- Sweating
- Irritability

Thyrotoxicosis

Thyrotoxicosis, also known as a thyroid storm, is caused by hyperthyroidism and can be classified as either acute or chronic. This is an acute condition in the presence of infection or stress, and presents with a worsening of the symptoms of hyperthyroidism. Something as simple as a laceration can cause the patient with hyperthyroidism to experience thyro-toxicosis. Many diseases and conditions can exacerbate this condition, including the following:

- Graves disease (accounts for 85% of all cases of hyperthyroidism)
- Growth abnormalities of the thyroid gland
- Tumors of the testes or ovaries
- Inflammation of the thyroid (as a result of viral infections or other causes)
- Overmedication with supplemental thyroid hormones
- Ingestion of excessive iodine
- Trauma to the thyroid

Myxedema Coma

Myxedema coma is a rare, life-threatening clinical condition in patients with long-standing, severe, untreated hypothyroidism. Most commonly seen in elderly female patients, myxedema coma has an extremely high mortality rate if left untreated. The three key features of myxedema coma are as follows:

- Altered mental status and lethargy (including lengthy sleeping patterns)
- Failure of the thermoregulatory system (hypothermia or the absence of fever despite infectious disease)
- A precipitating event (such as cold exposure, infection, drugs [diuretics, tranquillizers, sedatives, or analgesics], trauma, stroke, heart failure, or gastrointestinal bleeding)

Signs and Symptoms

Myxedema Coma

- Hypothermia
- Auditory and visual hallucinations
- Seizures
- Unresponsiveness

A person who initially presents with myxedema coma is usually hypothermic and may be experiencing both auditory and visual hallucinations, seizures, and unresponsiveness.

Hashimoto's Disease

Hashimoto's disease, also known as chronic lymphocytic thyroiditis, occurs when the immune system attacks the patient's thyroid gland. As a result of inflammation, Hashimoto's disease is the leading cause of hypothyroidism, most commonly affecting women between the ages of 30 and 50 years.

Thyroid Cancer

Thyroid cancer is a cancer that starts in the thyroid gland. The four main types are as follows.

- Papillary and/or mixed papillary/follicular (incidence, approximately 78%)
- Follicular and/or Hurthle cell (incidence, approximately 17%)
- Medullary (incidence, approximately 4%)
- Anaplastic (incidence, approximately 1%)

Most thyroid cancers are treatable with surgical removal of either the affected tissue or the entire thyroid gland.

Assessment of the Patient With Suspected Thyroid-Related Illness

A patient with a thyroid-related condition may present with general weakness, fever, altered mental status, abdominal pain, and lethargy (hypothyroidism), or heart-related complications that include rapid heart rate, congestive heart failure, and atrial fibrillation (hyperthyroidism). In cases of hyperthyroidism, thyrotoxicosis may occur.

Although hypothyroid emergencies are rare, they are medically urgent and require aggressive evaluation and treatment.

Abnormal vital signs in the patient will commonly be more pronounced with the hyperthyroid patient; these include tachycardia and hypertension.

The physical exam may reveal thyroid enlargement (goiter) **Figure 17-9**.

Laboratory Assessment

Laboratory tests that evaluate thyroid function include serum TSH, T3, and T4.

Serum TSH measures the amount of TSH in the blood. The normal range is 0.4 to 4.2 mIU/mL for persons with no symptoms of abnormal thyroid function. However, persons without signs or symptoms of hypothyroidism who have a TSH value greater than 2.0 mIU/mL but normal T4 levels may experience hypothyroidism in the future. This condition is called subclinical hypothyroidism (mildly underactive thyroid) or early-stage hypothyroidism. The condition of anyone with a TSH value greater than 2.0 mIU/mL should be followed very closely by a doctor. Patients being treated for a thyroid disorder should

have a TSH level between 0.5 and 3.0 mIU/mL. Low levels of TSH indicate possible hyperthyroidism or pituitary gland failure, and values above normal indicate thyroid gland failure or a pituitary gland tumor.

TABLE 17-5 Common Laboratory Studies for Thyroid-Related Conditions

Laboratory Study	Normal Range	Causes of Low Levels	Causes of High Levels
TSH	0.4–4.2 mIU/L	Hyperthyroidism, or pituitary gland failure	Thyroid gland failure, or pituitary gland tumor
T3	60–180 ng/dL	Hypothyroidism	Hyperthyroidism
Total T4	5.5–12.5 µg/dL	Hypothyroidism	Hyperthyroidism
Free T4	0.9–2.3 ng/dL	Hypothyroidism	Hyperthyroidism
RT3U	24%–37%	Hypothyroidism	Hyperthyroidism
Triglycerides	< 160 mg/dL	Clinically irrelevant with respect to thyroid disorders	Possible hypothyrodisim
Radioactive iodine uptake	8%–25%	Hypothyroidism	Hyperthyroidism
Glucose tolerance test	Fasting: 70–1,100 mg/dL	Clinically irrelevant with respect to thyroid disorders	Hyperthyroidism
Thyroglobulin antibody	< 2 IU/mL	Clinically irrelevant with respect to thyroid disorders	Hyperthyroidism

Abbreviations: RT3U, triiodothyronine reuptake; T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone.

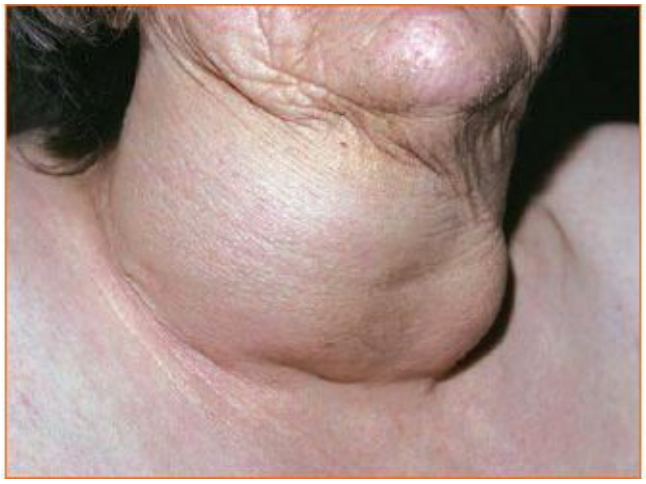

Figure 17-9 Thyroid enlargement.

Table 17-5 indicates common laboratory testing for thyroid-related conditions.

Management

Treatment of thyroid conditions varies depending on the cause of the condition and the severity of symptoms. As with most conditions discussed in this chapter, transport management is mainly supportive.

Hyperthyroidism is usually treated with antithyroid medications, radioactive iodine (which destroys the thyroid and stops the excess production of hormones), or surgery to remove the thyroid. Beta-blockers such as propranolol are indicated to treat the resultant tachycardia, and anxiety may be treated with diazepam, lorazepam, or midazolam until the hyperthyroidism can be controlled. If the thyroid must be removed with radiation or surgery, replacement thyroid hormones must be taken for the rest of the person's life.

TABLE 17-6 Common Medications Used to Treat Thyroid Conditions

Medication

Class	Examples
Antithyroid medications	Methimazole (Tapezole) or propylthioracil (PTU)
Thyroid hormone	Levothyroxine (L-Thyroxin, Levolet, Levo-T, Levothroid, Levoxyl, Novothyrox, Synthroid, Thyro-Tabs, or Unithroid), liothyronine (Cytomel), or liotrix (Euthroid or Thyrolar)
Radioactive iodine	N/A
Beta-blockers	Acebutolol (Sectral), atenolol (Tenormin), betaxolol (Kerlone), bisoprolol (Zebeta), carteolol (Cartrol), carvedilol (Coreg), labetalol (Normodyne or Trandate), metoprolol (Lopressor or Toprol-XL), nadolol (Corgard), nebivolol (Bystolic), penbutolol (Levatol), pindolol (Visken), propranolol (Inderal), sotalol (Betapace), or timolol (Blocadren)

Hypothyroidism is normally treated with hormone replacement therapy. Complications may be related to replacement of thyroid hormones. If too little hormone is given, symptoms of under-active thyroid can occur, including fatigue, increased cholesterol levels, mild weight gain, depression, and slowing of mental and physical activity. If too much hormone is given, the symptoms of hyperthyroidism will recur.

Table 17-6 lists common medications used to treat thyroid conditions.

Lipid Disorders

Lipid disorders cause a change in the production or use of cholesterol. They also may cause a change in the way cholesterol is circulated or processed in the body. As a result, patients with lipid disorders experience very high total cholesterol levels, very low high-density lipoprotein, or "good," cholesterol levels, or high triglyceride levels.

A lipid disorder increases the patient's risk for atherosclerosis and heart disease, but is rarely a true medical emergency.

Pathophysiology

Metabolic Syndrome

Metabolic syndrome is a group of symptoms that together may lead to coronary artery disease, stroke, and type 2 diabetes. Although the syndrome is not widely understood, patients display the risk factors and predisposition for lipid disorders and, consequently, heart disease.

Assessment

Patients with lipid disorder or metabolic syndrome may present with very few abnormal physical findings and vital signs on examination. Laboratory testing is required to definitively diagnose these underlying health factors.

Laboratory Assessment

Tests used to diagnose a lipid disorder may include total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein, or "bad," cholesterol, triglycerides, or very low-density lipoprotein cholesterol.

The desirable value for total cholesterol is less than 200 mg/dL. A borderline high value is 200 to

239 mg/dL. The normal value for high-density lipoprotein cholesterol is greater than 40 mg/dL. The normal value for low-density lipoprotein cholesterol is less than 160 mg/dL. The normal value for triglycerides is less than 160 mg/dL. Finally, the normal range for very low-density lipoprotein cholesterol is 2 to 38 mg/dL.

Management

Lipid disorders are normally managed as a component of the patient's long-term healthy lifestyle and by a physician. The patient's emergency conditions (cardiac/respiratory) are managed based on presentation.

TABLE 17-7 Common Medications Used to Treat Lipid Disorders

Medication Class	Examples
Antihyperlipidemic	Colestipol (Colestid), colesevelam hydrochloride (WelChol), gemfibrozil (Lopid), fluvastatin (Lescol or Lescol XL), or omega-3 polyunsaturated fatty acids (Lovaza)
Cholesterol absorption inhibitors	Ezetimibe (Zetia) or ezetimibe and simvastatin (Vytorin)
Fibrates	Fenofibrate (Tricor, Antara, or Lofibra)
Statins	Lovastatin (Mevacor), simvastatin (Zocor), fluvastatin (Lescol), pravastatin (Pravachol), atorvastatin (Lipitor), or rosuvastatin (Crestor)
Calcium channel blockers	Amlodipine and atorvastatin (Caduet)

Table 17-7 lists common medications used to treat lipid disorders.

Flight Considerations

The management of patients experiencing endocrine-related disorders requires the same management in flight as during ground transport, with no special management considerations when flying at altitude.

Summary

While endocrine disorders are not a common cause for a critical care transport, an understanding of endocrine-related conditions and their pathophysiology will help the CCTP manage these complex patients. As always, it is important to ensure appropriate blood glucose levels. An understanding of adrenal insufficiency in particular can help prevent patient death.

Case Study

YOU ARE JUST FINISHING UP YOUR DINNER after a relatively quiet shift when your dispatch center advises that you are being dispatched to Pondera Medical Center, approximately 95 miles away. The patient has been diagnosed with DKA and requires transport to your base hospital, Northern Rockies Medical Center, for further treatment in the intensive care unit. You and your partner, an EMT, depart for the rural hospital.

Once you arrive, you are met by the attending physician. He advises you that your patient is a 28-year-old, 84-kg man who had been complaining of general malaise and increasing thirst for the past 3 to 4 days. The patient's family called 9-1-1 this morning when they found him minimally responsive. He was subsequently transported to Pondera Medical Center via BLS ambulance with a Glasgow Coma Scale score of 8 (eye score, 2; verbal score, 2; and motor score, 4). Upon arrival, the patient's score had increased to 12 (eye score, 3; verbal score, 3; and motor score, 6) and he was able to answer some questions appropriately but with difficulty. According to the family members, the patient is generally healthy, and no medical problems, takes no medications, and has no allergies. They tell you that it seems like he has been drinking "a lot" of water over the past couple of days and has had to urinate excessively.

Treatment in the emergency department included oxygen via nonrebreathing mask at 15 L/min; a bedside glucose test that revealed high levels of glucose; laboratory studies including a complete blood cell count and comprehensive metabolic panel; a portable chest radiograph that showed the lungs were clear bilaterally; and an electrocardiogram that was unremarkable. The patient has a 14-gauge IV in his left forearm, currently running normal saline to keep the vein open, an 18-gauge saline lock in his right forearm, and an indwelling (Foley) catheter, with approximately 1,300-mL urine output in the last 75 minutes. The patient has received two boluses of normal saline, with each bolus based on 20 mL/kg, and 10 U of short-acting insulin, which decreased the blood glucose level to 580 mg/dL on recheck.

Vital signs included the following: blood pressure, 89/42 mm Hg; heart rate, 132 beats/min; sinus tachycardia with no ectopy; clear respirations, 36 breaths/min with a Kussmaul respiration pattern; and oxygen saturation as measured by pulse oximetry, 96% with oxygen therapy. Laboratory studies revealed the following abnormalities: glucose, 789 mg/dL; blood urea nitrogen, 60 mg/dL; creatinine, 2.0 mg/dL; blood urea nitrogen to creatinine ratio, 30:1, chlorine, 116 mEq/L; potassium, 5.5 mEq/L; and sodium, 151 mEq/L. A urinalysis was positive for ketones and glucose. Arterial blood gas readings showed metabolic alkalosis with a pH of 7.24; $Paco_2$, 19 mm Hg; bicarbonate, 4 mEq/L; and base excess, 1.

As your partner is securing the patient onto the cot for the return transport, you contact your base hospital physician to advise of the patient's status, obtain any additional orders, and give an estimated time of arrival. The receiving intensivist has requested that a continuous insulin infusion be initiated, with the blood glucose level titrated to a target decrease of 50 mg/dL/h.

Once en route back to Northern Rockies Medical Center, you prepare an insulin infusion by adding 100 U of insulin to a 100-mL bag of normal saline and begin the infusion at 8.4 U/h, based upon 0.1 U/kg/h. Because of the patient's altered mental status, you elect to administer 8 mg of ondansetron IV as prophylaxis against vomiting and insert a 16F nasogastric tube in the left nare. After approximately 20 minutes, you recheck the patient's blood glucose level using your glucometer and obtain a reading of 550 mg/dL. On the basis of this reading, you increase the infusion rate to 16.8 U/h and continue the transport. Once you arrive at Northern Rockies Medical Center, the patient is taken directly to the intensive care unit for continuing treatment.

1. How was the diagnosis of DKA determined?
2. Why was the patient given 3,360 mL of normal saline?
3. What are the potential problems that you may experience while en route with this patient?

Analysis

Because of your critical care training, you are able to recognize that this patient is experiencing DKA based on the presence of Kussmaul respirations, an elevated blood glucose reading, and arterial blood gas abnormalities. Symptoms of DKA include an elevated blood glucose level, acidosis, and an altered mental status. Signs include polydipsia, polyphagia, polyuria, and Kussmaul respirations.

This patient was given 3,360 mL of normal saline (20 mL/kg) in an attempt to hydrate the patient because of excessive urination; the lost fluids were demonstrated by tachycardia and hypotension. Short-acting insulin was administered in an attempt to decrease the blood glucose level. It is imperative not to decrease glucose levels too quickly in cases of diabetic ketoacidosis because of the possibility of cerebral edema. In an attempt to gradually decrease the glucose level, a continuous infusion of insulin was initiated, allowing the CCTP to accurately titrate the amount of insulin based on glucose level. Ondansetron was given prophylactically to minimize the risk of aspiration resulting from decreased mental status.

This patient has the potential to experience several problems en route. The ground transport was 95 miles; with the patient's decreased level of consciousness, aspiration must be considered. Insertion of a nasogastric tube minimizes this risk, but the CCTP must still recognize the possibility. With any patient presenting with an altered mental status in the transport environment, CCTPs need to be aware of the possible need to perform definitive airway management, which is often difficult in the confined spaces of an ambulance. Furthermore, with continued administration of insulin, the possibility of inadvertent hypoglycemia may develop. Hypoglycemia can be recognized via vigilant monitoring of the patient's blood glucose level and subsequently corrected by discontinuing the insulin infusion and following local protocols regarding hypoglycemia.

Prep Kit

Ready for Review

- CCTPs should be familiar with the endocrine system and its function.
- The thyroid gland secretes thyroxine to regulate the body's metabolism and calcitonin to maintain normal calcium levels in the blood.
- The adrenal glands consist of the adrenal cortex, which produces corticosteroids, and the adrenal medulla, which produces catecholamines (the hormones epinephrine and norepinephrine).
- During times of stress, adrenocorticotropic hormone causes the adrenal cortex to secrete cortisol, which stimulates body cells to increase their energy production.
- The adrenal cortex secretes aldosterone if blood pressure or volume drops, sodium level decreases, or potassium level increases. The function of aldosterone is to increase the reabsorption of sodium and water and release potassium into the kidneys, increasing circulating blood volume and thus increasing blood pressure.
- During the "fight-or-flight" response, the adrenal medulla secretes small amounts of norepinephrine and large amounts of epinephrine to enable the body to respond to a short-term emergency.
- The pancreas is both an endocrine and an exocrine gland. The exocrine component secretes digestive enzymes; the endocrine component comprises the islets of Langerhans.
- The main hormones secreted by the pancreas are glucagon and insulin, which regulate blood glucose levels. Insulin is the only hormone that decreases blood glucose levels. Insulin is essential for glucose to enter and nourish the cells.
- Diabetes is a metabolic disorder in which the body's ability to metabolize glucose is impaired as a result of insufficient production or inadequate utilization of insulin.
- There are two major types of diabetes mellitus: type 1 and type 2. Type 1 diabetes generally strikes children, whereas type 2 diabetes commonly occurs in adults.

- Most patients with type 1 diabetes do not produce insulin. In patients with type 2 diabetes, the pancreas produces enough insulin, but the body is not able to use it. This is known as insulin resistance. Type 2 diabetes can also be caused by a deficiency in insulin production.
- Hypoglycemia, a drop in blood glucose level, can develop rapidly in patients with diabetes and in alcoholics, those who have ingested a poison or certain drugs, and patients with other diseases such as cancer, liver disease, or kidney disease. If hypoglycemia persists, it can cause permanent brain damage.
- Hyperglycemia, an increase in blood glucose levels above normal range, can have rapid or gradual onset. If left untreated, hyperglycemia will progress to DKA, a life-threatening condition.
- Patients with DKA tend to be young—teenagers and young adults. The processes of DKA usually progress slowly—over a period of 12 to 48 hours—and can lead to dehydration and shock.
- Hyperosmolar hyperglycemic nonketotic syndrome occurs principally in patients with type 2 diabetes. It is characterized by hyperglycemia, hyperosmolarity, and an absence of significant ketones.
- Signs of hypoglycemia may mimic those of intoxication. Patients presenting with signs of intoxication, such as slurred speech and lack of coordination, should be checked for hypoglycemia because hypoglycemia can cause cerebral dysfunction.
- Hyperglycemia and DKA typically present with the three Ps: polyuria, polydipsia, and polyphagia. The two hallmark signs of DKA are Kussmaul respirations and fruity breath odor.
- Signs of HHNS are similar to those of DKA, except that patients with HHNS do not present with Kussmaul respirations or fruity breath odor.
- CCTPs may test for hypoglycemia or hyperglycemia during transport. Knowledge of the glucometer being used is important for understanding the readings.
- Insulin infusions are routinely used in critically ill patients. The CCTP should check the patient's blood glucose levels frequently using a blood glucometer and adjust the insulin infusion rate to maintain the blood glucose targets prescribed for the patient.
- Signs and symptoms of pituitary disorders commonly mirror other disease processes; obtaining a thorough medical history and physical exam results are important to finding the cause of the problem.
- The pituitary gland is divided into two lobes: the anterior pituitary and the posterior pituitary. The anterior pituitary produces and secretes six hormones; the posterior pituitary secretes two hormones, which it does not produce.
- Central diabetes insipidus, caused by damage to the posterior of the pituitary gland, is characterized by polyuria and polydipsia, which lead to dehydration.
- Pituitary lesions are classified into two types: nonfunctioning adenomas and functioning adenomas. Nonfunctioning adenomas do not secrete hormones; with functioning adenomas, overproduction of hormones may occur.
- Acromegaly results from excessive growth hormone secreted by the pituitary gland after the epiphyseal plate has closed. Gigantism results from excessive growth hormone secreted by the pituitary gland before the epiphyseal plate has closed. Both conditions are characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate. Both conditions are usually caused by a benign pituitary tumor.
- Acromegaly and gigantism are managed with medications.
- Abnormalities of the adrenal glands may be hereditary, corticosteroid resistance and hypersensitivity abnormalities, or genetic disorders.

- Pseudo-Cushing's states are a mixed group of disorders related to increased cortisol production. They may be caused by physiological conditions, such as severe illness or intense aerobic exercise, or nonphysiological conditions, such as chronic alcoholism and obesity.
- Endocrine hypertension is a result of hormonal disorders that cause clinically significant hypertension. Endocrine hypertension is often related to excess hormones produced by a tumor.
- Adrenal insufficiency is underproduction of cortisol and aldosterone caused by a decreased function of the adrenal cortex. Adrenal insufficiency may be temporary or permanent, and it affects men and women of all ages equally.
- In patients with chronic AI, signs and symptoms of Addisonian crisis may appear suddenly as a result of an increased period of stress, trauma, surgery, or severe infection. If undiagnosed, AI is potentially fatal.
- Addison's disease is a chronic disorder caused by a deficiency in cortisol and/or aldosterone. It occurs in all age groups. Young patients with Addison's disease are at risk for premature death.
- Cushing's syndrome is caused by prolonged exposure to elevated levels of cortisol in the body. It is relatively rare and affects patients aged 20 to 50 years. Excess cortisol affects many body systems, leading to hyperglycemia, muscle weakness, and bone weakness among other signs and symptoms.
- A pheochromocytoma is a catecholamine-producing benign tumor of chromaffin cells. Of pheochromocytomas, 97% are in the abdomen, 2% are in the thorax, and the remaining 1% are in other body areas. Most pheochromocytomas can be corrected if identified. If untreated, they can be fatal. Treatment is surgery to remove the tumor.
- There are two types of aldosteronism, a syndrome of high blood pressure and low blood potassium levels caused by an excess of aldosterone: primary and secondary.
- Primary aldosteronism, also known as Conn's syndrome, is caused by a tumor; most cases are benign. Secondary aldosteronism occurs as a result of reduced renal blood flow, which stimulates hypersecretion of aldosterone. A low plasma renin activity blood test indicates primary aldosteronism; high plasma renin activity indicates secondary aldosteronism. Treatment is surgery or treatment of the underlying condition.
- Amyloidosis results from abnormal deposits of the protein amyloid in various tissues of the body. Primary amyloidosis is the most common form; secondary amyloidosis occurs as the result of another illness. There is no cure for amyloidosis; treatments focus on managing the symptoms and limiting production of amyloid protein.
- Transport of patients with a life-threatening disorder resulting from a thyroid abnormality is rare. Transport management for patients with thyroid conditions is mainly supportive.
- The thyroid gland is the primary controller of the body's metabolism; it produces the hormones T3 and T4.
- Hypothyroidism is caused by a deficiency of the hormones T3 and T4. Patients with hypothyroidism are at increased risk for acute myocardial infarction and cerebrovascular accident.
- Hyperthyroidism is caused by increased production of the hormones T3 and T4. Thyrotoxicosis is caused by hyperthyroidism, and is classified as acute or chronic.
- Myxedema coma is a rare, life-threatening clinical condition in patients with long-standing severe untreated hypothyroidism. It occurs most often in elderly female patients and is fatal if untreated.
- Hashimoto's disease, the leading cause of hypothyroidism, occurs when the immune system attacks the patient's thyroid gland. It most commonly affects women aged 30 to 50 years.

- There are four main types of thyroid cancer: papillary and/or mixed papillary/follicular, follicular and/or Hurthe cell, medullary, and anaplastic. Most thyroid cancers are treatable with surgery.
- Laboratory tests that evaluate thyroid function are serum thyroid-stimulating hormone, T3, and T4.
- Lipid disorders cause a change in the production or use of cholesterol. They rarely cause a true medical emergency.
- Metabolic syndrome is a group of symptoms that together may lead to coronary artery disease, stroke, and type 2 diabetes. Laboratory testing is necessary to diagnose lipid disorders and metabolic syndrome.
- Lipid disorders are managed by lifestyle adaptations and medications.

Vital Vocabulary

acromegaly A syndrome that results from excessive growth hormone secreted by the pituitary gland after the epiphyseal plate has closed; characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate.

Addisonian crisis The sudden appearance of symptoms, especially shock, in a patient with chronic adrenal insufficiency; may appear suddenly as a result of an increased period of stress, trauma, surgery, or severe infection; other symptoms include weakness, altered mental status, hyperthermia, and severe pain in the lower back, legs, or abdomen.

Addison's disease A chronic hormonal or endocrine disorder caused by a deficiency of cortisol and/or aldosterone and characterized by weakness, fatigue, hypotension, unexplained weight loss, and darkening of the skin.

adrenal insufficiency (AI) Underproduction of cortisol and aldosterone caused by a decreased function of the adrenal cortex; occurs when at least 90% of the adrenal cortex has been damaged.

aldosterone One of the two main hormones responsible for adjustments to the final composition of urine; it increases the rate of active resorption of sodium and chloride ions into the blood and decreases the resorption of potassium.

aldosteronism A syndrome of high blood pressure and low blood potassium levels caused by an excess of aldosterone; there are two main types (primary and secondary).

amyloidosis A group of diseases that result from abnormal deposits of the protein amyloid in various tissues of the body; can occur in a localized area or may be systemic.

central diabetes insipidus A result of damage to the posterior part of the pituitary gland, which causes a lack of the hormone vasopressin, an anti-diuretic hormone, to be produced; because of the lack of anti-diuretic hormone, patients frequently experience polyuria and polydipsia.

Cushing's syndrome A condition caused by an excess of cortisol production by the adrenal glands or by excessive use of cortisol or other similar steroid (glucocorticoid) hormones.

diabetic ketoacidosis (DKA) A form of acidosis in uncontrolled diabetes in which certain acids accumulate when insulin is not available.

endocrine hypertension Significant high blood pressure caused by a hormonal disorder; often related to excess hormone produced by a tumor.

functioning adenoma A type of pituitary lesion in which overproduction of hormones occurs.

gigantism A syndrome that results from excessive growth hormone, secreted by the pituitary gland before

the epiphyseal plate has closed; characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate.

Hashimoto's disease A condition that occurs when the immune system attacks the patient's thyroid gland. The leading cause of hypothyroidism; also known as chronic lymphocytic thyroiditis.

hyperosmolar hyperglycemic nonketotic syndrome (HHNS) A metabolic derangement that occurs principally in patients with type 2 diabetes, and is characterized by hyperglycemia, hyperosmolarity, and absence of significant ketosis.

hyperthyroidism A condition caused by an increased production of T3 (triiodothyronine) and T4 (thyroxine) from the thyroid gland, resulting in an increase in the body's organ function, and characterized by an increase in body temperature, gradual weight loss, increased and/or irregular heart rate, sweating, and irritability.

hypothyroidism A condition caused by a deficiency of T3 (triiodothyronine) and T4 (thyroxine) from the thyroid gland, resulting in a slowing of the body's organ function, and characterized by a decrease in body temperature, gradual weight gain, and increased risk for acute myocardial infarction and cerebrovascular accident.

insulin resistance A condition in which the pancreas produces enough insulin but the body cannot effectively use it.

lipid disorders A group of disorders that cause a change in the production or use of cholesterol and that also may cause a change in the way cholesterol is circulated or processed in the body.

metabolic syndrome A group of symptoms that together may lead to coronary artery disease, stroke, and type 2 diabetes; usually related to predisposition to a lipid disorder.

myxedema coma A rare, life-threatening condition that can occur in patients who have severe, untreated hypothyroidism, and which is characterized by altered mental status and lethargy, failure of the thermoregulatory system, and a precipitating event; may be accompanied by auditory and visual hallucinations, seizures, or unresponsiveness.

nonfunctioning adenoma A type of pituitary lesion in which the tumor does not secrete any hormones.

pheochromocytoma A catecholamine-producing benign tumor of chromaffin cells located in the center of the adrenal gland, which can occur either sporadically or chronically as a result of genetic risk factors; causes stimulation of alpha-adrenergic and beta-adrenergic receptors, resulting in hypertension, increased cardiac contractility, glycogenolysis, gluconeogenesis, intestinal relaxation, and increased heart rate.

polydipsia Excessive thirst, resulting in excessive intake of fluid.

polyphagia Excessive desire to eat, resulting in overconsumption of food.

polyuria Frequent and plentiful urination.

primary aldosteronism A type of aldosteronism usually caused by a tumor of a single adrenal gland that overproduces aldosterone; also known as Conn's syndrome.

primary amyloidosis The most common form of amyloidosis, affecting the heart, kidneys, tongue, nerves, and intestines; classified as apolipoprotein.

pseudo-Cushing's state A condition in which a person has higher cortisol levels from a cause other than actual Cushing's syndrome; these causes include depression, alcoholism, malnutrition, and panic attack.

secondary aldosteronism A type of aldosteronism that occurs as a result of reduced renal blood flow,

which stimulates hypersecretion of aldosterone; can be caused by obstructive renal artery disease, renal vasoconstriction, and edematous disorders.

secondary amyloidosis A form of amyloidosis that occurs as a result of another illness, and which primarily affects the kidneys, spleen, liver, and lymph nodes; classified as amyloid A protein.

thyrotoxicosis An excess of thyroid hormones resulting in a hypermetabolic crisis, including tachycardia over 140 beats/min, hyperthermia (sometimes > 103.9°F), coma with agitation, nausea, vomiting, diarrhea, unexplained jaundice, and pulmonary edema, marked by an elevated thyroxine level; also called thyroid storm.

type 1 diabetes The type of diabetic disease that usually starts in childhood and requires daily injections of supplemental synthetic insulin to control blood glucose; sometimes called juvenile or juvenile-onset diabetes.

type 2 diabetes The type of diabetic disease that usually starts later in life and often can be controlled through diet and oral medications; sometimes called adult-onset diabetes.

Bibliography

Akamizu T. Hashimoto's thyroiditis. Available at: http://www.thyroidmanager.org/Chapter8/8-frame.htm. Accessed July 7, 2009.

Alexander EK, Dluhy RG. Aldosterone excess. Available at: http://endotext.com/adrenal/adrenal23/adrenalframe23.htm. Accessed March 1, 2009.

American Heart Association. Complementary and incremental mortality risk prediction by cortisol and aldosterone in chronic heart failure. *Circulation.* 2007;115:1754–1761.

Berner JA, Papanicolau DA. Carney's complex. Available at: http://www.endotext.org/adrenal/adrenal21/adrenalframe21.htm. Accessed February 23, 2009.

Cohan PM, Wang CM, McArthur DL. Acute secondary adrenal insufficiency after traumatic brain injury: a prospective study. *Crit Care Med.* 2005;33:2358–2366.

Curtis J, Rea C. Lipid disorders. Available at: http://www.revolutionhealth.com/articles/lipid-disorders/stl158164. Accessed July 7, 2009.

Charmandari E, Chrousos GP. Adrenal insufficiency. Available at: http://www.endotext.org/adrenal/adrenal13/adrenalframe13.htm. Accessed February 20, 2009.

Hashimoto's disease: what it is and how it's treated. Available at: http://familydoctor.org/online/famdocen/home/common/hormone/548.html. Accessed February 20, 2009.

Hyperthyroidism: what it is and how to treat it. Available at: http://familydoctor.org/online/famdocen/home/common/hormone/869.html. Accessed February 20, 2009.

Koch CA, Wofford MR, Ayala AR, Pacak K. Overview of endocrine hypertension. Available at: http://www.endotext.org/adrenal/adrenal26/adrenalframe26.htm. Accessed February 23, 2009.

Kumar RM, Gandhi SK, Little WC. Acute heart failure with preserved systolic function. *Crit Care Med.* 2008:S52–S56. Merry WH, Caplan RH, Wickus GG, et al. Postoperative acute adrenal failure caused by transient corticotropin deficiency. *Surgery.* 1994;116:1095–1100.

Norman J. Thyroid cancer. Available at: http://www.endocrineweb.com/thyroidca.html. Accessed

February 20, 2009.

Norman J. Your thyroid gland. Available at: http://www.endocrineweb.com/thyroid.html. Accessed February 20, 2009.

Pacak K, Adams KT, Eisenhofer G. Pheochromocytoma. Available at: http://endotext.com/adrenal/adrenal34/adrenalframe34.htm. Accessed July 7, 2009.

Pizarro CF, Troster EJ, Damiani DM, Carcillo JA. Absolute and relative adrenal insufficiency in children with septic shock. *Crit Care Med.* 2005;33:855–859.

Vottero A. Glucocorticoid resistance syndromes and states. Available at: http://www.endotext.org/adrenal/adrenal16/adrenalframe16.htm. Accessed February 22, 2009.

What is hypothyroidism? Available at: http://familydoctor.org/online/famdocen/home/common/hormone/548.html. Accessed February 20, 2009.

Wiersinga W. Adult hypothyroidism. Available at: http://www.thyroidmanager.org/Chapter9/ch_9-4.htm. Accessed February 20, 2009.


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Environmental Emergencies

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Objectives

1. Discuss risk factors for environmental emergencies (p 718).
2. Describe the process of thermoregulation, including the concepts of thermogenesis and thermolysis (p 718).
3. Explain the process of heat transfer, including radiation, conduction, convection, evaporation, and absorption (p 719).
4. Discuss signs, symptoms, and transport management of heat cramps (p 721).
5. Discuss signs, symptoms, and transport management of heat syncope (p 722).
6. Discuss signs, symptoms, and transport management of heat exhaustion (p 722–723).
7. Discuss signs, symptoms, and transport management of heat stroke (p 723–724).
8. Discuss signs, symptoms, and transport management of frostbite (p 725–726).
9. Discuss signs, symptoms, and transport management of hypothermia (p 726–727).
10. Discuss signs, symptoms, and transport management of drowning and submersion (p 727–728).
11. Discuss signs, symptoms, and transport management of diving injuries (p 728).
12. Explain the purpose of hyperbaric therapy and when it might be used (p 729–730).
13. Discuss signs, symptoms, and transport management of altitude illness (p 730–731).
14. List flight considerations relating to environmental emergencies (p 731).

Introduction

Environmental emergencies are medical conditions caused or worsened by the weather, terrain, or unique atmospheric conditions. People may experience severe illness from environmental conditions in a vast array of locations and situations. Heat, cold, submersion, and decompression illnesses can occur in remote wilderness locations, major urban metropolitan areas, and any place in between. People in harsh environmental conditions frequently survive environmental extremes far better than at-risk people (such as elderly, homeless, and chemically dependent people). The list of potential scenarios creating an environmental emergency is seemingly endless, regardless of geographic region.

A CCTP is often involved with patients with environmental emergencies during the critical care transport between facilities. Many preexisting medical and lifestyle factors predispose patients to critical illness created or exacerbated by environmental stressors. The challenge to CCTPs is to recognize the patients who are more susceptible to environmental exposure in unexpected conditions. To provide patients with the optimal chance for recovery, it is essential that CCTPs continue or initiate many critical interventions during patient transport.

Risk Factors

Risk factors that predispose people to environmental emergencies include extremes of age (young and

old), taking certain medications, and being in a poor state of health. Specifically, environmental factors can be worsened in people with diabetes, cardiovascular disease, restrictive lung disease, thyroid disease, and even psychiatric illnesses.

Thermoregulation

The body's ability to acclimatize, to adjust to a changing environment, is part of the homeostatic process. **Thermoregulation** refers to the body's natural ability to ensure and maintain a balance between heat production (**thermogenesis**) and heat elimination (**thermolysis**). The **hypothalamus** is located in the most inferior portion of the diencephalon, making up the portion of the brain between the cerebrum and the brain stem. Through the **hypothalamo-hypophyseal portal system**, the hypothalamus sends a releasing or inhibiting factor to the pituitary gland. The interrelationship between the glands is referred to as the **hypothalamic-pituitary axis**. The result is an increase or decrease in metabolism throughout the body. The hypothalamus serves as the "master thermostat" by using negative feedback control. A rise in the core body temperature elicits signals to shut off thermogenesis pathways and increase heat loss mechanisms; a fall in the core body temperature prompts heat production and limits thermolysis mechanisms. In the preoptic area of the hypothalamus, heat-sensitive neurons increase firing when the body senses an increase in temperature. In the posterior hypothalamus, signals from the preoptic area are combined with signals from the remainder of the body to cause a compensating heat loss to restore the temperature back to a tolerable range. This negative feedback system of the hypothalamus cannot work properly without a network of complementary detectors that are sensitive to temperature changes. Cold sensors are located predominantly in the skin, spinal cord, and other areas that are sensitive to the cold end of the temperature spectrum. They respond to excess cooling by transmitting neural action potentials to the hypothalamus; these halt the heat-reducing signals. The goal is to maintain a constant core body temperature within a degree of 98.6°F (37°C).

The negative feedback system has three major components:

- Temperature receptors
- Effector organ systems
- Integrator or controller

The majority of temperature receptors are found in the papillary layer of the dermis throughout widespread areas of the body. These exteroceptors respond to external stimuli affecting the skin directly. Areas of nerves, or spots, respond to hot or cold stimuli, with cold spots being more numerous. Receptors are nerve endings that act as transducers, converting one form of energy into nerve impulses, sent along afferent nerves, including the lateral spinothalamic tract, to the diencephalon. The expression of this neural processing is to stimulate motor nerves and peripheral pathways to use effectors. The result is for muscles to contract or relax, blood vessels to constrict or dilate, and glands to secrete hormones. These impulses are coordinated as necessary by the specific integrator or controller against information already stored (such as the "normal" body temperature of 98.6°F [37°C]). This hypothalamic thermostat reduces excesses in body heat. Three major mechanisms have a part:

- Blood vessels in the skin dilate, resulting in eight times as much heat transfer to the skin's surface.
- Perspiration is increased at a ratio of 10 times the normal perspiration for each degree of temperature increase.
- Increases in muscle activity, including shivering, are inhibited.

In response to cold, the three main regulatory processes to increase body heat include the following:

- Stimulation of the posterior hypothalamus to cause a constriction of blood vessels

- Hypothalamic stimulation of the primary motor center in the posterior hypothalamus that stimulates the brain stem, spinal cord, and motor neurons to ultimately cause muscle activity, including shivering
- The hypothalamic stimulation of piloerector muscles causing minimal production of heat and the visible sign of "goose flesh" and the body hair to "stand on end"

Basal Metabolic Rate

At rest, the body's chief source of heat production is the metabolism of nutrients. The heat produced at rest from normal metabolic reactions, including breathing, circulation, and digestion, is referred to as the **basal metabolic rate (BMR)**. It is a calculation of the number of calories metabolized per square inch of body surface area per hour and can be thought of as the minimal requirement to sit still all day long. Calculation of the BMR requires fairly sophisticated laboratory equipment. A BMR provides useful information in specialized clinical settings, such as studies in endocrinology and metabolism. The average BMR of a 70-kg adult is around 60 to 70 kcal/h. A brisk walk can produce 300 kcal/h. The BMR can be greatly affected by age, sex, stress, hormones, and body surface area. As the ratio of body surface area to body volume increases, heat loss increases, so that the shorter of two persons of the same weight will lose heat at a faster rate; this is most relevant to pediatric patients, who have a very high surface area to body volume ratio and are most susceptible to heat loss.

Heat Transfer

Heat produced by metabolism and glycogen breakdown is used to warm the body and maintain the core body temperature. Excess heat must be eliminated. Usually this elimination is accomplished through the temperature gradient between the body and the immediate surrounding environment **Figure 18-1**. The body transfers heat to the surrounding environment through four primary mechanisms:

- **Radiation**—the transfer of heat through electromagnetic waves
- **Conduction**—the transfer of heat through direct contact with a cooler object
- **Convection**—the loss of heat carried away from the body by currents of air or water
- **Evaporation**—the loss of heat carried away as liquid converts to gas

If the surrounding environment is hotter than the body, the body gains heat through **absorption**. Convection and radiation cease as ambient temperatures reach or exceed skin temperatures. Furthermore, the humidity of the immediate environment can affect thermoregulation as well. A healthy adult can sweat up to about 1 L/h (although not for very long), but if the humidity in the surrounding air is more than 75% to 80%, the evaporative process is impaired.

When dealing with a cold environment, the body shifts to thermogenesis. This process is mainly controlled by increases in BMR and physical exertion. The sympathetic nervous system increases muscle tone and initiates shivering to produce more kilocalories per hour for the immediate needs of the body. The thyroid is stimulated to meet longer heat-production needs. The hypothalamus stimulates peripheral vasoconstriction, sweating stops, and blood is shunted toward the core.

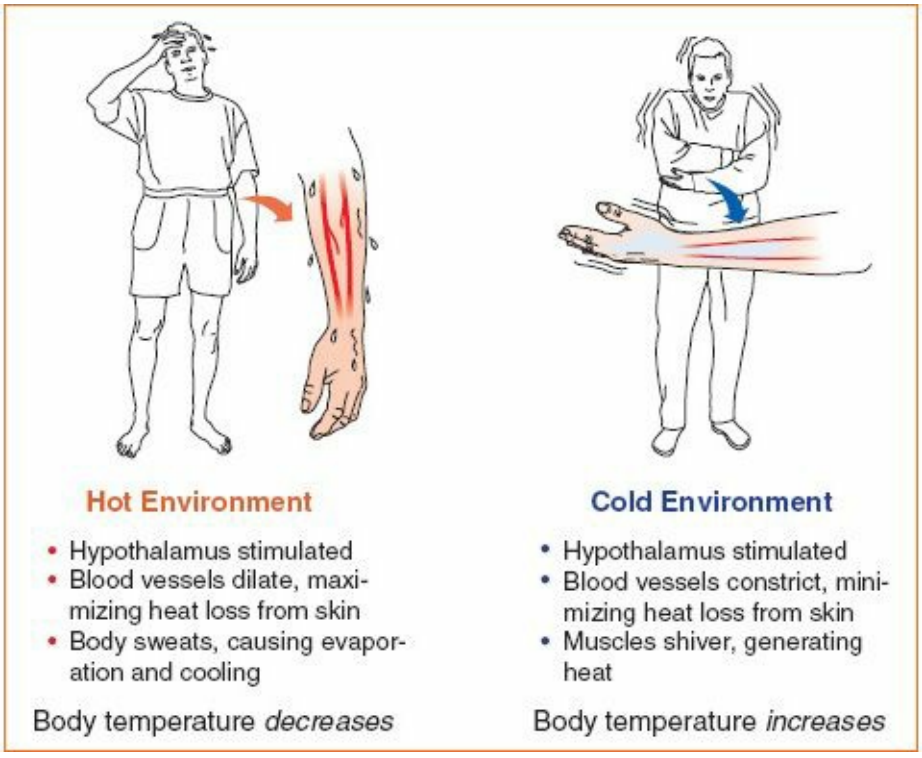

Figure 18-1 The hypothalamus notes a rise or fall in core body temperature and elicits responses to regulate it.

In very cold temperatures, a fair amount of heat is lost through the process of respiration. When cold air is inhaled, it is warmed by the body and then exhaled, causing heat to be lost.

Expecting the Unexpected

A patient need not be found in the desert or plucked from a snow bank to have a "temperature emergency." For example, patients with a spinal cord injury may have a disruption in the negative feedback loop and, therefore, a disruption of thermoregulation. CCTPs must be cognizant of these patients' inability to self-regulate body temperature and take the necessary external steps to maintain warmth or prevent overheating.

Heat Emergencies

Heat illness occurs when there is an increase in core body temperature as a result of inadequate thermolysis. A patient's general state of health, age, certain medications **Table 18-1**, amount of clothing, mobility, and surroundings all have a role in the effects of heat on the body, **Table 18-2**. According to the US Centers for Disease Control and Prevention, more people die of heat-related illness than in all natural disasters (such as floods, hurricanes, tornadoes, lightning, and earthquakes) combined. The United States averages more than 300 heat-related deaths each year. CCTPs in all geographic regions should be prepared to transport patients experiencing heat-related illnesses.

TABLE 18-1 Substances Contributing to Heat Illness

Alcohol
Alpha-agonists
Amphetamines

Anticholinergic medications (such as atropine sulfate, scopolamine, benztropine mesylate, belladonna, and synthetic alkaloids)
Antihistamines
Antiparkinsonian agents
Antipsychotics (such as haloperidol)
Beta-blockers
Calcium channel blockers
Cocaine
Diuretics (such as furosemide, hydrochlorothiazide, and bumetanide)
Heroin
Laxatives
Lithium
Lysergic acid diethylamide
Monoamine oxidase inhibitors
Phencyclidine hydrochloride
Phenothiazines (such as prochlorperazine, chlorpromazine, and promethazine)
Sympathomimetic medicines (such as amphetamines, epinephrine, ephedrine, cocaine, and norepinephrine)
Thyroid agonists (such as levothyroxine)
Tricyclic antidepressants (such as amitriptyline, imipramine, nortriptyline, and protriptyline)

Medications can cause or contribute to heat illness through a variety of possible mechanisms. Medications that depress the central nervous system, such as alcohol and barbiturates, impair a person's ability to properly dissipate heat generated within the body. Many central nervous system depressant medications can cause heat illness or hypothermia.

Stimulant medications promote increased muscle activity and metabolism, causing increased thermogenesis. Cocaine, amphetamines, and other sympathomimetic medications lead to hyperthermia through this mechanism. The increase in heat production cannot be compensated for by increased heat dissipation, leading to hyperthermia.

Any medication that interferes with cardiovascular performance will undermine the body's ability to dissipate heat. Beta-blockers, calcium channel blockers, and diuretics each interfere with heat loss, causing or worsening hyperthermia. Diuretics further complicate heat illness by limiting the fluid volume available for evaporative cooling (sweating).

Medications that impair sweating, such as antihistamines, tricyclic antidepressants, and other anticholinergics, compromise this essential thermoregulatory mechanism, another factor leading to heat illness. Other specific medication-related causes of hyperthermia or heat illness include neuroleptic malignant syndrome and malignant hyperthermia, discussed further in Chapter 20.

TABLE 18-2 Factors That Predispose to Heat Illness

Factors That Increase Internal Heat Production	Factors That Interfere With Heat Dissipation
Physical exertion	High ambient temperature
Response to infection (fever)	High humidity
Hyperthyroidism	Obesity (insulation effect, and less efficient dissipation)
Agitated and tremulous states (such as Parkinson disease, psychosis, mania, drug withdrawal—opiate and alcohol)	Impaired vasodilatation
Drug overdoses (such as sympathomimetics, cocaine, caffeine, lysergic acid diethylamide, phencyclidine hydrochloride, methamphetamine, and Ecstasy)	Diabetes
	Alcoholism
	Drugs: diuretics, tranquilizers, beta-blockers, antihistamines, and phenothiazines
	Impaired ability to sweat (as in cystic fibrosis, skin diseases, and healed burns)
	Heavy or tight clothing
Factors That Increase Heat Absorption	**Factors That Impair the Body's Response to Heat Stress**
Confined, unventilated, hot living quarters	Dehydration
Working in hot conditions (such as in bakeries, steel mills, and construction sites)	Prior heat stroke
Being in parked automobiles in the summer	Hypokalemia
	Cardiovascular disease
	Previous stroke or other central nervous system lesion

If thermoregulation mechanisms fail or become taxed beyond limits, the core body temperature will rise dramatically and quickly. The core body temperature can rise to 106°F (41.1°C) or more in less than 15 minutes. Older persons are particularly at risk. As a result of underlying illness and other metabolic changes they do not acclimatize well. They perspire less, feel thirst more slowly, have difficulty swallowing, and have decreased mobility (making it more difficult just to reach a glass of water). They are also more apt to be taking medications that can alter or impede thermoregulation.

Among the young and healthy, children exposed to hot environments are most vulnerable to heat stressors. Compared with adults, children have higher rates of metabolism and do not dissipate heat as well. Infants and young children are completely dependent on others to provide the proper thermal environment. Excessive blankets or bedding, prolonged confinement in hot vehicles, and other lapses in

environmental control place infants and young children at serious risk for heat-related injury.

Heat illness and heat stroke are the third leading cause of death of high school athletes in the United States; adolescent athletes are at high risk for heat-related illness. Older children may elect to continue strenuous activities in hot or humid environments without understanding the risks or symptoms of heat-related illness or the importance of adequate hydration.

Heat Cramps

Heat cramps are involuntary muscle pains, usually in the abdomen, lower extremities, or both, as a result of profuse sweating and loss of sodium. These events usually afflict healthy persons who are overexerting their bodies in a hot environment. Generally, people recognize the problem and instinctively move to a cooler place and replenish lost fluids. However, if the person has lost a lot of sweat and drinks only water, the sodium loss has not been resolved, and cramps may ensue. In addition, a person may attempt to quench thirst by drinking excessive amounts of water in a very short time, leading to **water intoxication**. Heat cramps are a result of a water-sodium imbalance and are often not accompanied by hyperthermia. If a patient also has some degree of heat exhaustion, heat stroke, or significant hyperthermia, additional cooling measures may be required.

Treatment

The treatment for heat cramps is largely supportive. The patient should be removed from the hot environment, lie supine if he or she feels faint, and be instructed to drink a salt-containing liquid or electrolyte drink (if not at risk for aspiration) **Figure 18-2**. As the patient's salt balance is restored, the symptoms will abate. If symptoms continue, the underlying cause should be sought, and the patient should be rapidly transported to the emergency department. It is unlikely that patients will require critical care transport for heat cramps unless there is a significant degree of hyponatremia or the heat cramps are accompanied by some form of hyperthermia. Significant heat cramps, refractory to oral electrolyte replacement, may be managed with the administration of 1 to 2 L of IV normal saline. CCTPs should use caution when administering IV fluids to correct presumed hyponatremia without adequate patient evaluation or laboratory studies.

Severe hyponatremia requires very specific management depending on the cause. It is essential that CCTPs avoid rapid correction of an abnormal sodium level in any patient with chronic hyponatremia. Devastating neurologic injury associated with central pontine myelinolysis occurs when patients with chronic hyponatremia have their sodium level rapidly corrected or inadvertently overcorrected.

Water Intoxication

The normal process of homeostasis provides for the body to balance intake against output. In general, an average adult takes in about 1,000 mL of fluid throughout an average day. Water intoxication or poisoning can result when the normal balance of electrolytes in the body is pushed outside of safe limits by overconsumption of free water. Normal, healthy (physically and nutritionally) people need not be overly concerned about accidentally consuming too much water. Nearly all deaths related to water intoxication from healthy people have resulted from water drinking contests or in people attempting to correct dehydration when they consume more than 5 to 10 L of water in a short period.

Figure 18-2 Give the patient with heat cramps one or two glasses of a salt-containing solution if he or she is not nauseated and there is no risk of aspiration.

Human kidneys can excrete up to 15 to 20 L of free water each day or up to 1 L per hour. When a euvolemic patient (one with normal blood volume) consumes or receives more free water (without sodium) than the kidneys are able to excrete, water intoxication and hyponatremia begin. In addition to the populations previously described, individuals consuming only water during prolonged endurance activities, such as marathons and hiking, are also at high risk for water intoxication.

Signs and Symptoms

Heat Cramps

- Involuntary muscle pain, usually in the abdomen or lower extremities
- Profuse sweating

Differential Diagnosis

Heat Cramps

- Heat exhaustion
- Heat stroke
- Musculoskeletal trauma (such as sprains or strains)
- Electrolyte abnormality
- Autoimmune disease
- Medication side effect or medication withdrawal symptoms

Transport Management

Heat Cramps

- Remove the patient from the hot environment.
- Place the patient in a supine position.

- If there is no risk of aspiration, give the patient a drink containing salt.
- Consider administering 1 to 2 L of IV normal saline; use caution if there is any possibility of chronic hyponatremia.

Heat Syncope

Heat syncope is an episode of collapse or near collapse that typically occurs in a nonacclimatized person. It is often seen at outdoor events where crowds are standing for long periods. Heat syncope is also common in outdoor settings when a person stands suddenly after sitting or lying on the ground for an extended period. It is thought that peripheral vasodilation, possibly exacerbated by some degree of dehydration, is the likely cause. Treatment is aimed at placing the patient in the recovery position and allowing fluid intake. If the patient does not recover quickly, heat exhaustion or heat stroke should be suspected.

Signs and Symptoms

Heat Syncope

- Postural hypotension from vasodilation
- Volume depletion
- Syncopal episode
- Subjective or documented hyperthermia

Differential Diagnosis

Heat Syncope

- Heat exhaustion
- Heat stroke
- Vasovagal syncope
- Cardiovascular dysfunction
- Psychogenic syncope
- Seizure
- Dehydration
- Pseudosyncope

Transport Management

Heat Syncope

- Place the patient in the recovery position.
- Provide fluids.
- Remove the patient from the hot environment.
- Manage the ABCs as clinically indicated.

Heat Exhaustion

Heat exhaustion is a clinical syndrome representing a moderate form of heat illness on the continuum to heat stroke. Two classic forms of the syndrome are recognized: water-depleted and sodium-depleted. Water-depleted exhaustion primarily occurs in elderly people. Age-related immobility, decreased thirst sensitivity, and medications that contribute to dehydration are suspected culprits.

This condition may also occur in active younger workers and in athletes who do not adequately replenish fluids during activities in a hot environment. Sodium-depleted exhaustion results from excessive losses of sodium from sweating and may take hours to days to develop. When a person loses sweat but replenishes only with water, the lost sodium is not replaced. A closely related condition is **exertional hyponatremia**, which shares many characteristics with (nonexertional) water intoxication. This condition occurs with prolonged exertional activity, usually, but not always, in a warm or hot environment coupled with excessive hypotonic fluid intake and begins with nausea, vomiting, mental status changes, and can eventually lead to cerebral edema and seizure.

Symptoms of heat exhaustion include headache, fatigue, dizziness, nausea, vomiting, and abdominal cramps. Transient syncope may also be present in heat exhaustion, but any significant alteration in neurologic status would be considered heat stroke. Profuse sweating and pale, clammy skin are generally seen. Heart rate and respirations are typically elevated. Tachypnea may lead to signs of hyperventilation syndrome: carpopedal spasm, perioral numbness, and a lowered end-tidal carbon dioxide ($ETCO_2$) level. In heat exhaustion, the patient's core temperature typically ranges from 100.4°F (38°C) to 104°F (40°C), although it is also possible for core temperatures to remain normal. The blood pressure may be low owing to peripheral pooling or volume depletion. Orthostatic hypotension will generally be present. The patient may report having darker, brown urine, which may suggest rhabdomyolysis. CCTPs may find it helpful to consider heat exhaustion as a fluid and electrolyte disorder caused by a hot environment or strenuous activity rather than a disruption of a person's thermoregulatory ability. If left untreated, heat exhaustion may completely compromise a patient's thermoregulatory ability and may progress into heat stroke.

Signs and Symptoms

Heat Exhaustion

- Headache
- Fatigue
- Dizziness
- Nausea
- Vomiting
- Abdominal cramps
- Profuse sweating
- Pale, clammy skin
- Elevated heart rate and respirations
- Carpopedal spasm
- Perioral numbness
- Lowered $ETCO_2$ level
- Normal or elevated skin temperature
- Low blood pressure

- Darker, brown urine

Differential Diagnosis

Heat Exhaustion

- Summer flu
- Heat stroke

Transport Management

Heat Exhaustion

- Move the patient to a cooler environment.
- Remove excess clothing.
- Place the patient in a supine position with the legs elevated.
- Rehydrate the patient with sports drinks if nausea is not present.
- Obtain blood samples for electrolyte analysis, and initiate IV administration of normal saline.
- Consider IV fluids with approval from medical control. Cooling a patient may redistribute fluids within the body, improving intravascular volume.
- Administer fluid based on blood pressure and heart rate.
- Monitor the electrocardiogram (ECG), vital signs, temperature, and $ETCO_2$.
- Consider external cooling measures, such as evaporative cooling.
- Exercise caution when using ice packs or ice water immersion.
- Care for patients receiving specialized cooling techniques, such as cooling blankets; cold water peritoneal, rectal, thoracic, or gastric lavage; cold IV fluids; or cold humidified oxygen, as directed.
- Avoid giving antipyretic medications, which may cause severe complications in patients with heat exhaustion.
- Consider benzodiazepine medications for patients with withdrawal symptoms, excessive shivering, or excessive muscle activity.

Treatment

Treatment of heat exhaustion is aimed at repairing the derangement. Move the patient to a cooler environment, remove excess clothing, and place the patient supine with the legs elevated. Rehydration should be undertaken. Oral rehydration with sport drinks may be appropriate if nausea is not present.

Obtain blood samples for electrolyte analysis and consider IV fluids with approval from medical control for patients who appear volume-depleted. As cooling occurs, fluids distributed throughout the body may return to the intravascular space, improving intravascular fluid volume. Administer fluid based on blood pressure, heart rate, and urine output.

Closely monitor the ECG for signs of electrolyte-induced arrhythmia. Rhabdomyolysis may release excess potassium from the destruction of muscle tissue. Hyperkalemia may result in tall, peaked T waves on the ECG. Exertional hyponatremia may lead to a relative increase in calcium. Heat illness can cause mutliple electrolyte disturbances, including hyponatremia and hypocalcemia, which may result in

electrocardiographic changes. Finally, monitor vital signs, temperature, and $ETCO_2$.

External cooling measures may be taken in patients with an elevated body temperature, but the patient should not be chilled excessively, which can result in shivering and thermogenesis. External cooling is easily accomplished by sponging or spraying the patient with water and fanning gently to promote evaporation. Evaporation will not likely take place if the relative humidity is high (> 80%). Rubbing alcohol should not be used to sponge a patient. Ice packs may cause localized vasoconstriction, restricting heat transfer and removal.

CCTPs may be requested to transport patients who are receiving additional aggressive cooling techniques. Cold water may be lavaged into peritoneal, rectal, gastric, and thoracic cavities of patients with severe heat exhaustion or heat stroke. In addition, many health care facilities have commercial cooling blankets to assist with the management of hyperthermic patients. Cold IV fluids and cold humidified oxygen are also potentially helpful for patients with severe heat exhaustion or heat stroke.

Heat Stroke

Heat stroke is the least common but most deadly of the heat illnesses. It is defined as a severe disturbance in the body's thermoregulation, resulting in altered mental status and a core body temperature of more than 104°F (40°C). It is a profound emergency, with mortality as high as 80% in untreated patients. Death occurs in as many as 10% of treated patients. The "critical thermal maximum" is reached as the core body temperature exceeds 109.4°F (43°C). When this occurs, cellular respiration becomes impaired, cell membranes have increased permeability, protein denaturing begins, and tissue necrosis results.

Two distinct syndromes have been identified: classic and exertional **Table 18-3**. Classic (or passive) heat stroke usually occurs during heat waves. It is most likely to be seen in persons who are very old, very young, or bedridden. Patients with chronic illnesses (such as diabetes and heart disease), people taking certain medications (such as anticholinergics, diuretics, and beta-blockers), and people with alcoholism are more susceptible. As high environmental temperatures elicit thermolysis, the previously mentioned conditions impair heat loss and the core body temperature continues to rise unchecked. In exertional heat stroke, a younger healthy person is exposed to high heat and humidity during a period of strenuous activity. As the ambient temperature reaches that of the body, radiation and convection are interrupted and thermolysis is no longer effective. Evaporative cooling through sweating may be further impaired when the relative humidity is more than 75%. If the person continues the activity in these conditions, heat will continue to generate without a mechanism for removal, resulting in an increase in the core body temperature to more than tolerable levels. In addition to the heat emergency, patients may also have depleted glucose and electrolytes. Their conditions may have progressed to rhabdomyolysis or acute renal failure.

TABLE 18-3 Classic vs Exertional Heat Stroke

Characteristic	Classic Heat Stroke	Exertional Heat Stroke
Age	Older	Younger
General health	Chronic diseases or schizophrenia	Healthy person
Medications	Beta-blockers, diuretics, or anticholinergics	Often none; consider stimulant abuse
Activity	Very little to bedridden	Strenuous
Sweating	Absent	Present

Skin	Hot, red, and dry	Moist and pale
Blood glucose level	Normal	Hypoglycemic
Rhabdomyolysis	Rare	Common
Acute renal failure	Rare	Common

Both types of heat stroke present with similar signs and symptoms. The earliest signs of the altered mental status are irritability, bizarre behavior, combativeness, and hallucinations. These symptoms often mislead bystanders who commonly think the problem is related to substance abuse or behavior rather than the environment. Other presenting signs may lead responders into suspecting stroke or head injury. The diagnostic sign of heat stroke is a markedly elevated core body temperature. Tachycardia, tachypnea with a n $ETCO_2$ less than 20 mm Hg, and dry, red skin are common. The blood pressure may be normal or decreased depending on the degree of dehydration. Other central nervous system disturbances may be seen as tremor, seizure, constricted pupils, and decerebrate or decorticate posturing. Heat stroke is an easy diagnosis to miss. CCTPs must keep the possibility of heat stroke constantly in mind during heat waves, when dealing with the presentation of coma of unknown origin and with cases of the summer flu.

Signs and Symptoms

Heat Stroke

- Altered mental status
- Confusion
- Irritability
- Bizarre behavior
- Combativeness
- Hallucinations
- Elevated core body temperature
- Tachycardia
- Tachypnea with $ETCO_2$ less than 20 mm Hg
- Dry, red skin
- Normal or decreased blood pressure
- Tremors
- Seizure
- Constricted pupils
- Decerebrate or decorticate posturing

Differential Diagnosis

Heat Stroke

- Intracranial hemorrhage
- Ischemic stroke
- Diabetic ketoacidosis

- Malignant hyperthermia
- Neuroleptic malignant syndrome
- Sympathomimetic toxicity
- Salicylate toxicity
- Phencyclidine toxicity
- Encephalopathy
- Withdrawal syndrome

Transport Management

Heat Stroke

- Evaluate the ABCs.
- Administer supplemental oxygen.
- Consider endotracheal intubation.
- Remove the patient's clothing.
- Move the patient to a cooler environment.
- Sponge or spray water over the patient.
- Fan the patient.
- Place ice packs at the nape of the neck, armpits, and groin.
- Initiate an IV line of normal saline, and obtain blood samples for glucose and electrolyte analysis.
- Contact medical control, and consult a physician regarding the use of lactated Ringer's solution.
- Watch closely for developing signs of pulmonary edema.
- Closely monitor the ECG, vital signs, and $ETCO_2$.
- Be prepared to treat a seizure with common antiepileptic drugs (such as diazepam).

Expecting the Unexpected

A heat emergency can occur in any season of the year, especially in elderly people who have difficulty acclimatizing and often have inadequate hydration and take multiple medications, which affect the negative feedback loops in thermoregulation. CCTPs should give particular attention to signs of poor skin turgor and lack of sweating during transport of elderly people with an elevated temperature.

Fever and other conditions may mimic heat stroke and, therefore, increase the challenge for clinicians. The patient's history may provide important clues. For example, a recent complaint of cough or dyspnea, an obvious skin rash, a change in urine color, or intermittent shaking "chills" are more likely to be associated with infection rather than an environmental cause. Pyrogens (proteins excreted in the body's fight of an infection) act on the hypothalamus to increase the body temperature. Fever in children may result in seizure. Fever is most often treated with antipyretics. These measures can prove dangerous in treating heat-related illness.

Anticholinergic and organophosphate exposure and poisoning can also cause hot, red, dry skin; mental status changes; and tachycardia. These agents usually cause dilated pupils, whereas a heat emergency generally results in pupil constriction. In addition, **neuroleptic malignant syndrome**, caused by some antiemetic and antipsychotic medications, and **malignant hyperthermia** may occur with common anesthesia agents (most notably succinylcholine) and may present with hyperthermia, a hyperdynamic state, and altered mentation.

Treatment of Heat Stroke

Treatment of heat stroke is focused on regaining control of the core body temperature. CCTPs should evaluate the ABCs, administer oxygen, and consider controlling the airway. Immediate attempts to decrease the core body temperature begin with removing the patient's clothing and moving him or her to a cooled environment.

Two main methods for rapid cooling are evaporative cooling and ice water immersion. For obvious reasons, ice water immersion, although shown by research to be the superior method, has limited usefulness in the transport environment. Evaporative cooling can be easily started and continued during transport by sponging or spraying water over the patient and fanning to promote the evaporation. Using rubbing alcohol and covering the patient with water-soaked sheets need to be avoided because they can promote thermogenesis. Placement of commercial ice packs at the nape of the neck, armpits, and groin further promotes cooling. In high humidity, evaporation will be restricted. Cooling should be continued until the rectal temperature falls below 102°F (38.8°C). Cooling promotes peripheral vasoconstriction and may raise the blood pressure.

Treatment also includes the following:

- Initiate an IV line of normal saline, and obtain blood samples for glucose and electrolyte analysis.
- Contact medical control, and consult a physician about the use of lactated Ringer's solution.
- Watch closely for developing signs of pulmonary edema.
- Closely monitor ECG, vital signs, and $ETCO_2$, and be prepared to treat a seizure with a benzodiazepine; keep a close eye on the ECG for indications of hyperkalemia and arrhythmia.

The patient should be monitored for changes in central venous pressure, pulmonary wedge pressure, systemic vascular resistance, and cardiac index. If rhabdomyolysis is present, treatment includes aggressive hydration and administration of normal saline. Closely monitor the patient's fluid status and contact medical control for further medications (such as mannitol and sodium bicarbonate) if the patient's condition continues to deteriorate. Additional efforts, such as dialysis, may be needed for elevated potassium or uremia.

Cold Emergencies

Most cold-related injuries are localized to exposed body parts, especially the face, where there are fewer cold receptors in the skin compared with areas of the body covered by clothing. The tip of the nose, ear lobes, fingers, and toes, where peripheral circulation is weakest and body temperature is the lowest, are most susceptible.

Frostbite

Frostbite is classified as **superficial frostbite** (frostnip) and **deep frostbite** depending on the extent of tissue involvement. Frostbite is a mild form of cold injury and comes on slowly and generally without a lot of pain. People with frostbite tend to be unaware of the occurrence. This condition is easily treated by placement of a warm hand over the chilled ear, nose, or fingers. The return of warmth to the affected part is noted by some degree of redness and tingling. Capillary leakage may result in localized edema. The surrounding tissue may appear white and waxy **Figure 18-3**. A burning or stinging sensation may result during warming and may persist for weeks following the event.

Deeper frostbite involves the freezing of tissues exposed to temperatures below the freezing point (32°F [0°C]). Subjected to the low, freezing temperature, the fluid within cells becomes ice crystals, which damage the cells. This condition is further complicated by the "sludge" effect of cooled blood,

resulting in increased viscosity, poor flow, capillary leakage, poor perfusion, thrombus, and ischemia. Frozen tissues appear hard and cold, are without sensation, and take on a yellow-white or mottled blue-white appearance. The most serious complication of frostbite is seen with gradual thawing or refreezing. When tissues thaw slowly, partial refreezing may occur. Because the newer ice crystals formed are larger, they cause greater tissue damage. During thawing, as circulation returns, the area becomes purple and excruciatingly painful. Gangrene, a result of permanent cell death, may set in within a few days, requiring amputation **Figure 18-4**.

Figure 18-3 Frostbitten parts are hard and usually waxy to the touch.

Figure 18-4 Gangrene can occur when tissue is frozen and chemical changes occur in the cells.

Signs and Symptoms

Frostbite

- Hard, cold, waxy, and yellow-white or mottled blue-white skin

Differential Diagnosis

Frostbite

- Arterial occlusion

- Hypoxia or cyanosis
- Dermal exposure to corrosive chemicals
- Vasospasm

Transport Management

Frostbite

- Place a warm hand over the chilled ear, nose, or fingers.
- Do not warm, rub, or massage the area.
- If transport time is less than 1 hour, leave the part frozen.
- If transport time is more than 1 hour or longer, contact medical control to discuss field warming.
- If medical control advises field warming:
  - Immerse the affected tissue completely in water between 95°F and 104°F (35°C and 40°C). Monitor the water temperature.
  - Establish an IV line, and administer pain medication.
  - Dry the part thoroughly once it has thawed.
  - Apply a dressing.
- Take great care to prevent refreezing.

Treatment

Treatment of deep frostbite depends on two factors:

- The distance to the receiving hospital
- The degree of thawing before arrival at the hospital

If the part is still frozen or the transport time is reasonable (< 1 hour), the part should be left frozen. The part should be padded excessively to protect it from further injury. Warming should not be attempted during transport because rapid warming is difficult. The patient should not rub or massage the area because the ice crystals in the cells would lacerate delicate tissues. If the tissue is partially thawed or transport will exceed 1 hour, medical control should be contacted for advice on field warming. The affected tissue should be immersed completely in water between 95°F and 104°F (35°C and 40°C). An IV line should be established for the administration of pain control medication (such as morphine or fentanyl). The water temperature should be monitored to maintain it in the therapeutic range. This treatment procedure typically takes 30 minutes or more. The patient should not smoke during this procedure because nicotine causes vasoconstriction that would further interfere with the thawing process. Once circulation has returned and the part is thawed, it should be dried thoroughly and a dry dressing should be applied to further protect the tissues during transport. The tissues should not be allowed to refreeze.

Hypothermia

Hypothermia is defined as a decrease in core body temperature below 95°F (35°C). Hypothermia may be a result of impaired thermogenesis, excessive thermolysis, or excess environmental cold stress. Any environmental temperature below an individual's physiologic core body temperature can result in hypothermia. Hypothermia may occur in any season. Alcohol use has been identified as a factor

contributing to hypothermia. Hypothyroidism, liver disease, and malnutrition may further contribute to a predisposition to hypothermia. Trauma is the most important other factor contributing to hypothermia. Additionally, hypovolemia and hypotension interfere with normal thermoregulation.

Mild hypothermia is a core body temperature between 90°F and 95°F (32.2°C and 35°C). At these temperatures, the body usually compensates with increased thermogenesis and interrupted thermolysis. Shivering is in full force, and the "umbles" (stumbles, mumbles, fumbles, and grumbles) are present. The heart rate, blood pressure, and cardiac output may rise. These processes continue until the core body temperature returns to normal or glycogen stores are exhausted. If left unchecked, more serious conditions will result.

Severe hypothermia begins as the core body temperature drops below 90°F (32.2°C). At these temperatures, the body reacts by slowing things down. This slowed metabolism decreases thermogenesis, and the heart rate, blood pressure, and cardiac output decrease. As peripheral vasoconstriction occurs to shunt warm blood in the core, the volume receptors interpret the change as an increase in blood volume and stimulate the kidneys to produce more urine (cold diuresis). There is a simultaneous shift of fluids from the intravascular to the extravascular space, which increases the viscosity of blood. As the cold continues, the heart rate plummets. Shivering ceases at a body temperature less than 91°F (32.7°C). Tracheobronchial secretions increase and bronchospasm may occur. At a core body temperature of less than 90°F (32.2°C), hypoventilation is profound.

Bradycardia and cardiac arrhythmia, in particular atrial arrhythmias, may be seen. An Osborn or J wave may be seen on the ECG tracing **Figure 18-5**. Abnormal cardiac conduction and hypothermia-induced arrhythmias may lead to ventricular fibrillation, especially as the core body temperature falls below 82.5°F (28°C). Repeated defibrillation is not recommended until the patient has been warmed to a core body temperature of more than 86°F (30°C).

Special Populations

Elderly patients are more susceptible to hypothermia. Even in warm weather or inside a heated transport unit, older patients are susceptible to significant heat losses. Monitoring temperature is as important in the older population as in neonates.

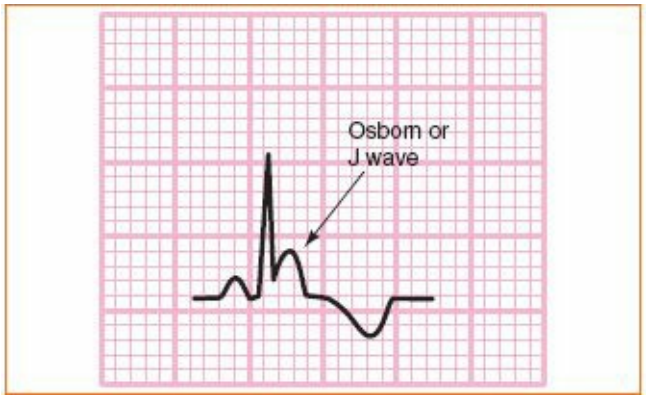

Figure 18-5 Osborn or J wave.

Many of the usual ACLS medications will not be effective for patients with severe hypothermia. American Heart Association guidelines recommend administering ACLS medications at greater intervals for patients with moderate hypothermia (86°F to 93.2°F [30°C to 34°C]). ACLS medications should be withheld for patients with severe hypothermia (< 86°F [30°C]) until the core body temperature has been

increased to more than 86°F (30°C). In general, atropine and cardiac pacing are ineffective for hypothermia-induced bradycardia. Lidocaine is also ineffective for preventing or treating hypothermia-induced ventricular arrhythmias. There is some limited evidence in animal studies that bretylium may be beneficial for preventing ventricular fibrillation in patients with hypothermia, but this practice is not currently recommended by American Heart Association guidelines.

Treatment

Treatment is based on warming the patient, which involves removing cold, wet clothing; drying and moving the patient to a warmed environment; and further passive or active warming of the truncal area. Active warming may include an infusion of warmed IV fluid (102°F to 105°F [38.8°C to 48.5°C]); warm, humid oxygen delivery; and a peritoneal lavage of a potassium chloride-free solution.

Expecting the Unexpected

Hypothermia can occur in any season of the year, especially in elderly people who have difficulty acclimatizing and are often prescribed multiple medications that affect the negative feedback loops of thermoregulation. CCTPs should pay particular attention during transport of elderly people. The transport environment creates numerous opportunities to cause or worsen preexisting hypothermia. Even brief episodes of exposure to cold temperatures while moving to or from a transport vehicle can significantly undermine rewarming efforts. Even when the ambient temperature is room temperature, young or elderly patients can be hypothermic. Although CCTPs may be sweating on a hot summer day, a patient may be too cold. It is essential that CCTPs sacrifice personal comfort by creating an adequately warm environment for the resuscitation or transportation of patients with hypothermia.

Signs and Symptoms

Hypothermia

- Decrease in core body temperature to less than 95°F (35°C)
- Shivering or lack of shivering
- Stumbling, mumbling, fumbling, and grumbling
- Increased or decreased heart rate
- Increased or decreased blood pressure
- Increased or decreased cardiac output
- Cardiac arrhythmias
- Osborn or J wave on the ECG

Differential Diagnosis

Hypothermia

- Drowning
- Profound shock
- Cardiac arrest
- Toxic chemical exposure
- Overdose

Transport Management

Hypothermia

- Remove cold, wet clothing.
- Dry the patient.
- Move the patient to a warmed environment.
- Consider warmed IV fluid, humidified oxygen, and peritoneal lavage of a potassium chloride-free solution.

Drowning

Drowning has been identified as the second leading cause of injury-related death among people between 1 and 15 years old. The term "near-drowning" was abandoned following the 2002 meeting of the first World Congress on Drowning. Experts narrowed the 33 former definitions of drowning to one definition that includes any process of respiratory impairment caused by submersion or immersion in liquid. Initial rescue should be conducted by specially trained personnel. CCTPs should never expose themselves or their crews to unwarranted hazards.

Transport Management

Drowning

- Provide spinal stabilization for patients witnessed to have dived into the water and when alcohol use is suspected.
- Provide supplemental oxygen.
- Perform endotracheal intubation.
- Provide positive end-expiratory pressure.
- Consider inserting a nasogastric tube to decompress the stomach.
- Consider administering a beta-2-adrenergic agonist to treat bronchospasm and tracheobronchial irritation.

Treatment

Treatment begins with spinal stabilization of patients witnessed diving into the water and when alcohol use is suspected. Ventilation should be assisted with supplemental oxygen as soon as possible, and advanced airway management where necessary. Widespread atelectasis and pulmonary shunt may be present in a drowning victim. The use of 10 cm H_2O of positive end-expiratory pressure, through the use of a commercially made additive valve designed for this purpose, may aid in keeping alveoli open and driving fluid into the interstitial or capillary spaces. A nasogastric tube may be used, after airway control, to decompress the stomach. Bronchospasm and tracheobronchial irritation may be treated with a beta-2-adrenergic agonist. Acute respiratory distress syndrome is a common complication of submersion incidents. Chemical or bacterial pneumonitis and renal failure are also complications that can occur days after resuscitation.

Diving Injuries and Decompression Sickness

Diving injury associated with recreational and professional scuba diving activities can result regardless of diver experience. All divers are subject to pressure effects. For each 33′ of depth, the pressure on the body increases by 1 atmo, or 14.7 lb/in^2. The most common effect is on nitrogen within the body. Nitrogen dissolves from gas inhaled into fatty tissues. Nitrogen readily displaces oxygen in the brain, resulting in feelings of euphoria and disorientation known as **nitrogen narcosis**. Nitrogen gas bubbles also expand rapidly on ascent, causing excruciating pain when trapped in body cavities such as joints or in the folds of the intestinal tract (the "bends") **Figure 18-6**. In addition to the bends, patients with decompression sickness may experience respiratory disturbances (the "chokes"), discussed later, neurologic impairment (the "staggers"), and skin sensation abnormalities (the "creeps" or "skin bends").

Barotrauma may occur during either ascent or descent, depending on the situation. Gases stored in confined spaces within the body may either expand or contract during changes in elevation. As a patient travels to a higher elevation, trapped gases attempt to expand, causing increased pressure within a particular body cavity. As a patient descends, the pressure of the outside environment exceeds the pressure of various gases within the body, resulting in inward pressure. The patient's lungs, ears, and gastrointestinal tract are prone to injury from barotrauma. Indwelling devices with inflated balloons, such as endotracheal tubes and laryngeal mask airways (LMAs), are also affected by changes in altitude. CCTPs should closely monitor these devices during ascent and descent. Rapid ascent is also responsible for **pulmonary overpressurization syndrome (POPS)**, in which gases in the lungs expand rapidly as the pressure decreases and pneumothorax or mediastinal or subcutaneous emphysema result. Simply holding one's breath during the last 6′ of the ascent is sufficient to cause these injuries. Arterial gas embolism may result, in which air bubbles escape from ruptured alveoli and enter pulmonary capillaries, travel through the pulmonary veins to the heart, and if they end up in the coronary arteries or cerebral vasculature, result in acute myocardial infarction or stroke. The vast majority proceed to cerebral arteries, resulting in a stroke.

Figure 18-6 Decompression sickness affects divers who ascend to the surface too quickly.

Transport Management

Diving Injuries

- Provide rapid transport.
- Provide oxygenation.
- Do not use positive end-expiratory pressure or continuous positive airway pressure (CPAP).

- Consider hyperbaric therapy.

Treatment for Decompression Sickness

Treatment in the prehospital setting includes rapid transport and oxygenation. Providers should administer 100% oxygen to promote nitrogen washout from the lungs. Positive end-expiratory pressure and CPAP should not be used for patients with diving injuries or decompression sickness unless necessary to maintain oxygenation.

A valuable resource for dealing with dive-related injuries is the Divers Alert Network (DAN), which provides a 24-hour hotline (919-684-8111). Hyperbaric oxygen therapy (HBOT) is often necessary to minimize the morbidity and mortality from decompression sickness. The DAN can guide CCTPs through a variety of complex decisions about treatment, transport, and patient disposition.

CCTPs should closely monitor patients with decompression sickness, even if they begin to show improvement or recovery. Relapses of symptoms are possible and may be associated with an overall deterioration of the patient's condition.

The use of a pure Trendelenburg or the combination of a head-down with left lateral decubitus (Durante) position is not currently recommended owing to increases in intracranial pressure. Patients should be transported in a supine position at the lowest cabin altitude pressure possible. Depending on the topography of a transport route, even ground ambulance transport may subject patients to increased physiologic stressors and worsen their condition.

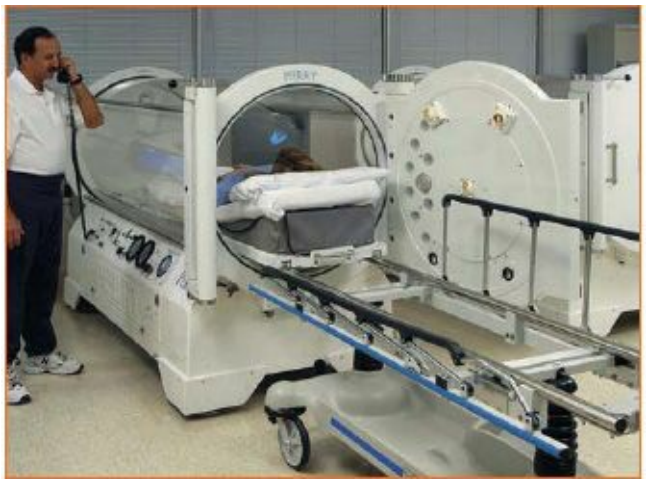

Figure 18-7 A hyperbaric chamber, usually a small room, is pressurized to more than atmospheric pressure and used in the treatment of decompression sickness and air embolism.

Other potential interventions for treatment or during transport may include the following: (1) aspirin for prevention of thrombus formation, (2) management of tension or simple pneumothorax with a thoracostomy (chest) tube, (3) aggressive IV fluid resuscitation, and (4) ACLS protocols for profound cardiopulmonary dysfunction. Patients with decompression sickness are likely to require analgesics, sedatives or anxiolytics, and antiemetics during transport. The patients may also have other concerns, such as trauma or hypothermia, that require additional resuscitative measures. Severe manifestations of decompression illness are likely to require transport for HBOT.

Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy is generally accomplished by placement of the patient in a specially constructed chamber designed to withstand internal pressures well in excess of normal atmospheric pressures. The chamber is filled with compressed air to a desired level, typically expressed as

atmospheres or feet of seawater. The patient receives pure or blended oxygen via mask or endotracheal tube **Figure 18-7**. Hyperbaric chambers can be in a fixed location or portable so they can be in proximity to high-risk diving activities. Chambers are configured to handle one patient or multiple patients. In larger chambers, a trained medical provider typically accompanies the patient into the chamber to monitor equipment and intervene if the patient's condition begins to deteriorate.

Most patients will receive 100% inspired oxygen while undergoing HBOT. In addition to saturating blood hemoglobin, oxygen under greater than atmospheric pressure becomes dissolved into the circulating plasma. HBOT has a number of interesting benefits to a variety of patients. Localized vasoconstriction from hyperoxygenation decreases tissue edema following crush injuries, burns, and other types of trauma. This vasoconstriction is offset by improved blood flow at the capillary level. HBOT also creates oxygen-free radicals and alters other components of a patient's inflammatory response, leading to improved wound healing and improvement of a pathologic inflammatory response. In specific situations, nitrogen and helium can be blended into the inspired oxygen.

TABLE 18-4 Indications for Hyperbaric Oxygen Therapy

Air or gas embolism
Carbon monoxide poisoning
Carbon monoxide poisoning complicated by cyanide poisoning
Clostridal myositis and myonecrosis (gas gangrene)
Crush injury, compartment syndrome, and other acute traumatic ischemias
Decompression sickness
Enhancement of healing in selected problem wounds
Exceptional blood loss (anemia)
Intracranial abscess
Necrotizing soft-tissue infections
Osteomyelitis (refractory)
Delayed radiation injury (soft-tissue and bony necrosis)
Skin grafts and flaps (compromised)
Thermal burns

Source: Reprinted with permission from the Undersea and Hyperbaric Medical Society (http://www.uhms.org).

The indications for HBOT have been established by the Committee on Hyperbaric Oxygenation of the Undersea and Hyperbaric Medical Society **Table 18-4**. HBOT facilities in North America and the Caribbean can be identified by contacting Duke University at 919-684-8111 or the DAN. The Undersea and Hyperbaric Medical Society also maintains a database of US and international hyperbaric chambers. With the wide variety of indications for HBOT, it is quite possible that hyperbaric chambers can be found in locations not typically associated with underwater activities. "Treatment tables" are used to determine the length of HBOT and treatment steps. These tables take into account the depth, time of dive,

decompression stops, and previous dives performed. A hyperbaric specialist can recommend which table to use. Patients with severe illness often require several HBOT treatments. HBOT may prove beneficial even several days or longer in select situations, such as carbon monoxide poisoning. Any time HBOT is considered, CCTPs or medical providers should seek specialty consultation with a provider experienced with hyperbaric medicine.

Transport to these special facilities often requires air medical flight. In cases of barotrauma, the flight should be planned to take place at low-level altitude (lowest altitude safely possible) or in a pressurized aircraft cabin set to 1 atm or ata. The sending medical providers, in conjunction with CCTPs, must carefully weigh the risks and potential benefits to a patient when selecting the appropriate transport vehicle for a patient requiring transport for HBOT. The DAN recommends that cabin altitude not exceed 800′ for aircraft transport of patients with decompression sickness. This altitude limit may not always be possible owing to terrain or topography, weather, aircraft limitations, or other factors.

Controversies

Although many training materials may not reflect the evolution of the ideal position for an injured diver, the trends have shifted over the years. Currently, certain observations have suggested that use of the Trendelenburg position may not provide the best in patient care. These findings include the limitations for resuscitation, increased cephalic venous volumes, and compromised subsequent middle ear equalization that occur in a head-down position, as well as the difficulty in distinguishing cerebral arterial gas embolism from cerebral decompression sickness. Likewise, significant arterial gas embolism most likely occurs in decompression sickness through the arterialization of venous bubbles. In the Trendelenburg position, venous return is increased, sending the bubbles back to the heart. Finally, studies show that brain function recovery time is longer for patients who are placed in a head-down position.

Ultimately, although there may be some occasional benefit to placing specific patients in a head-down position, many practitioners prefer keeping most patients supine.

Flight Considerations

According to DAN, if air evacuation is used, cabin pressure must remain near sea level and the aircraft should not go above 800′ unless the captain is required to do so for safety precautions. The diver should be placed in the lateral recumbent position, or the recovery position, with the patient on his or her side, the head supported at a low angle, and the upper leg bent at the knee. Gravity will help to keep the patient's airway clear. Other experts recommend that patients be transported in a supine position. Additional discussion of this subject is found in the controversies box.

Altitude Illness

Extremes of altitude pose life-threatening hazards for experienced mountain climbers pushing the limits of human exploration on remote peaks throughout the world. Scores of other people have similar, potentially catastrophic effects of altitude when they travel from lower elevations to higher elevations in the course of everyday life. People with preexisting medical problems, extremes of age, sedentary lifestyles, and poor health choices are at increased risk of altitude-related illness following even modest changes in elevation. The severity of altitude-related illness can range from mild symptoms, such as imperceptible tachypnea or sleep disturbances, to life-threatening manifestations, such as pulmonary edema, cerebral

edema, and hypoxia.

High altitude can also affect the body negatively, usually from a lack of acclimatization. Hypoxia from low atmospheric pressure is the main culprit in altitude sickness.

Altitude sickness is most commonly associated with mountain climbing and alpine skiing and is often called acute mountain sickness. Acute mountain sickness can occur at altitude changes of 3,000′ to 6,000′, but generally occurs at elevations above 8,000′ or higher. The Lake Louise criteria are useful for identifying acute mountain sickness. In the setting of a recent gain in altitude, these criteria include the presence of at least two of the following signs (crackles or wheezing in at least one lung field, central cyanosis, tachypnea, and tachycardia) and at least two of the following symptoms (dyspnea at rest, cough, weakness or decreased exercise performance, and chest tightness or congestion). More serious problems, **high-altitude pulmonary edema (HAPE)** and **high-altitude cerebral edema (HACE)** are likely at higher elevations.

HAPE is a noncardiogenic form of pulmonary edema that develops within 24 to 72 hours of reaching higher altitudes. Patients may demonstrate a variety of symptoms, including cough, respiratory distress, chest tightness, fatigue, and fever. The presence of fever may mislead clinicians and delay diagnosis.

The pathophysiologic features of HAPE are poorly understood. Pulmonary hypertension from alveolar hypoxia, localized inflammation, and capillary or arterial thromboses are implicated in patients with HAPE. People who make frequent changes in altitude are at increased risk of HAPE.

HACE should be considered a life-threatening emergency and should prompt immediate descent when recognized. Any patient who has recently experienced a significant gain in altitude and who now has mental status changes or ataxia should be evaluated for HACE. Either of the previously mentioned symptoms is presumed to be HACE if a particular person already has signs or symptoms of acute mountain sickness. If ataxia *and* mental status changes are present, HACE is presumed even if other signs or symptoms of acute mountain sickness are not present.

HACE is thought to be a result of cerebral vasodilation from hypoxia, although the precise mechanism has not been identified. Cerebral edema results from this enhanced blood flow, leading to the previously mentioned mental status changes and ataxia.

Once HACE is recognized, immediate descent should be initiated. Patients should receive supplemental oxygen during descent. Dexamethasone should be administered to patients with HACE. Diuretic medications are not indicated and have the potential to produce harmful alterations in fluid volume.

Signs and Symptoms

Altitude Illness

- Hypoxia
- Fatigue
- Weakness
- Near-syncope
- Headache
- Dyspnea
- Cough
- Pulmonary edema
- Chest tightness
- Tachycardia

Differential Diagnosis

Altitude Illness

- Any other possible cause of respiratory distress (such as asthma, chronic obstructive pulmonary disease, pneumonia, and bronchitis)
- Any other possible cause of altered mental status or ataxia (such as stroke, toxic exposure, encephalitis, and traumatic brain injury)
- Unrelated cardiovascular dysfunction (such as cardiogenic shock, cardiogenic pulmonary edema, and acute myocardial infarction)
- Unrelated gastrointestinal condition (such as gastroenteritis, gastritis, and pancreatitis)
- Labyrinthitis
- Electrolyte or hematologic abnormality

Transport Management

Altitude Illness

- Provide oxygenation.
- Evacuate the patient and begin descent.
- Consider hyperbaric therapy.

Rapid descent is the preferred treatment for HAPE. Patients should also receive supplemental oxygen during the descent. Nifedipine, salmeterol, and portable hyperbaric bags are effective and should be used if available for the management of HAPE. CPAP may also play a role in the management of these patients.

Flight Considerations

Patients with various altitude-related illnesses pose a significant challenge for CCTPs. It is quite conceivable that CCTPs may be asked to perform a rescue or evacuation function from a remote area with a high elevation. These locations are particularly hazardous for ground transport vehicles and aircraft. When requested to perform a rescue or evacuation mission, CCTPs should carefully consider safety issues, such as training and experience of the personnel, capabilities of the particular transport vehicle, and weather and environmental concerns, as well as the severity of illness and capabilities of a potential patient. Numerous emergency responders have been killed or severely injured while attempting rescue or evacuation of a patient from mountainous terrain.

During the transport, CCTPs must minimize the impact of environmental conditions on the patient. For patients with altitude-related illnesses, CCTPs must balance capabilities and safety concerns related to a particular transport vehicle with the patient's clinical needs. Ideally, descent should occur rapidly for patients with severe manifestations of altitude illness. Rapid or prompt descent may not be possible when weather, topography, and terrain complicate the transport.

Patients with altitude illness may have other issues, such as hypothermia, dehydration, trauma, or a preexisting medical condition that requires additional interventions. CCTPs must balance the needs for

warmth, privacy, and protection against patient care needs such as exposure, thorough assessment, and patient access for monitoring or procedures. It may be advisable in many situations to defer a thorough visual assessment or invasive interventions in the interest of keeping a patient clothed, covered, and warm during extreme environmental conditions.

Prolonged evacuation of a patient with altitude illness may also exceed the supplies, equipment, and capabilities of a particular transport team. When performing such transports, CCTPs should be mindful of critical supplies, such as oxygen, that may become rapidly depleted. Monitoring equipment, ventilator, and infusion pump batteries may not have the longevity to continue functioning during prolonged evacuation or transportation from remote locations.

Summary

Patients with conditions related to environmental emergencies can pose a significant challenge to CCTPs. These conditions are often difficult to diagnose and manage while in the transport environment, and there is a risk of further deterioration in a patient's condition during movement from one location or facility to another. CCTPs need to keep these conditions in mind when dealing with patients in a coma or those who have collapsed suddenly as a result of unknown causes. Knowing the risk factors that predispose a patient to environmental emergencies can aid in recognizing these conditions early in the patient encounter.

Case Study

YOU HAVE JUST ARRIVED FOR YOUR REGULARLY SCHEDULED ROTOR-WING SHIFT , when your dispatch center advises that you are being dispatched to the Rocky Mountain Rural Clinic, situated at 8,200′ above sea level, for a patient diagnosed as having probable HAPE. The patient needs to be transported to Mercy Hospital for HBOT. After your flight team (the pilot, flight nurse, and you, a critical care paramedic) completes the safety briefing, you depart for the rural clinic.

On arrival, your flight team is met by the attending physician. She advises you that the patient is a 42-year-old, 106-kg man who had been climbing in the local national park for the past 2 or 3 days with three other people when he started to complain of fatigue and severe dyspnea. The party began its descent from approximately 12,000′, which took approximately 9 hours, and then drove a vehicle to the local clinic. On arrival, the patient's dyspnea was mildly resolved; however, he had a decreasing level of consciousness.

Treatment in the clinic included oxygen via nonrebreathing mask at 15 L/min to treat central cyanosis, a chest x-ray that showed patchy bilateral infiltrates, and an ECG that indicated a right-sided heart strain pattern. The patient has an IV line with an 18-gauge needle in his left wrist running normal saline to keep the vein open; he also has an indwelling urinary catheter, with minimal urine output. Medications that were given included 10 mg of nifedipine (Procardia) by mouth and 80 mg of furosemide (Lasix) IV. The patient is presently resting but in moderate distress and has a Glasgow Coma Scale score of 13 (eye opening score, 3; verbal score, 4; and motor score, 6). Vital signs are a blood pressure of 142/81 mm Hg, a heart rate of 132 beats/min, sinus tachycardia with no ectopy, respirations of 28 breaths/min with rales bilaterally, and an Sp_{O_2} of 86% despite aggressive oxygen therapy. After a positive Allen test result, an arterial line was inserted into the right radial artery. Arterial blood gas readings show acute respiratory alkalosis with a pH of 7.49; Pa_{CO_2}, 30 mm Hg; Pa_{O_2}, 88 mm Hg; $H_{CO^-_3}$, 22 mEq/L; and base excess, 1. All available laboratory values are unremarkable.

As you are preparing to secure the patient onto your litter for the flight, he coughs up a small amount of blood-tinged sputum. Rather than electively intubating your patient for the flight, you decide to apply CPAP in an effort to decrease the patient's work of breathing. CPAP is applied initially at 5 cm H_2O, which seems to have no effect. You subsequently titrate the CPAP up to 7.5 cm H_2O, which decreases his

work of breathing and decreases his anxiety level. Your partner contacts your base hospital physician to advise of the patient's status, obtain any additional orders, and give an estimated time of arrival. The base hospital physician requests that 8 mg of dexamethasone (Decadron) and 250 mg of acetazolamide (Diamox) be administered en route. As your partner transfers the patient's IV to your pump and calibrates the arterial line to your monitor, you speak to the pilot and advise him that owing to the patient's condition, it is imperative to fly at a lower altitude to minimize the effects of altitude on the patient.

Once airborne, 8 mg of dexamethasone and 250 mg of acetazolamide are given IV, and all hemodynamic parameters are monitored throughout the 48-minute flight. Once you arrive at the Mercy Hospital helipad, the patient is taken directly to the hyperbaric chamber for definitive treatment.

1. How was the diagnosis of HAPE determined?
2. Why was the patient given nifedipine, furosemide, dexamethasone, and acetazolamide?
3. What are the potential problems that you may experience en route with this patient?

Analysis

Because of your critical care training, you are able to recognize that this patient is experiencing severe HAPE based on the Lake Louise acute mountain sickness criteria, which, in the setting of a recent gain in altitude, include the presence of at least two of the following signs (crackles or wheezing in at least one lung field, central cyanosis, tachypnea, and tachycardia) and at least two of the following symptoms (dyspnea at rest, cough, weakness or decreased exercise performance, and chest tightness or congestion).

Although you realize that HAPE usually resolves with descent from altitude, this patient was experiencing a severe case that did not resolve, and, in fact, the patient's condition appeared to be deteriorating.

Nifedipine is given in cases of HAPE in an effort to promote vasodilation of the pulmonary vessels, and furosemide is given to promote diuresis. Acetazolamide is given in cases of HAPE to increase the amount of bicarbonate excreted in the urine, making the blood slightly acidic, which, in turn, stimulates ventilation, increasing the amount of oxygen in the bloodstream. Dexamethasone is given to treat pulmonary edema and to minimize cerebral edema.

This patient has the potential to experience several problems en route. Owing to the 48-minute flight to the receiving hospital and the fact that the patient is receiving CPAP, the potential to run out of oxygen is a risk; however, by performing a thorough pre-flight of your aircraft, oxygen cylinders that are almost depleted should have been noted and replaced before your mission. In any patient with an altered mental status in the flight environment, CCTPs need to be aware of the possible need to perform definitive airway management, which is often difficult in the confined space of a helicopter. Finally, this patient was experiencing an acute case of HAPE, and, depending on the altitude required during the flight, his pulmonary edema could progressively worsen. Good communication between the crew and the pilot will help to minimize the effects of pressurization, thus minimizing complications en route.

Prep Kit

Ready for Review

- Very young and very old people, people taking certain medications, and people in a poor state of health are predisposed to environmental emergencies. The challenge for CCTPs is to recognize patients who are susceptible to environmental exposure in conditions that are not extremely hot or cold. CCTPs must also initiate or continue critical interventions during transport.

- The hypothalamus acts as the body's master thermostat, using negative feedback control. The three major components of the negative feedback system are temperature receptors, effector organ systems, and integrator or controller.
- To reduce excess body heat, blood vessels in the skin dilate, perspiration increases, and muscle activity is inhibited.
- To increase body heat, the posterior hypothalamus is stimulated to constrict blood vessels, and muscle activity, and the piloerector muscles are stimulated.
- The heat produced at rest by normal metabolic reactions is referred to as the basal metabolic rate (BMR). The BMR can be affected by age, sex, stress, hormones, and body surface area. As the ratio of body surface area to body volume increases, heat loss increases.
- Heat warms the body and maintains the core body temperature. The body transfers heat to the environment through radiation, conduction, convection, and evaporation. If the environment is hotter than the body, the body gains heat through absorption. Humidity can affect thermoregulation by impairing the evaporative process.
- In very cold temperatures, a fair amount of heat is lost through respiration. The body shifts to thermogenesis when the environment is cold. The BMR and physical exertion are increased.
- Heat illness occurs when an increase in core body temperature exceeds the body's ability to dissipate the excess heat. CCTPs in all geographic regions of the United States should be prepared to transport patients with heat-related illnesses.
- CCTPs should be familiar with medications and other factors that contribute to heat illness.
- Heat cramps are the result of a water-sodium imbalance and are often not accompanied by hyperthermia. Water intoxication may occur if the person tries to replenish lost fluids by drinking excessive amounts of free water in a very short time. Additional cooling measures may be needed if the person has heat exhaustion, heat stroke, or significant hyperthermia.
- The condition of patients with heat cramps usually improves after they are removed from the hot environment and drink a salt-containing liquid or an electrolyte drink. CCTPs should use caution when administering IV fluids to correct presumed hyponatremia if there is any possibility of hyponatremia.
- Treatment of heat syncope focuses on placing the patient in the recovery position and allowing fluid intake if mental status permits. If the patient does not recover quickly, heat exhaustion or heat stroke should be suspected.
- Two forms of heat exhaustion are water-depleted and sodium-depleted. Symptoms range from headache and fatigue to orthostatic hypotension and darker, brown urine. If left untreated, heat exhaustion may progress to heat stroke.
- Heat stroke is a severe disturbance in the body's thermoregulation, resulting in altered mental status and a core body temperature of more than 104°F (40°C). The critical thermal maximum is reached when the core body temperature exceeds 109°F (42.7°C).
- CCTPs should be aware of the differences between classic and exertional heat stroke and be suspicious of heat stroke during heat waves, when dealing with coma of unknown origin, and with cases of summer flu.
- Heat emergency can occur at any time of year, especially in elderly people. CCTPs should pay attention to signs of poor skin turgor and lack of sweating when transporting an elderly patient with a fever. The patient's history is also an important source of information in diagnosing heat stroke.
- Treatment of heat stroke focuses on regaining control of the core body temperature. Evaporative

cooling can be started and continued during transport.

- Most cold-related injuries are localized to exposed body parts. The most susceptible parts are the tip of the nose, the ear lobes, and the fingers and toes.
- Superficial frostbite (frostnip) comes on slowly, generally occurs without much pain, and is easily treated by placing a warm hand over the chilled area.
- Deeper frostbite involves the freezing of tissues. The most serious complication of frostbite occurs in gradual thawing or refreezing. Gangrene may set in within a few days, requiring amputation. Treatment depends on the distance to the receiving hospital and the degree of thaw before arrival at the hospital.
- Hypothermia is a decrease in core body temperature to less than 95°F (35°C). Mild hypothermia is defined by a core body temperature between 90°F and 95°F (32.2°C and 35°C). The body usually compensates for mild hypothermia with increased thermogenesis and interrupted thermolysis.
- Severe hypothermia occurs when the core body temperature drops below 90°F (32.2°C). The body reacts by slowing metabolism, which decreases thermogenesis, heart rate, blood pressure, and cardiac output and has many other effects. Treatment is based on warming the patient.
- Hypothermia can occur at any time of year, particularly in elderly people. CCTPs should sacrifice personal comfort to create an adequately warm environment for the resuscitation or transport of patients with hypothermia.
- Drowning includes any process of respiratory impairment from submersion or immersion in liquid. Only specially trained personnel should attempt to rescue a drowning victim from water.
- Divers may experience nitrogen narcosis, caused by nitrogen dissolving from gas inhaled into fatty tissues. Nitrogen gas bubbles expanding rapidly on ascent may cause the “bends,” the “chokes,” the “staggers,” and the “creeps” or “skin bends.”
- Barotrauma may occur during either ascent or descent. The patient’s lungs, ears, and gastrointestinal tract are prone to injury from barotrauma.
- Pulmonary overpressurization syndrome is caused by rapid ascent. Arterial gas embolism may occur, which can result in acute myocardial infarction or stroke.
- CCTPs can use the Divers Alert Network (DAN) hotline for guidance through decisions about treatment, transport, and patient disposition. CCTPs should closely monitor patients with decompression sickness, even if they show improvement or recovery. Relapses are possible.
- Hyperbaric oxygen therapy may be indicated for patients with decompression sickness. CCTPs should also be aware of other indications for hyperbaric therapy. Hyperbaric oxygen therapy facilities can be identified by contacting Duke University or the DAN.
- If air evacuation is used for an injured diver, cabin pressure must be kept near sea level and the aircraft should not fly above 800′ unless necessary for safety precautions. The latest evidence suggests that the supine position is best for transporting patients with diving injuries.
- Altitude sickness is also called acute mountain sickness. The Lake Louise criteria are useful for identifying acute mountain sickness.
- High-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE) are likely at higher elevations.
- HAPE may develop within 24 to 72 hours of reaching higher altitudes. Symptoms include cough, respiratory distress, chest tightness, fatigue, and fever. Rapid descent is the preferred treatment for HAPE, along with supplemental oxygen.

- HACE is life threatening; when it is recognized, immediate descent should be initiated. Mental status changes and ataxia are symptoms of HACE.
- CCTPs may be asked to rescue or evacuate a patient from a remote area at a high elevation. These locations are very hazardous, and safety issues such as training and experience of personnel, capabilities of the transport vehicle, weather and environmental concerns, and the severity of illness and capabilities of the patient must be considered.
- Capabilities and safety concerns related to the transport vehicle must be balanced with the patient's clinical needs. Although rapid descent is ideal for patients with severe altitude illness, it may not be possible owing to environmental factors. A thorough visual assessment or invasive interventions may have to be deferred because of environmental conditions. Critical supplies and equipment must be monitored closely.

Vital Vocabulary

absorption Acquisition of additional heat, radiation, or other energy from the environment; or, a substance's molecules are moved from the site of entry on the body into the systemic circulation.

barotrauma Injury to tissues, organs, or structures within the body, resulting from rapid or significant changes in environmental air pressure.

basal metabolic rate (BMR) The heat energy produced at rest by normal body metabolic reactions, determined mostly by the liver and skeletal muscles.

conduction Transfer of heat to a solid object or a liquid by direct contact.

convection The mechanism by which heat (body heat in the context of this chapter) is picked up and carried away by moving air currents.

deep frostbite A type of frostbite in which the affected part looks white, yellow-white, or mottled blue-white and is hard, cold, and without sensation.

evaporation The conversion of a liquid to a gas.

exertional hyponatremia A low sodium level as a result of prolonged exertion in hot environments coupled with excessive hypotonic fluid intake that leads to nausea, vomiting, and, in severe cases, mental status changes and seizures.

frostbite Localized damage to tissues resulting from prolonged exposure to extreme cold.

heat cramps Acute and involuntary muscle pains, usually in the lower extremities, the abdomen, or both, that occur because of profuse sweating and subsequent sodium loss in sweat.

heat exhaustion A clinical syndrome characterized by volume depletion and heat stress that is thought to be a milder form of heat illness and on a continuum leading to heat stroke.

heat stroke The least common and most deadly heat illness, caused by a severe disturbance in thermoregulation, usually characterized by a core body temperature of more than 104°F (40°C) and altered mental status.

heat syncope An orthostatic or near-syncopal episode that typically occurs in nonacclimated people who may be under heat stress.

high-altitude cerebral edema (HACE) An altitude illness in which there is a change in mental status and/or ataxia in a person with acute mountain sickness or the presence of mental status changes and ataxia in a person without acute mountain sickness.

high-altitude pulmonary edema (HAPE) An altitude illness characterized by dyspnea at rest, cough, severe weakness, and drowsiness that may eventually lead to central cyanosis, audible rales or wheezing, tachypnea, and tachycardia.

hypothalamic-pituitary axis An interrelationship between the hypothalamus and pituitary gland in which a releasing or an inhibiting factor is sent from the hypothalamus to the pituitary, resulting in an increase or a decrease in metabolism and other functions throughout the body.

hypothalamo-hypophyseal portal system The venules between the capillaries in the hypothalamus and pituitary gland by which the hypothalamus sends releasing or inhibiting factors to the pituitary gland, thereby increasing or decreasing metabolism.

hypothalamus The most inferior portion of the diencephalon; it is responsible for control of many body functions, including heart rate, digestion, sexual development, temperature regulation, emotion, hunger, thirst, and regulation of the sleep cycle.

hypothermia A condition in which the core body temperature decreases to less than 95°F (35°C).

malignant hyperthermia A condition that can result from common anesthesia medications (notably succinylcholine) and present with hyperthermia, muscular rigidity, altered mental status, and a hyperdynamic state.

mild hypothermia A condition in which the core body temperature is between 90°F and 95°F (32.2°C and 35°C); at this stage, the body usually compensates with increased thermogenesis and interrupted thermolysis.

neuroleptic malignant syndrome A condition caused by anti-psychotic and common antiemetic medications that presents with hyperthermia, muscular rigidity, altered mental status, and a hyperdynamic state.

nitrogen narcosis A state resembling alcohol intoxication produced by nitrogen gas dissolved in the blood at high ambient pressure.

pulmonary overpressurization syndrome (POPS) Also called “burst lung,” this diving emergency can occur during ascent and can cause pneumothorax, mediastinal and subcutaneous emphysema, alveolar hemorrhage, and the lethal arterial gas embolism.

radiation Emission of heat from an object into surrounding, colder air.

severe hypothermia A condition in which the core body temperature drops to less than 90°F (32.2°C).

superficial frostbite A type of frostbite characterized by altered sensation (numbness, tingling, or burning) and white, waxy skin that is firm to palpation, but the underlying tissues remain soft.

thermogenesis The production of heat in the body.

thermolysis The liberation of heat from the body.

thermoregulation The process by which the body maintains temperature through a combination of heat gain by metabolic processes and muscular movement and heat loss through respiration, evaporation, conduction, convection, and perspiration.

water intoxication A condition that occurs when the normal balance of electrolytes in the body is pushed outside safe limits by the overconsumption of water.

Bibliography

American Heart Association. 2005 American Heart Association Guidelines for Cardiopulmonary

Resuscitation and Emergency Cardiovascular Care, part 10.4: hypothermia [published online ahead of print November 28, 2005]. *Circulation*. 2005(suppl 1);112:IV-136–IV-138. doi:10.1161/CIRCULATIONAHA.105.166566. Available at: http://circ.ahajournals.org/cgi/content/full/112/24_suppl/IV-136. Accessed June 22, 2009.

Barotrauma/decompression sickness treatment. WebMD. 2006. Available at: http://firstaid.webmd.com/barotraumadecompression-sickness-treatment. Accessed April 27, 2009.

Beers M, Berkow R. *The Merck Manual of Diagnosis and Therapy: Centennial Edition*, 17th ed. Whitehouse Station, NJ: Merck Research Laboratories; 1999:2447–2468, 2507–2508.

Caroline N. *Emergency Care in the Streets*, 6th ed. Sudbury, MA: Jones and Bartlett Publishers and the American Academy of Orthopaedic Surgeons; 2008:35.4–35.32.

Centers for Disease Control and Prevention. Extreme heat: a prevention guide to promote your personal health and safety. 2006. Available at: http://www.bt.cdc.gov/disasters/extremeheat/heat_guide.asp. Accessed June 19, 2009.

Craig S. Hyponatremia: treatment and medication. 2009. Available at: http://emedicine.medscape.com/article/767624-treatment. Accessed June 19, 2009.

Divers Alert Network. Diving medicine FAQs: proper position for emergency care. Available at: http://www.diversalertnetwork.org/medical/faq/faq.aspx?faqid=193. Accessed April 27, 2009.

Edelstein JA, Li J. Hypothermia. 2007. Available at: http://emedicine.medscape.com/article/770542-overview. Accessed June 19, 2009.

Goldberg S. *Clinical Physiology Made Ridiculously Simple*. Miami, FL: MedMaster Inc; 1997:47, 54–55, 60, 137.

Haskell GH. *Paramedic Pearls of Wisdom*, 2nd ed. Sudbury, MA: Jones and Bartlett Publishers; 2006:273–286.

Helman RS, Habal R. Heatstroke. *emedicine*. 2007. Available at: http://emedicine.medscape.com/article/166320-overview. Accessed June 18, 2009.

Jardine DS. Heat illness and heat stroke. Pediatrics in Review. American Academy of Pediatrics. 2007;28(7):249–258. Available at: http://pedsinreview.aappublications.org/cgi/content/extract/28/7/249. Accessed July 8, 2009.

Kale R, Anholm JD. Altitude-related disorders. *emedicine*. 2009. Available at: http://emedicine.medscape.com/article/303571-overview. Accessed June 18, 2009.

Knochel JP. Heat cramps. *The Merck Manuals Online Medical Library: The Merck Manual for Health Professionals*. 2009. Available at: http://www.merck.com/mmpe/sec21/ch318/ch318b.html. Accessed June 19, 2009.

Latham E, Hare MA, Neumeister M. Hyperbaric oxygen therapy. 2008. Available at: http://emedicine.medscape.com/article/1464149-overview. Accessed June 18, 2009. MedicineNet.com. Definition of barotraumas. Available at: http://www.medterms.com/script/main/art.asp?articlekey=31722. Accessed July 8, 2009.

Pulley SA. Decompression sickness. 2007. Available at: http://emedicine.medscape.com/article/769717-overview. Accessed June 18, 2009.

Rothrock SG. *Tarascon Adult Emergency Pocketbook*. 4th ed. Sudbury, MA: Jones and Bartlett Publishers; 2009.

Simon EE. Hyponatremia. 2009. Available at: http://emedicine.medscape.com/article/242166-overview.

Accessed July 8, 2009.

Thalmann ED. Decompression illness: what is it and what is the treatment? Divers Alert Network Web site. 2004. Available at: http://www.diversalertnetwork.org/medical/articles/article.asp?articleid=65. Accessed June 22, 2009.

Undersea and Hyperbaric Medical Society. A directory of hyperbaric treatment chambers. 2007. Available at: http://www.uhms.org/Default.aspx?tabid=175. Accessed June 22, 2009.

Undersea and Hyperbaric Medical Society. Indications for hyperbaric oxygen therapy. 2007. Available at: http://www.uhms.org/Default.aspx?tabid=270. Accessed June 22, 2009.

Vachiery JL, Reuse C, Blecic S, Contempre B, Vincent JL. Bretylium tosylate versus lidocaine in experimental cardiac arrest. *Am J Emerg Med*. 1990;8:492–495.

Vellaichamy M. Hyponatremia. 2009. Available at: http://emedicine.medscape.com/article/907841-overview. Accessed July 8, 2009.

Wake County EMS ICE Chest. Available at: http://www.wakeems.com/saem. Accessed April 27, 2009.

Infectious and Communicable Diseases

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Objectives

1. Describe the differences between eukaryotic and prokaryotic microorganisms (p 741).
2. Describe the types of immunity and the components of humoral and cell-mediated immunity (p 742–744).
3. Understand the types of anaphylaxis and state the signs, symptoms, and treatment of anaphylaxis (p 744–746).
4. Discuss the implications of transporting patients with immunodeficiencies (p 746).
5. Discuss the differences between normal, opportunistic, and pathogenic organisms (p 746–747).
6. State the virulence factors found in pathogenic organisms and how they can be spread from bacterial cell to bacterial cell (p 747–748).
7. Describe the portals of entry for infectious organisms and the process of infection once entry is gained (p 747).
8. Compare the effects of bacterial endotoxins and exotoxins (p 748).
9. Describe viral pathogenesis (p 749).
10. Give examples of reportable or notifiable infectious diseases (p 750).
11. Discuss epidemiology, including reservoirs of infection and transmission of infectious disease (p 749–751).
12. Discuss the etiologic agents, mode of transmission, signs and symptoms, and treatment of the following:
    - Meningitis (bacterial and viral) Respiratory syncytial virus
    - Necrotizing fasciitis
    - Epiglottitis
    - Tuberculosis
    - Pneumonia Fungal diseases Influenza
    - Various herpesviruses
    - Viral hepatitis
    - Human immunodeficiency virus Severe acute respiratory syndrome
    - *Escherichia coli* O157:H7
    - West Nile virus
    - *Helicobacter pylori*
    - Methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant *S aureus*
    - Vancomycin-resistant enterococci
    - Rickettsial diseases such as Rocky Mountain spotted fever (p 751–775)
13. Understand acquired immunodeficiency syndrome–defining conditions (p 763–766).

14. State the recommendations for use of standard precautions in all health care settings (p 768).
15. Understand how the overuse and misuse of antimicrobials leads to resistant infectious organisms (p 772–773).
16. Discuss the appropriate and correct use of personal protective devices (both donning and doffing) (p 769).
17. Understand the vaccinations needed for CCTPs and health care workers in general (p 776—777).
18. State universal precautions and discuss situations when these should be used (p 777).
19. State isolation, airborne, and droplet precautions and discuss situations when these should be used (p 777–778).
20. Discuss the importance of handwashing and use of hand sanitizers and/or hand antiseptics in preventing contamination and transmission of infectious diseases (p 778).
21. Understand decontamination chemicals and techniques appropriate for cleanup after transporting patients harboring various infectious diseases. Include decontamination of equipment and vehicle surfaces as well as uniforms (clothing and shoes) and personal protective equipment (p 778–782).
22. Discuss actions to take after blood exposure (p 782–783).

Introduction

CCTPs must be keenly aware of their risk of exposure to a multitude of infectious agents in the course of transporting patients. Patients, health care workers, and members of the community are at risk for contracting infectious diseases from their interactions with one another. Infectious agents are found in body fluids, on solid surfaces, and in the air. Transmission can occur directly from either a symptomatic or asymptomatic carrier, indirectly from contact with a **fomite** (eg, contaminated fluids or equipment), or from inoculation by a **vector** (eg, exchange of a pathogen via an animal or insect bite or sting). Patients are more susceptible to contracting infectious diseases because their defenses are diminished from sickness, immunocompromised states, broken skin, open wounds, and/or **iatrogenic** events. CCTPs are at increased risk for contracting an infectious agent because of the idiosyncratic nature of their job. They provide care for a large number of patients and, therefore, have a high frequency of exposure to serious diseases. Additionally, the work of a CCTP often involves the use of invasive instruments while being jostled about in a moving vehicle, providing for an increased risk of needlesticks and exposure to body fluids. The community is at risk if an infection is not contained, if members of the community are not immunized, or if the medical equipment used is not properly and adequately sanitized between patients. A solid understanding of the fundamental principles of infectious disease is an essential tool that will enable CCTPs to assess risk, minimize exposure, and prevent transmission of infection.

Fundamental Principles of Infectious Disease

Classification

Before the realm of the microscopic world was discovered, living organisms were classified as either animal or vegetable. After the discovery of microorganisms, scientists were not able to classify microbial life into the dichotomous groups of animal vs vegetable because some had characteristics of both, and others (such as viruses and prions) fit into neither category. A formal classification scheme for microorganisms was not developed until the late 1960s, when Carl Woese devised a system that classified microorganisms according to their physical properties and cellular components. A list of the

major classes of microorganisms and their properties is found in **Table 19-1.**

The same physical properties and components used to determine the classification of microorganisms also affect how they invade hosts, set up an infection, and cause disease. Microorganisms possess properties that are unlike their human hosts, and these differences are exploited when scientists are developing agents for inhibiting growth or eliminating infection. The practice of chemotherapy began as an attempt to find chemical agents effective in controlling microbial infection. The goal of such chemical therapy is to eradicate the offending organism without causing irreparable injury to the host. During World War I, Paul Ehrlich, a German physician, was the first to develop a synthetic agent—a "magic bullet"—that would target and destroy the invader while minimizing collateral damage to the host. This "magic bullet" archetype is still used in medicine today. All antibiotic, antiviral, and antifungal drugs are designed to target specific proteins, macromolecules, cellular organelles, and enzymatic pathways; or reproductive strategies unique to the infectious agent that are not shared by their human host.

Human Immune System

The diseases that affect humans do not arise in a vacuum, but result from complex interactions between the host, the agent, and the environment. For an organism to contract a disease, the host must be susceptible. Susceptibility is determined by factors such as nutritional status, immune status, genetic makeup, living conditions, and exposure. Disease can be transmitted directly through inhalation, ingestion, or direct contact with infectious agents; or indirectly via vectors, fomites, or some other complex cycle of interactions.

TABLE 19-1 Classification of Microorganisms

Organism	General Information	Cell Wall/ Cell Membrane Components	Drug Classes, Groups of Actions (Use)	Medication Examples
Bacteria	Prokaryote vs eukaryote: prokaryotes Cellularity: unicellular Reproduction: binary fission	Peptidoglycan cell wall No sterols in the membrane (except *Mycoplasma*) Mycolic acid in the cell wall of mycobacteria	Cell wall synthesis inhibitors • Beta-lactams • Glycopeptides Protein synthesis inhibitors • Tetracyclines • Aminoglycosides • Macrolides DNA/RNA synthesis inhibitors • Quinolones or fluoroquinolones Alter cell membrane permeability Antimetabolites • Sulfonamides	 Penicillins and cephalosporins Vancomycin (Lyphocin, Vancocin, Vancophed) Doxycycline (Vibramycin, Pentostat) Amikacin (Amikin) and gentamicin (Garamycin) Erythromycin (many brand names) Ciprofloxacin (Ciloxan, Cipro) Polymyxin B (Aerosporin) Sulfamethoxazole (Gantanol)
Viruses	Prokaryote vs eukaryote: neither Cellularity: acellular Reproduction: N/A	None (lack cells)	Viral attachment inhibitor Viral uncoating inhibitor (influenza) Neuraminidase inhibitors (influenza) DNA/RNA synthesis inhibitors (HIV, herpes, cytomegalovirus) Protease inhibitors (HIV) Interferon (hepatitis)	Enfuvirtide (Fuzeon) Amantadine (Symmetrel) and rimantadine (Flumadine) Zanamivir (Relenza) and oseltamivir (Tamiflu) Acyclovir (Zovirax), ganciclovir (Cytovene), and zidovudine (AZT, Retrovir) Indinavir (Crixivan) and saquinavir (Fortovase, Invirase) Interferon (Intergen)
Protozoa	Prokaryote vs eukaryote: eukaryotes Cellularity: unicellular Reproduction: asexual or sexual	No cell wall	Antimetabolites DNA synthesis inhibitors • Nitroimidazoles Unknown mode of action • Quinolines	Trimethoprim-sulfamethoxazole combination (Septra) Metronidazole (Flagyl, Metrogel) Chloroquine (Aralen) and quinacrine
Helminths	Prokaryote vs eukaryote: eukaryotes Cellularity: multicellular Reproduction: complex	No cell wall	Antimetabolites • Benzimidazole	 Mebendazole (Vermox) and Praziquantel (Biltricide)
Fungi	Prokaryote vs eukaryote: eukaryotes Cellularity: unicellular (yeasts) or multicellular (molds) Reproduction: asexual or sexual	Chitin cell wall Unique sterols (ergosterol) in the membrane	Ergosterol synthesis inhibitors • Azoles • Allylamines Alters cell membrane permeability • Polyene Mitotic inhibitor (prevents cell replication)	 Ketoconazole (Nizoral) and miconazole (Monistat, Micatin) Terbinafine (Lamisil) Amphotericin B (Fungizone) Griseofulvin (Fulvicin, Grisactin, Grisfulvin)
Prions	Prokaryote vs eukaryote: neither Cellularity: acellular Reproduction: N/A	None (lack cells)	N/A	N/A

Abbreviations: HIV, human immunodeficiency virus; N/A, not applicable.

Within the human body, there is a constant struggle between the host and would-be invaders, both competing for nutrients, energy, and control of cellular synthetic mechanisms. The perpetual struggle between host and microbe pits the human immune defenses against the microbial virulence factors. For the immune system to protect the body from invading microorganisms, it must be able to recognize "self" components or cells and distinguish those from nonself components or cells. The immune system must be both highly specific (keeping watch for specific antigens) and general (able to keep up with numerous assaults without sending the body into a perpetual state of inflammation). The protection it provides must consist of several layers so that once one level of immunity has been breached, additional methods of fighting infection are launched and continue the assault. This allows a gradation of response, appropriate for the current threat. The immune system must be capable of "remembering" previous interactions so that it is capable of mounting a response to the same or a closely related agent years after the initial exposure. All of these components must work in a coordinated effort, providing protection from infection and

malignancy, without self-destruction.

Types of Immunity

Immunity can be either innate or acquired. **Innate immunity** is the type of immunity an individual is born with. It provides protection from invading pathogens despite a lack of prior exposure. The skin and mucous membranes and their secretions provide mechanical and chemical barriers to invasion and are the primary components of innate immunity, providing protection against infection from birth. Innate immunity is nonspecific in nature, is activated immediately on invasion, and has no memory. It exists before exposure to the agent has occurred, and does not improve with repeated exposures. The components of the innate immune system work to eliminate invading organisms through various methods, including activation of the adaptive immune response, prevention of entry of the microorganisms into the body, and limitation of microbial growth within the body. Nonspecific factors of the immune system help limit growth of microorganisms within the body and include cells such as natural killer cells, neutrophils, and macrophages; proteins such as complement or transferrin; and the body's general responses to invasion with actions such as fever or inflammation. Macrophages are a type of white blood cell that ingest and destroy microbes. Natural killer cells, another type of white blood cell, have the job of recognizing and killing host cells that are infected with a virus. The complement system is a group of proteins that, when activated, begin a cascade of events that aid in the clearing of bacterial cells. Transferrin is a carrier protein that sequesters iron, keeping that vital element unavailable for the invading microbes.

Acquired immunity develops as a result of interactions between components of the immune system and the invading microbe, thus providing improved protection with repeated exposures to specific agents. The two main types of acquired immunity are humoral and cell-mediated immunity. Humoral immunity is provided by B cells through the production of antibodies and complement. Cell-mediated immunity is provided by T cells. Cytokines and macrophages are modulators of both cell-mediated and humoral immunity, providing signals to the players—revving up or toning down the immune response as appropriate. Acquired immunity results from the interaction of the agent with the host, is specific in nature, improves with repeated interactions, and allows for the development of immunologic memory. It is mediated by antibodies, B and T lymphocytes, and can be either active or passive.

Active immunity refers to an ongoing process of developing antibodies and activating T cells in response to invading agents. This occurs either when an individual is exposed to microbes through the natural process of infection or artificially stimulated through a vaccine.

Passive immunity is the process of giving an individual a preformed antibody (from a donor) in the event of an exposure to which the individual has not yet developed immunity. For example, if a provider were to be exposed to a patient known to have hepatitis B, the provider could be given hepatitis B immunoglobulin (HBIG) during the incubation period to prevent infection. Another example of passive immunity is the passing of antibodies from the mother to her fetus or infant by crossing the placenta into fetal circulation, or through breast milk.

Active immunity, once developed, provides long-lasting protection against invading microbes, but is initially slow to develop. Passive immunity is immediate, but does not confer a lasting effect, and because it does not originate with the individual, it must be continually re-dosed. There is also some level of risk of contracting blood-borne infectious diseases from the use of preformed or pooled human immunoglobulin.

Antigen

Antigens are typically proteins and/or sugars that are anchored into the cell membrane and displayed on the cell's surface. An antigen is recognized by the immune system as either self or nonself. All cells

display antigens, even our own cells. If an antigen is foreign or nonself, it will induce a response, either humoral or cell-mediated. An antigen can be presented to B or T cells by antigen-presenting cells, or it can react directly with an antibody. An antigen usually elicits an immune response and is, therefore, termed immunogenic, but there are some low-molecular-weight molecules called **haptens** that react with antibodies without turning on the immune system.

Organs and Cells of the Immune System

The immune system is complex and is composed of many organs and cell types. The organs of the immune system are categorized by anatomic location, either central or peripheral. The lymphatic system is the network of capillaries, vessels, ducts, nodes, and organs that produces lymph and conveys it through the body **Figure 19-1**. The central lymphoid organs are the thymus and the bone marrow. The thymus is located in the mediastinal cavity anterior and rostral to the heart. It weighs 15 to 35 g at birth and continues to increase in size until puberty, when it begins to regress and is replaced by fibrotic tissue. The thymus is where the T lymphocytes differentiate. In contrast, the bone marrow is the site of B-cell synthesis. The precursor B cells are first recognized in the fetal liver, but migrate to the bone marrow before birth, and lymphopoesis continues there throughout childhood into adult life. The central lymphoid organs are the site of synthesis and differentiation of immunocompetent cells.

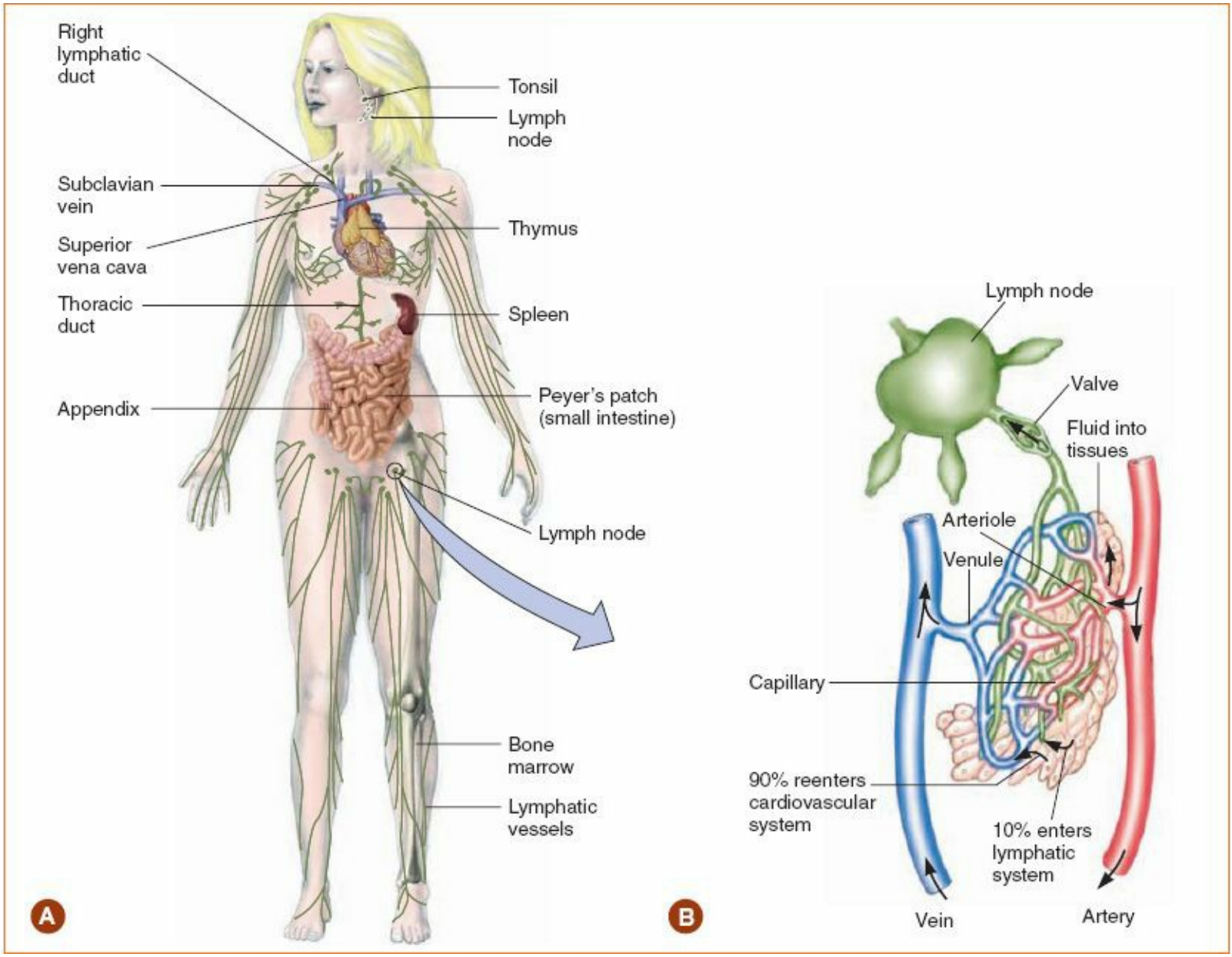

Figure 19-1 The lymphatic system. **A.** The lymphatic system consists of vessels that transport lymph and excess tissue fluid back to the circulatory system. **B.** Lymph is picked up by lymphatic capillaries that drain into larger vessels. Like the veins, the lymphatic vessels contains valves that prohibit backflow. Lymph nodes are interspersed along the vessels and filter the lymph.

The peripheral lymphoid organs are where immunocompetency is expressed; they include the spleen, lymph nodes, tonsils, intestinal Peyer's patches, and mucosa. The spleen is where encapsulated bacteria are encountered and destroyed. If the organ is removed, the patient may be more susceptible to infection, especially from *Streptococcus pneumoniae* and *Haemophilus influenzae*.

Lymph nodes are made up of lymphoidal tissue located throughout the body as single units or in chains of units and are prominent in the neck, axilla, and groin. Lymphadenopathy is an increase in the size of a lymph node or lymph nodes and indicates a high level of immunologic activity. Lymphadenopathy is a clinical sign commonly seen in patients fighting infection or cancer.

T lymphocytes acquire their distinctive characteristics while maturing in the thymus. They mature to express phenotypic markers or clusters of differentiation called CD markers. CD4+ cells are the helper T cells, and CD8+ cells are the cytotoxic suppressor T cells. The CD4+ helper T cells are regulators of the immune system. They help B cells develop into antibody-producing plasma cells. They also help to activate CD8+ cells, and they participate in delayed hypersensitivity reactions, which is important in limiting *Mycobacterium tuberculosis* infections. CD8+ lymphocytes kill virus-infected and tumor cells. The B lymphocytes mature in the bone marrow, where they acquire their characteristic markers that include CD20, CD21, and CD37. B cells have two important roles in the immune system. They differentiate into plasma cells that produce specific antibodies on encountering an antigen, or they become memory cells.

Humoral Immunity

Humoral immunity is mediated by antibodies, which are produced by mature B cells, called the plasma cell. Antibodies are globulin proteins (immunoglobulins) that react to antigen. There are five classes of antibodies: IgA, IgM, IgG, IgE, and IgD. Immunoglobulins are glycoproteins made from differing combinations of protein chains, connected together in various combinations, leading to the different classes. IgA is the antibody found in secretions such as colostrum (early breast milk), saliva, and tears. This antibody is also present in the respiratory, intestinal, and genital tracts, where it prevents the attachment of bacteria and viruses to mucous membranes. IgM is the immunoglobulin produced in the primary immune response, whereas IgG is the immunoglobulin found in both the primary and secondary immune responses, but in much greater quantities in the secondary response. IgG is the only immunoglobulin that crosses the placental barrier. IgG and IgM are the only two immunoglobulins that activate complement. IgE is important in protecting against certain parasitic infections, such as helminths or worms, and mediates the immediate hypersensitivity reaction. In other words, IgE is responsible for the immunoglobulin involved in the anaphylaxis reaction. Antibodies protect against infection by neutralizing toxins and viruses, and they **opsonize** or make microorganisms more easily phagocytized.

The humoral or antibody-mediated response is effective against agents that produce toxins, against bacteria that have polysaccharide capsules, and against some viral infections. The primary response occurs when the immune cells encounter a specific antigen for the first time. Reactions between antigen and antibody are highly specific. In the primary immune response, antibodies do not appear until 7 to 10 days after the initial encounter. The first antibodies to appear are IgM. When there is a second encounter with the same antigen, or a closely related antigen, the response is more rapid, and is called the secondary response. In this case, the lag time until the antibody is detected in the serum is only 3 to 5 days, and the

antibody titers are much higher than those seen in the primary response. This is because of the actions of memory B cells that were produced as a result of the first encounter as well as their quick ability to produce a large quantity of their "memorized" antibody. During the second encounter, IgM is made in the same quantity as in the primary response, but IgG is produced in larger quantities. With repeated encounters, the response is elicited faster and becomes more specific.

Infection with a related virus may impart immunity against other viruses in the same family that are not so benign. Viruses within one family sometimes contain similar structural proteins. If there are enough similarities, the immune system recognizes both as nonself and destroys them before allowing infection to take hold.

Cell-Mediated Immunity

Cell-mediated immunity (CMI) plays an important role in defending against intracellular infections, such as tuberculosis or gonorrhea; and viral infections, fungal infections, parasites, and tumors. The strongest evidence of its importance is illustrated in human immunodeficiency virus/acquired immunodeficiency syndrome when the destruction of cell-mediated immunity results in overwhelming infections and unusual tumors. An antibody is not involved in the reactions of CMI except in the antibody-dependent cellular cytotoxic reactions. Cells involved in CMI include the following:

- **CD4-helper T cells**—participate in antigen recognition and modify the response by activating CD8 cytotoxic T cells, B cells, and macrophages
- **CD8 cytotoxic T cells**—kill other infected or endogenous cells by producing perforins (proteins that cause holes to form in cell membranes, causing cell death as a result of loss of the osmotic gradient) or by inducing apoptosis (programmed cell death)
- **Macrophages**—ingest or destroy foreign cells, viruses, or debris (phagocytosis); present antigens (by activating T cells that can recognize foreign antigens); and produce cytokine (namely, interleukin 1 and tumor necrosis factor). The process of phagocytosis is carried out by macrophages, neutrophils, and monocytes. They engulf and destroy bacteria, foreign antigen, and cellular debris. These cells process the bacteria, breaking them down by specialized cell processes. Fragments of the cell, known as **epitopes** or antigen, are displayed by the phagocytic cell in close association with specialized proteins on the exterior of the cell. These proteins are called major histocompatibility complex. There are two general classes of major histocompatibility complex—class I and class II. Class I proteins process antigen from virus-infected cells and present the processed antigen as epitopes in association with major histocompatibility complex to cytotoxic T cells, whereas class II proteins process bacterial antigen to the helper T lymphocytes.
- **Natural killer cells**—large granular lymphocytes that do not express T-cell antigen receptor. They destroy tumor cells or virus-infected cells by recognizing that those cells are not expressing "self" markers. Although they are regulated by other proteins in the immune response, they do not require specific activation to produce perforin and granzymes that, when released on target cells, cause apoptosis or necrosis of the nonself cell.
- **Cytokines**—proteins produced by immune cells that modify the immune response. Chemokines are a type of cytokines that attract macrophages or neutrophils to the site of an infection.

Hypersensitivity Reactions

The immune response also can be harmful to the host when it is inappropriate or exaggerated. These types of harmful reactions are called **hypersensitivity** or allergic reactions, and they occur when individuals are exposed to a specific antigen. The first encounter sensitizes the person to the antigen and induces an

antibody response. The subsequent encounters with the same or closely related antigen elicit the allergic response. There are four major types of hypersensitivity reactions. Types I, II, and III are antibody-mediated responses, whereas type IV is a cell-mediated response.

Type I is an immediate hypersensitivity reaction involving IgE and an antigen. The term immediate hypersensitivity reflects the fact that these reactions occur within minutes of exposure to the antigen. There are two categories of type I reactions: (1) atopy or a localized reaction and (2) anaphylaxis, which is a systemic reaction. IgE is produced in excessive amounts and binds to mast cells or basophils that then become activated and release their contents. The degranulation of mast cells releases histamine, leukotrienes, and tryptase and causes the production of various cytokines. In turn, the cytokines produce various reactions, including the rapid contraction of smooth muscle, increased vascular permeability, hypotension, and changes in the coagulation pathway. Localized histamine release causes urticaria, whereas systemic histamine release causes dose-dependent hemodynamic and cardiovascular changes, such as an increased heart rate, hypotension, and shock, which can be life threatening. Tryptase is an enzyme found in mast cells that, when released, activates complement and the coagulation pathway and has the potential to cause angioedema, clotting, and clot lysis, leading to disseminated intravascular coagulation in severe cases. Common triggers for anaphylaxis include foods, drugs, antibiotics, and *Hymenoptera* venom. The treatments for anaphylaxis are aimed at counteracting the mediators released from the mast cells. The goal of treatment is early recognition and the use of epinephrine to prevent progression to life-threatening symptoms.

Anaphylaxis is a serious allergic reaction that has a rapid onset of symptoms, a variable clinical presentation, and, if unrecognized, can lead to respiratory arrest, circulatory collapse, and death. Previously stated, the goal of treatment is early recognition, use of epinephrine, and reduced reliance on less-effective medications such as antihistamines and glucocorticoids. Most patients with anaphylaxis—up to 90%—will have cutaneous involvement, including flushing, itching, urticaria, or angioedema. Upper airway involvement occurs in up to 70% of patients, with signs and symptoms such as changes in phonation, the sensation of throat closure or choking, cough, wheezing, and shortness of breath. Cardiovascular symptoms are less common, but treatment should not be withheld if signs of tachycardia or hypotension are absent.

Anaphylaxis is usually characterized by a defined exposure, followed by rapid onset of symptoms, and evolution and resolution of symptoms within minutes to hours. Variations on the typical time course have been reported and may complicate treatment. Biphasic anaphylaxis has been reported in up to 20% of reactions. In a biphasic reaction, the initial resolution of symptoms is followed by a reappearance of symptoms, and, if not anticipated, can be the cause of a life-threatening recurrence. In some rare cases, the reaction can last for many hours to days in those with protracted anaphylaxis.

When anaphylaxis is not recognized by the health care provider, it is left untreated and this places the patient at the highest risk for complications, including death. There are unique circumstances that exist that place patients at risk for unrecognized anaphylaxis. For example, persons with a known history of asthma may be treated for an acute asthma exacerbation without taking into account any cutaneous symptoms, placing them at greater risk for unrecognized anaphylaxis. Their wheezing may be assumed to result from asthma when it actually is secondary to the respiratory symptoms associated with anaphylaxis. Having asthma puts patients at a high risk of death from anaphylaxis simply because it is not recognized until much later in the presentation, delaying appropriate treatment. Patients with underlying neurologic impairment and psychiatric illnesses that interfere with cognition or impair judgment may not be able to express symptoms of pruritus or throat closure. Additionally, a sedated patient is unable to recognize or communicate the presence of early symptoms and is, therefore, dependent on the health care provider to recognize any subtle changes in the skin or vital signs that may signal that an allergic reaction is taking place.

Prompt assessment and treatment are critical in cases of anaphylaxis. The first step in its management begins with the removal of the inciting antigen. Intramuscular epinephrine should be injected, and, if symptoms persist, an epinephrine drip should be established. As with all successful resuscitations, begin with airway management. If there is any evidence of stridor, significant airway edema, or impending respiratory arrest, perform intubation immediately. Maintain breathing and support circulation with supplemental oxygen and volume resuscitation. Place two large-bore IV lines for rapid fluid administration, and place the patient on continuous cardiac/respiratory monitoring until the episode has resolved. All patients with anaphylaxis require fluid resuscitation. Those with hypotension or signs of shock should receive large-volume resuscitation. Adjunctive agents such as H_1 blockers—antihistamines—can be used for the treatment of itching or hives that fail to resolve after the administration of epinephrine. Patients who are wheezing can be treated with an inhaled beta-2 agonist if wheezing does not resolve after treatment with epinephrine. The use of glucocorticoids does not appear to be helpful in acute anaphylaxis but may offer some protection against the biphasic or protracted reaction.

All patients who are discharged from the hospital after anaphylaxis should be educated about their allergy and should have an emergency action plan for any recurrence, including a prescription for and instruction in the proper use of an epinephrine autoinjector.

Anaphylactoid reactions are IgE-independent systemic reactions. In these reactions, the same types of cells are activated—namely, the mast cells and basophils via a direct mechanism independent of IgE activation. Although the mechanism that triggers activation differs between anaphylaxis and anaphylactoid reactions, the symptoms, treatment, and risks for complications are the same for both; therefore, the distinction is not one of clinical significance.

Type II or cytotoxic hypersensitivity reactions are antigen-antibody reactions in which antibody directed against antigen on the surface of the host cell membrane causes activation of complement and subsequent lytic and osmotic damage to the host cells. The cytotoxic antibody-dependent cell-mediated cytotoxic reaction is included in this category. This type of reaction is seen in transfusion reactions, hemolytic disease of the newborn (erythroblastosis fetalis), and some autoimmune diseases. The antibodies involved in this type of reaction are IgM or IgG.

Type III hypersensitivity reaction involves the interaction of antigen and antibody. In those types of reactions, circulating antigen-antibody complexes deposit in tissue or on the endothelium of blood vessels and cause damage by activating complement, releasing chemotactic and clotting factors, and attracting other immune cells to the area. Examples of this type of reaction include the arthus reaction, which is a rare severe inflammatory response to intravascular precipitates of antigen-antibody complexes, serum sickness, polyarteritis nodosa, glomerulonephritis, and systemic lupus erythematosus.

The fourth and final type is the delayed-type hypersensitivity reaction, in which tissue damage results from excessive activation of macrophages. Type IV reactions involve the interaction of macrophage and T cells, but the response is delayed from hours to days after the antigen is encountered. This hypersensitivity reaction is cell mediated and is not a humeral response as are the first three. This type of reaction is exemplified in contact hypersensitivity as seen in exposure to poison ivy or poison oak.

Signs and Symptoms

Anaphylaxis (Type I Hypersensitivity Reaction)

Cutaneous involvement:

- Flushing
- Itching

- Urticaria
- Angioedema

Upper airway involvement:

- Changes in phonation
- Sensation of throat closure or choking
- Cough
- Wheezing
- Shortness of breath

Cardiovascular involvement:

- Tachycardia
- Hypotension

Transport Management

Anaphylaxis (Type I Hypersensitivity Reaction)

- Remove the antigen.
- Inject intramuscular epinephrine.
- If symptoms persist, establish an epinephrine drip.
- If there is any evidence of stridor, significant airway edema, or impending respiratory arrest, intubate the patient immediately.
- Provide oxygen supplementation and volume resuscitation.
- Place two large-bore IV lines for rapid fluid administration.
- Place the patient on continuous cardiac/respiratory monitoring.
- In case of hypotension or signs of shock, provide large-volume resuscitation.
- Give H_1 blockers for ongoing itching or hives.
- Give an inhaled beta-2 agonist for ongoing wheezing.
- In case of a biphasic or protracted reaction, give glucocorticoids.

Immunodeficiency

Immunodeficiency occurs when one or more of the components of the immune system is damaged or is nonfunctioning as a result of illness or lack of functioning immune cells, or when intentionally suppressed by various immunosuppressant therapies. The specific component of the immune system that is not fully functional predisposes the patient to certain types of infection or disease. Various diseases or conditions that induce immunodeficiency demonstrate which arm of the immune system is responsible for protecting us against specific infections. For example, patients with B-cell deficiencies have recurrent infections with pyogenic bacteria such as *S pneumoniae* and *H influenzae*. A deficiency in T cells allows recurrent infections with viruses, fungi, or protozoa. Critically ill patients have little reserve to fight infections as the result of a decrease in host defenses and loss of innate immunity. The features of chronic illness that decrease innate immunity include stress, malnutrition, invasive procedures, loss of physical barriers of skin and mucosal surfaces, and pathogenic bacterial overgrowth or bacterial growth in normally sterile tissue. Patients who are maintained on immunosuppressive therapy for the management of autoimmune diseases or to prevent rejection of a transplanted organ are at increased risk for a variety of infections and

cancers.

Management of the transplant patient is another important aspect of critical care transports. Common management of illnesses and injuries may be complicated by the side effects of medications or by the patient's immunosuppressed condition. Immunosuppressive agents, such as cyclosporine, tacrolimus, azathioprine, and prednisone, are routinely prescribed to transplant patients in an effort to prevent rejection of the donor tissue. These medications can have serious side effects, including hypertension, hyperglycemia, hyperkalemia, nephrotoxicity, and neurotoxicity, so it is important to discuss the care plan with a transplant team representative prior to initiating transport.

In addition to surgical complications or manifestations of acute rejection, prevention and detection of infection are important when transporting these patients. For example, the presence of a life-threatening infectious illness may not be apparent until the diagnostic evaluations have been completed. In addition, mild symptoms that may be overlooked in routine transport patients could indicate serious problems in transplant patients.

Pathogenicity

Normal Bacteria

The relationship between host and microbe is not always adversarial, and not all microbes are pathogenic. Most bacteria are either beneficial or neutral. The presence of normal bacteria (normal flora) in and on the body plays an important role in disease prevention because their presence prevents colonization by pathogenic species. Normal flora refers to colonies of nonpathogenic species that assist the immune system by preventing the overgrowth of pathogenic strains by using up available nutrients, maintaining a certain pH, and creating bac-teriocins (antibacterial toxins). A good example of this beneficial relationship is demonstrated in the normal microbiota of the adult vagina. Lactobacilli normally present in the vagina produce acid and keep the pH of the adult vagina between 3.5 and 4.5. This creates a hostile environment for *Candida albicans*, the causative agent of vaginal yeast infection. If the normal flora of the vagina are reduced in numbers by extended use of antibiotics or excessive douching, the pH increases and *C albicans* can dominate, leading to fungal vaginitis.

Opportunistic Bacteria

Opportunistic infections are diseases caused by normally non-pathogenic agents in patients with an abnormally functioning immune system. The normally harmless organisms that cause these infections are termed opportunistic bacteria. In patients who are **immunocompromised**, the normal balance between host and microbe is upset by a weakness in the immune system that can be caused by a genetic defect, specific medications, and/or chronic diseases. When the host defense system is compromised, microbes that are otherwise harmless may replicate without check. In fact, AIDS is defined by the unusual infections that human immunodeficiency virus (HIV)-infected patients with compromised immune systems contract. These rare and unusual opportunistic illnesses are called **indicator conditions** or AIDS-defining diseases (listed later in Table 19-5) because they are rarely, if ever, seen in persons with intact immunity. Many of the patients requiring critical care transport will fall into the category of immunocompromised patients. Thus, CCTPs need to be keenly aware that these very sick patients can be inadvertently infected. CCTPs must adhere scrupulously to proper technique, keeping their equipment sanitized or sterile and using universal precautions such as masks and gloves when appropriate. CCTPs must be aware that even a mild dermatitis on the hands of a healthy person or a few bacteria left on respiratory equipment can translate to a lethal infection for the immunocompromised patient.

Pathogenic Bacteria

Pathogens are simply defined as those microorganisms capable of causing disease. Pathogenic organisms cause disease because they possess unique factors that help them gain entry into the body, colonize and overcome host defenses for at least a period of time, produce toxins that cause cytopathic effects, or do mechanical damage to the body. Entry of pathogens into the body and colonization of tissue may be enhanced by the production of certain excreted enzymes (invasins) and glycoproteins (adhesins) produced by some pathogenic bacteria. Pathogens may evade immune system destruction by producing capsules, slime layers, or specialized cell walls; mounting preemptive strikes by the destruction of phagosomes; or preventing phagocytosis by infecting only epithelial tissue or walling cells off on colonization.

Virulence Factors

The degree of pathogenicity is called the **virulence** of an organism. Properties of microbes that influence their virulence (called virulence factors) include:

- Host and tissue specificity (the ability of the organism to gain entry into a host)
- Adherence to specific host cells
- Invasion of host tissue
- Evasion of host defenses
- Toxicity

Pathogenic organisms may possess one or more virulence factors. Virulence can be experimentally quantified by two measures: the infective dose of an organism (ID_{50}) and the lethal dose of an organism (LD_{50}). The ID_{50} measures the number of organisms required to infect a susceptible test animal. The LD_{50} measures the number of organisms required to kill 50% of the test animals inoculated with the microbe. The lower the ID_{50} or LD_{50}, the more virulent the organism. The infective dose of Shigella is about 10 to 100 organisms, whereas the infective dose of Salmonella is about one million organisms. This means that it takes a larger quantity of Salmonella to produce an infection than it does Shigella or, simply, Shigella is more virulent.

Host and Tissue Specificity

A potential pathogen must gain access into the body before it can cause an infection. The typical portals of entry into the body are through broken skin, hair follicles, or sweat glands; through the mucous membranes of the respiratory, digestive, and genitourinary tracts; or through the conjunctiva of the eye **Figure 19-2**.

Pathogenic bacteria typically infect specific tissues or systems in the body; however, some bacteria are capable of infecting multiple tissues and can cause very different diseases. For example, various *Staphylococcus* species can cause localized skin infections such as an abscess. It can also cause toxic shock syndrome when present in large numbers in sites such as the vagina or sinuses and can release the exotoxin TSST (toxic shock syndrome toxin) into the bloodstream. Other organisms are extremely site specific for portal of entry and infectivity. For example, *Salmonella typhi* causes no infection when inhaled, but does cause infection if it enters the digestive tract. Conversely, *S pneumoniae* does not cause disease if ingested but does cause pneumonia if inhaled.

Adherence

Entry of a pathogen into a host does not mean that disease will necessarily follow. Once inside the body, the pathogenic organism must be able to adhere to specific cells to begin colonization. Most bacteria adhere to cell surfaces using specialized structures that project out of the cell wall, called pili or fimbriae **Figure 19-3**. Gram-negative bacteria possess common pili that allow them to adhere to surfaces in the

gastrointestinal tract. Other bacteria produce unique pili that bind tissues specifically.

Most organisms can begin to colonize as soon as they have adhered to the host tissue. As these microbes reproduce, they continue to bind to the surface of the host cells and spread along the tissue surface as they increase in numbers. Some microbes also can form biofilms, which are accumulations of bacteria and other organisms embedded in a matrix of polysaccharide along the surface of tissues. These microcolonies tend to be highly resistant to antibiotics and to the host immune system.

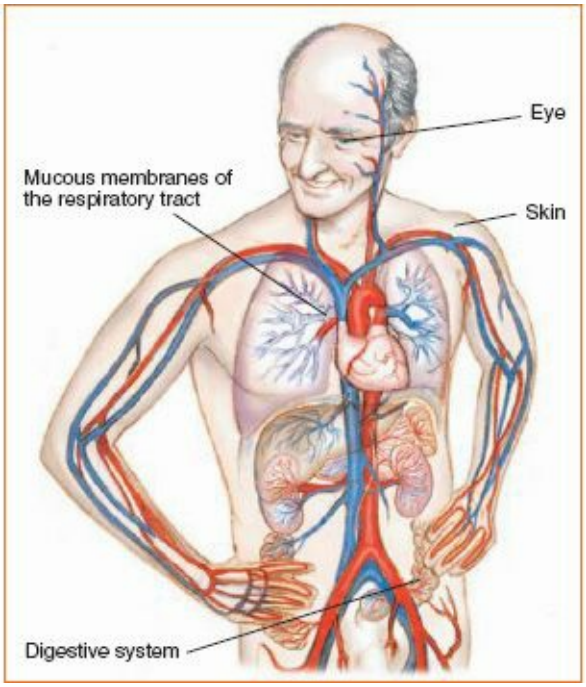

Figure 19-2 Portals of infection.

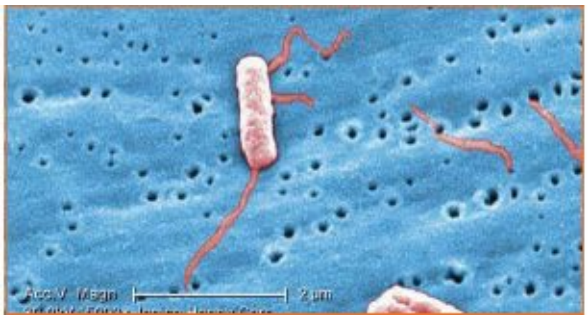

Figure 19-3 Pili (or fimbriae) on a bacteria.

Invasion

Once attached to tissue surfaces, some bacterial cells must then invade tissues in order to colonize. One mechanism for entry is the activation of receptor-mediated endocytosis. Because microbes are bound to tissue-specific receptor proteins, they trigger uptake or movement into the host cell by endocytosis. Once inside the host cell, the organisms multiply and easily invade neighboring cells through pores connecting the cells. This cell-to-cell infection also allows bacteria to evade destruction by the immune system. Other microbes invade tissues through the production of extracellular enzymes that break down the molecules that hold cells together.

Evasion

Colonization of tissues requires that microbes evade destruction by the host immune defense mechanisms. Different bacteria have developed various means to escape detection and destruction. Some organisms

produce polysaccharide layers and surround themselves with capsules or slime layers. These polysaccharide layers inactivate proteins essential for the immune system phagocytic mechanism, making ingestion by macrophages impossible. Other extracellular enzymes produced by bacteria also contribute to pathogenicity by protecting the organism from phagocytosis.

A number of bacteria are able to live and multiply within a phagocyte that protects them from antibody detection and destruction. Others avoid antibody recognition by mimicking host molecules and "trick" the immune system into ignoring them because they are disguised as "self" tissue.

Toxicity

Bacteria are capable of producing two types of toxins that are classified as exotoxins or endotoxins. Exotoxins are produced inside the cell and are released into surrounding tissues or fluids (blood or lymph). Endotoxins are part of the gram-negative cell wall and are not released until the bacteria are destroyed. The endotoxin component of the gram-negative cell wall is lipopolysaccharide (LPS). The bacterial cells release the LPS when they break apart. The free LPS causes fever, chills, and malaise; in extreme cases, it can lead to shock and death. All endotoxins elicit the same response from the host organism.

Exotoxins are generally specified by genetic information carried on plasmids (small pieces of DNA that are not part of the bacterial chromosome) or on bacteriophages (viruses capable of infecting bacterial cells). Exotoxins are highly specific and have differing modes of action. They are produced mostly by gram-positive bacteria but can be made by a few gramnegative organisms. Exotoxins are protein products that are soluble in the bloodstream and can be inactivated by heating. Exotoxins can be grouped according to the tissue type affected. There are neurotoxins that damage nervous tissue, enterotoxins that affect the tissues of the gastrointestinal tract, and cytotoxins that damage a variety of host tissues. Botulinum toxin is a potent neurotoxin that inhibits the release of acetylcholine and results in paralysis. Enterotoxins affect the digestive tract and disrupt the epithelial lining of the gut, leading to severe diarrhea such as that caused by *Vibrio cholerae*, the causative agent of cholera. Erythrogenic toxins specifically target capillaries and produce a red skin rash such as that seen in scarlet fever caused by *Streptococcus pyogenes*.

Transmission of Virulence Factors

The DNA of various bacteria encodes virulence factors and antibiotic resistance genes. Many virulence factors are carried on circular, extrachromosomal, self-replicating pieces of DNA called plasmids. Plasmid DNA is released into the environment upon lysis of the host bacterial cell. Other nearby bacterial cells can take up these plasmids and incorporate them into their own cells through the process of transformation. Transformation of bacteria can also take place by the uptake of fragments of the chromosomal DNA released upon lysis of a nearby cell. Once inside the recipient cell, the DNA may be either degraded by nucleases or integrated into the host chromosome, becoming part of that cell's genome. If the fragment of DNA integrated encodes a virulence factor, the newly transformed cell acquires that virulence factor and passes it on to ensuing generations as the cell divides. More frequently, plasmids carry antimicrobial resistance genes.

Bacterial cells are subject to infection by bacterial viruses called bacteriophages. Bacteriophages cause destruction of a host bacterial cell by growth and lysis of the host bacterium. Sometimes, however, the bacteriophage DNA integrates into the host chromosome (now called a prophage) and remains there as the bacteria undergo replication generation after generation. Any genetic information carried on the bacteriophage will be expressed as part of the bacterial protein complement, a process known as transduction. Sometimes bacteriophages carry genes for the production of virulence factors, such as capsules, exoenzymes, or toxins. At some point, usually when there is some environmental stress on the

host bacterial cell, the bacteriophage will excise from the bacterial chromosome and begin a lytic cycle, killing the host bacterium and releasing free bacteriophage into the environment. The free bacteriophage is then ready to infect another bacterial cell.

Viral Pathogenicity

Viruses, such as bacteria, must gain entry into the body to cause disease. Some viruses simply enter through broken skin, such as a puncture wound, an insect bite, or the entry port of a hypodermic needle. Others enter the body through the respiratory or digestive tract and bind to host cell surface receptors using viral proteins found on the surface of the virions. The binding of these protein "spikes" induces receptor-mediated endocytosis that brings the virus directly into the cell. Once inside, the virus particle can be "uncoated," releasing viral nucleic acid. The viral DNA or RNA begins to direct synthesis of viral-encoded protein and nucleic acid molecules, and the process of new virion assembly begins. The illness caused by the virus may be the result of the production of viral-specific molecules that deplete the host cell's resources and lead to eventual cell death. Other viruses cause illness because they induce programmed cell death or apoptosis, even when they do little harm to the cell during their active growth cycle. A third pathogenic mechanism is that some viruses induce large inflammatory responses, even with little or no actual damage to the host cell.

Epidemiology

Epidemiology is the study of how disease is distributed within populations and the factors that influence that distribution. The specific objectives of epidemiology are to identify the **etiology** (causes and risk factors that influence the occurrence of disease); to determine the extent of disease within a given community; to understand the natural history of disease; to evaluate the effectiveness of therapeutic methods; to identify sources of disease outbreaks; to identify modes of disease transmission; and to develop methods of disease prevention.

When discussing disease and the impact it has on the population, the incidence and prevalence of the disease must be quantified. The incidence of disease is the number of new cases of disease within a defined population over a defined period of time. The prevalence of a disease is defined as the number of cases of disease detected in a specified period of time, regardless of when the illness was contracted. Prevalence is expressed as the number of cases of disease found within a population at a specified time divided by the number of persons in that same population over the same specified time. Prevalence includes both old and new cases of disease. Incidence measures the risk of contracting the disease, whereas prevalence measures the burden of disease within a community. Both are used for understanding the patterns of disease occurrence and for utilization planning of health services, facilities, and the training of future providers.

The pattern of disease occurrence is another important element in understanding how populations are affected by disease. If a disease occurs only occasionally, the disease is called sporadic. If there are always a number of cases of a disease within a given population, and the numbers are expected and predictable, the disease is said to be endemic. If there is a clear rise in the number of cases (above a threshold level) of a disease for a given time or geographic area, the disease is said to be epidemic. Pandemic refers to a worldwide epidemic. The detection of emerging or reemerging infectious diseases is possible through the systematic collection of data and the constant surveillance of the incidence of disease. The Centers for Disease Control and Prevention (CDC) continually identifies certain "notifiable" diseases that require reporting to the health department in their effort to monitor and track the incidence of contagious diseases **Table 19-2**. New diseases can only be recognized as such when the normal background cases of infectious disease are well understood.

Disease is not randomly distributed, and the "where" of the disease outbreaks can be an important clue for identifying the cause or source of the disease. An example of the importance of the location of disease outbreaks was demonstrated in the severe acute respiratory syndrome (SARS) epidemic. Late in 2002, there was an outbreak of cases of life-threatening respiratory illnesses in the Guangdong Province of China with no identifiable cause. The first cases went unnoticed by international health organizations until February 2003, when a physician from Guangdong Province became ill while staying in a hotel in Hong Kong. During his visit to Hong Kong, 12 additional guests in the hotel contracted the illness, including seven who were staying on the same floor of the hotel as the physician. These hotel guests became the index patients, who then transported the disease to Singapore, Canada, Vietnam, Ireland, and the United States. Linking the cases together by location became an important component in the successful isolation and identification of the organism causing this mysterious disease, which was subsequently identified as a novel coronavirus.

TABLE 19-2 Infectious Disease Designated as Notifiable at the National Level

Acquired immunodeficiency syndrome	Hansen's disease (leprosy)	Novel influenza A virus (swine flu)	Streptococcal toxic shock syndrome
Anaplasma phagocytophilum	Hantavirus	Pertussis	*Streptococcus pneumoniae*, invasive and drug-resistant (children < 5 y)
Anthrax	Hemolytic uremic syndrome, postdiarrheal	Plague	Syphilis
Botulism	Hepatitis A, B, C	Poliomyelitis	Tetanus
Brucellosis	Human immunodeficiency virus, adult (≥ 13 y)	Psittacosis	Toxic shock syndrome (staphylococcal)
Chancroid	Human immunodeficiency virus, pediatric (< 13 y)	Q fever	Trichinellosis
Chlamydia	Influenza-associated mortality (children < 18 y)	Rabies	Tuberculosis
Cholera	Legionellosis	Rocky Mountain spotted fever	Tularemia
Coccidioidomycosis	Listeriosis	Rubella	Typhoid fever
Cryptosporidiosis	Lyme disease	Rubella, congenital syndrome	Vancomycin-intermediate and resistant *Staphylococcus aureus*
Cyclosporiasis	Malaria	Salmonellosis	Varicella (chickenpox)
Diphtheria	Measles	Severe acute respiratory syndrome	Vibriosis (noncholera)
Ehrlichia/ehrlichiosis	Meningococcal disease	Shiga toxin-producing *Escherichia coli*	Yellow fever
Encephalitis	Mumps	Shigellosis	
Giardiasis	Neurosyphilis	Smallpox	
Gonorrhea		*Streptococcus*, invasive group A	
Haemophilus influenzae, invasive disease			

Data Source: Nationally notifiable diseases and other conditions of public health importance event code list, Division of Integrated Surveillance Systems and Services, National Center for Public Health Informatics, Centers for Disease Control and Prevention. Published January 2009, pp 1–26. Available at: http://www.cdc.gov/ncphi/disss/nndss/PHS/files/NNDSS_event_code_list_January_2009_CLEARED.do Accessed June 29, 2009.

In infectious disease epidemiology, it is important to understand that there is a broad spectrum of disease severity. Often the prevalence or incidence of disease within a population only accounts for clinical illness or disease that produces symptoms. Disease without symptoms does not mean the same as no disease or no risk of spreading disease. Factors such as the incubation period of the disease or the presence of different carrier states may cause the disease to go undetected and unaccounted in an individual, but can place others at risk for contracting the disease because prevention and disease control efforts are not employed. This becomes a critical concept in the safe transport of patients. CCTPs are at risk for contracting disease from each patient transported; therefore, the use of disease control and prevention practices must be evaluated and employed as appropriate every time care is provided to patients regardless of whether the patient is or is not currently exhibiting symptoms.

Reservoirs of Infection

For a microorganism to infect and reinfect, it must have the ability to survive, duplicate or reproduce, and spread to new hosts. Reservoirs of infection provide a supply of nutrients and an environmental niche that enables long-term microbial survival. Bacteria have developed systems that enable them to withstand hostile environments. Spore-forming bacterial species are resistant to drying and environments with low nutrients. Encapsulated bacteria are resistant to changes in pH levels. Certain bacteria can make complex molecules from more basic components if certain nutrients are not available in their immediate environment. These adaptations allow organisms to survive in soil and water for significant lengths of time.

Both wild and domestic animals can also act as reservoirs of infection. Diseases that occur in animals and can also be transmitted to humans are called zoonoses. Influenza is the prototypic zoonosis. In this case, the virus transmission between the animal reservoir and the human host provides selective pressure to the virus that enables its long-term survival. The influenza virus will alter its surface proteins and can then reinfect and cause illness in individuals who had developed immunity to the old strain. The new strain is just different enough to escape activation of the immune system.

In the same way that animals can serve as reservoirs of infection, humans can act as reservoirs of infection. Persons who are colonized by pathogenic species but do not have symptoms of the infection are called asymptomatic or latent carriers. They have important roles in perpetuating diseases such as diphtheria, AIDS, hepatitis, gonorrhea, and amoebic dysentery.

Patients in whom pathogenic bacteria are colonized can serve as sources of infection during extended hospital admissions. These organisms coexist with normal skin or intestinal flora and are selected by the continual use of antimicrobial agents in the inpatient setting, meaning their numbers increase over the normal flora. Selection for increasing antibiotic-resistant bacteria happens when antibiotics are present at detectable levels in a particular environment over a period of time, as seen at inpatient settings. Bacteria that are sensitive to the antibiotics are killed easily in the presence of the antibiotic. Those bacteria that are resistant to antibiotics can survive and, therefore, “outcompete” over those that are sensitive, thus increasing antibiotic-resistant strains throughout patient treatment facilities. Health care providers unwittingly act as a major source of cross-infection when they interact with different patients without proper intervening antisepsis.

Finally, the health care environment itself may serve as a reservoir of infection. Some organisms are remarkably resistant to the effects of drying and, therefore, can survive on inanimate objects for an extended period of time. Durable medical equipment, even when treated with antimicrobial solutions, can harbor bacteria several days to several weeks after inoculation.

Modes of Transmission

Understanding the different modes of transmission of microorganisms is the first step in developing effective infection prevention measures. The unique properties and characteristics of the infectious agents determine how the disease is spread and how the infection is contracted. Infectious disease can be transmitted directly, indirectly, or mechanically.

Direct contact is person-to-person transmission (ie, no intermediate carrier is involved in the transfer). Direct transmission can occur vertically or horizontally. Horizontal transmission is the transmission of infection between members of the same generation (same generation refers to bacteria that are living at the same time, as opposed to vertical transmission, which is genetic information passed on to progeny). Types of direct horizontal transmission include sexual transmission, exchange of respiratory droplets or secretions, and transmission by contact. To guard against direct transmission, CCTPs should use protective barriers such as gloves, gowns, masks, and eye shields, which prevent direct contact with

infectious materials during invasive procedures or when providing care for an infectious patient.

Vertical transmission is the exchange of an infectious agent between mother and fetus *in utero*. Perinatal transmission is another form of direct transmission in which the exchange of the infectious agent from the mother to the baby occurs during the birthing process. It has been shown that the risk of vertical transmission of HIV can be greatly reduced by initiating therapy with zidovudine, a nucleoside reverse transcriptase inhibitor, between 14 and 34 weeks of gestation.

Indirect transmission occurs when there is an intervening step in the transmission from reservoir to susceptible host. The intermediate step involves an inanimate object or fomite such as towels, bedding, thermometers, and contaminated syringes. Prevention of infection can be as simple as properly disposing contaminated materials and cleaning equipment between patients.

Many infectious diseases are transmitted through the use of a medium, such as water, food, air, or contaminated body fluids. Water can be either the reservoir of disease or a vehicle for the transmission of that disease. Water that contains raw or poorly treated sewage is the perfect vehicle for the **fecal-oral route** of cholera transmission. Vehicle transmission can be prevented by good hygiene and proper food preparation and storage.

Vector transmission is the exchange of a pathogen from an infected organism to a susceptible host via an animal or insect. The method of vector transmission can be either mechanical or biological. Mechanical transmission is the passive transport of pathogens on the body of the animal to the susceptible host. Typhoid is an example of an infectious agent that is transmitted on the feet of houseflies. Biological transmission involves the vector, usually an arthropod, ingesting a blood meal from an infected organism and transmitting it after the pathogen has multiplied in the digestive tract of the vector, increasing the likelihood of transmission to a new host.

Selected Diseases

CCTPs are exposed to deadly agents on a daily basis. When providing care for sick patients, the risk of contracting their diseases is inherent, as well as the risk of passing the diseases onto other patients, fellow providers, or even family members. Knowledge of common infectious diseases and the appropriate responses can prevent the spread of disease.

Meningitis

Meningitis is the inflammation of the leptomeninges, the membranes that cover and enclose the spinal cord and brain. The meninges are composed of three distinct layers—the dura, arachnoid, and pia maters—that are the external, middle, and internal layers, respectively. Meningitis is a serious medical condition caused by bacterial, viral, or fungal infectious agents. Bacterial meningitis is a life-threatening illness that has an acute onset and is considered a medical emergency that requires prompt diagnosis and treatment to preserve life and neurologic function. Bacterial meningitis is fatal in up to 30% of patients and causes permanent neurologic impairment in 10% of patients who survive the illness.

Bacterial meningitis is caused by a bacterial infection that migrates to the meninges. Viral meningitis exhibits the same symptoms as bacterial meningitis and is also considered a serious medical condition; however, it typically resolves within 7 to 10 days. Because it is difficult to distinguish between bacterial and viral meningitis with the onset of symptoms, all cases should be treated as bacterial in origin until ruled out. The infectious agents may originate from structures close to the brain, such as the middle ear, sinuses, or respiratory tract, which can be colonized by pathogenic species. Bacterial meningitis can occur after trauma to the cranium in which the normal protective barriers to infection have been breached by penetrating objects or fractures, and it can occur by hematogenous spread of septicemia.

Although the classic triad of symptoms observed in patients with bacterial meningitis includes fever,

nuchal rigidity, and altered mental status, these symptoms are seen in less than 50% of patients. Other symptoms of meningitis include headache, significant photophobia, and cutaneous manifestations such as petechiae or palpable purpura. Most patients have fever (up to 95% of patients at presentation) of usually greater than 100.4°F (38°C), and virtually all patients with meningitis have at least one of the classic triad of symptoms. Additionally, neurologic complications may be present early in the disease course or can occur as a late complication. Seizures, papilledema (swelling of the optic nerve), and neurologic deficits have been described in up to 30% of patients. Hearing loss is a late neurologic complication. Treatment with dexamethasone, given at the same time as the first dose of antibiotics, may reduce this late neurologic complication, especially if the bacterial culprit is pneumococcal.

Patients may not specifically complain of a stiff neck, but nuchal rigidity can be demonstrated clinically by a few simple maneuvers. If the patient is unable to touch the chin to the chest by either active or passive flexion, one must suspect nuchal rigidity. This is classically demonstrated using the Brudzinski and Kernig signs, both of which demonstrated an extremely low sensitivity rating when tested in a large well-designed prospective study in 2002.

The Brudzinski sign is positive when there is spontaneous involuntary flexion of the hips during attempted passive flexion of the neck. The Kernig sign is elicited by straightening a patient's flexed legs. The inability to allow full extension of the knees when the hips are flexed is interpreted as a positive result.

When meningitis is suspected, the patient should undergo a lumbar puncture to obtain cerebrospinal fluid (CSF) unless contraindicated. If the collection of CSF is delayed, blood culture results are often positive and can be useful for diagnosis in the event that CSF cannot be obtained prior to the administration of antibiotics. Some physicians will delay the lumbar puncture until a computed tomographic (CT) scan has been obtained to exclude the possibility of increased intracranial pressure that may lead to cerebral herniation during the removal of CSF. It is not normally necessary to obtain a CT scan prior to a lumbar puncture except in the following circumstances:

- History of immunosuppression (a patient with HIV or a patient who has received a transplant)
- Seizure (new onset, within 7 days of presentation)
- History of central nervous system disease (tumor, stroke, or focal infection)
- Abnormal level of consciousness
- Focal neurologic deficit
- Papilledema

If for any reason the collection of CSF is delayed, the administration of antibiotics should not be withheld. Empirical antibiotic treatment is warranted when bacterial meningitis is suspected clinically.

Clinicians use epidemiologic knowledge to predict the infecting organism by understanding which bacterium (or virus) is the most likely pathogen, given the patient's age, medical history, immune status, and presenting symptoms. The organisms that cause community-acquired bacterial meningitis are quite different from those causing meningitis in the hospital or in immunosuppressed patients. The major causes of community-acquired bacterial meningitis in adults are *S pneumoniae* and *Neisseria meningitides*, and *Listeria monocytogenes* in patients older than 55 years.

Signs and Symptoms

Viral Meningitis

- Fever

- Severe headache
- Nuchal rigidity
- Nausea
- Vomiting
- Photophobia
- Malaise

The epidemiology of bacterial meningitis underwent a major change after the introduction of the *Haemophilus influenzae* type b conjugate vaccine in 1987. Before the 1990s, most cases of meningitis were seen in infants and children, and caused by *H influenzae*, a gram-negative bacterium. Currently, most of the cases of bacterial meningitis are observed in adults and can be caused by a variety of different bacterial species, including *N meningitidis, S pneumoniae*, and *H influenzae*.

H influenzae type b (Hib) is a small, pleomorphic, encapsulated, gram-negative coccobacillus that can colonize the respiratory tract of humans. Individuals colonized with *H influenzae* may be asymptomatic, and most isolates of *H influenzae* are nonencapsulated and are not pathogenic. *H influenza* type b is the most virulent, and it is encapsulated, which enables it to set up infection by evading destruction by phagocytic cells. It spreads from one individual to another via airborne droplets or direct contact with contaminated secretions. *H influenzae* is most commonly found in the nasopharynx of healthy asymptomatic children aged 2 to 5 years. When *H influenzae* meningitis is encountered in the adult population, it is suggestive of the presence of a predisposing medical condition such as paranasal sinusitis, otitis media, alcoholism, head trauma, asplenia, and other immunocompromised states. For this reason, the *H influenzae* vaccine is recommended for children and susceptible adults.

Signs and Symptoms

Bacterial Meningitis

Classic triad of symptoms:
- Fever
- Nuchal rigidity
- Altered mental status Possible other symptoms:
- Headache
- Photophobia
- Focal neural deficits
- Seizures

N meningitidis meningitis:
- Fever
- Headache
- Meningismus
- Petechial lesions (1- to 2-mm purple hemorrhagic spots that do not blanch with pressure), progressing to palpable purpura (larger areas of bleeding under the skin)

Differential Diagnosis

Bacterial Meningitis

- Viral meningitis
- Brain tumor
- Subarachnoid hemorrhage
- Herpes simplex encephalitis
- Febrile seizure
- Delirium tremens
- Hepatic encephalopathy
- Brain abscess

Transport Management

Bacterial Meningitis

- Administer IV fluids.
- Administer corticosteroids (such as dexamethasone) if indicated per protocols.
- Continue IV administration of antibiotics or other medications that have been started by the sending facility.

N meningitidis is a gram-negative diplococcus that causes 4,000 cases of meningitis each year in the United States. *N meningitidis* is pathogenic only in humans and is spread by prolonged close contact with infected individuals through direct contact with respiratory secretions. This is why outbreaks most often occur in prisons, military barracks, or college dormitories. The bacteria can be isolated from 10% to 30% of healthy adults, but the disease is not common. When a case of *N meningitidis* is reported, all close contacts of the infected individual are treated prophylactically.

N meningitidis enters the body through the epithelial surfaces of the nasopharynx. If the bacteria reach the bloodstream, they can cause gram-negative sepsis, extensive tissue damage, and death. The onset of symptoms is abrupt (ie, fever, headache, and meningismus). Patients with *N meningitidis* meningitis have a characteristic rash caused by meningococcal emboli that start as **petechial lesions** (small 1- to 2-mm purple hemorrhagic spots that do not blanch with pressure) and advance to **palpable purpura** (larger areas of bleeding under the skin) within a short period of time. This rash is classic and should not be missed because the mortality rate for untreated *N meningitidis* meningitis nears 100%.

S pneumoniae is the most common cause of community-acquired bacterial meningitis in adults. *S pneumoniae* are encapsulated, gram-positive cocci that are lancet shaped and are commonly arranged in pairs or short chains. They possess several pathogenic properties that make infection with *S pneumoniae* a serious medical event. Their polysaccharide capsule prevents them from being opsonized and phagocytized. They are able to adhere to the surface of epithelial cells that allows them to colonize, and they produce toxins that directly inhibit the body's cell-mediated phagocytic activity. They also possess specialized enzymes called neuraminidases and proteases, which allow them to grow in mucosal surfaces, facilitating colonization.

S pneumoniae is commonly isolated from the nasopharynx of healthy individuals. The rate of colonization is estimated to be as high as 70% in the winter and spring. Patients with chronic bronchitis are frequently colonized by *S pneumoniae*.

The development of *S pneumoniae* meningitis is often preceded by a mild upper respiratory tract

infection. Individuals at highest risk for contracting *S pneumoniae* include those with sickle cell anemia, chronic alcoholism, multiple myeloma, and general disability. The diagnosis is certain when the characteristic lancet-shaped gram-negative diplococci are isolated from the normally sterile cerebrospinal fluid. The mortality rate of *S pneumoniae* meningitis in the elderly and immunocompromised is quite high, even when treatment is initiated promptly. Therefore, vaccination against *S pneumoniae* is strongly encouraged for at-risk populations, and is now included in the routine childhood vaccination schedule.

Like bacterial meningitis, viral meningitis typically presents with fever, severe headache, and nuchal rigidity. Additional symptoms include nausea, vomiting, photophobia, and malaise. The severity of the symptoms and the disease is less serious than that of bacterial meningitis. Complete recovery can occur in 2 to 7 days without medical intervention.

There are 10,000 cases of viral meningitis reported each year in the United States. All age groups are affected, but viral meningitis is more commonly seen in young adults. Viral meningitis can be acute, subacute, or relapsing. The peak incidence occurs in summer.

There are a variety of species that can cause viral meningitis, including coxsackie A and B, echovirus, adenovirus, lymphocytic choriomeningitis virus, cytomegalovirus, poliovirus, and Epstein-Barr virus. Enterovirus is the most common cause of aseptic meningitis. All enterovirus species are transmitted through the fecal-oral route. Generally when providing care for a patient suspected to have viral meningitis, careful handwashing should be employed to prevent the spread of the disease.

Respiratory Syncytial Virus

Respiratory syncytial virus (RSV) causes seasonal outbreaks of acute respiratory infections. RSV is caused by an RNA paramyxovirus. The virus is called a syncytial virus because the proteins on the virus surface are not hemagglutinins, but fusion proteins that cause cells to fuse and form multinucleated giant cells. The clinical manifestations of RSV are dependent on the age and health of the individual infected. RSV season in the United States occurs in the winter months, with the peak of the season in January and February. RSV accounts for more than 100,000 hospitalizations of children in the United States each year. For infants 1 year or younger, the virus usually causes a lower respiratory tract infection, either bronchiolitis or pneumonia. Apnea is a risk for 20% of all infants hospitalized with the infection and may put the child at risk for sudden death.

RSV can occur in individuals older than 1 year. In fact, most children test positive for RSV by the time they reach the age of 2 years. Persons at risk for severe infections or complications from infections are those with a history of prematurity or those who are immunocompromised. RSV can also be detected in adults, but it usually does not cause long-term pulmonary dysfunction. Currently, no vaccine exists to prevent respiratory tract infections by RSV. A monoclonal antibody called palivizumab is available, and it confers passive immunization to recipients. It can be used for **prophylaxis** in premature infants at risk for severe infections.

Transmission occurs via respiratory droplets and via direct contact with hands that have been contaminated with mucous from the nose or mouth of an infected individual. CCTPs should employ droplet and contact precautions when transporting patients with a suspected or a confirmed case of RSV. Diagnosis is made by testing the respiratory secretions of the infected individual by polymerase chain reaction.

Signs and Symptoms

Respiratory Syncytial Virus

- Runny nose
- Congestion
- Cough
- Sore throat
- Headache
- Malaise
- In infants and young children: listlessness, decreased appetite, fever, wheezing, tachypnea, and cyanosis

Differential Diagnosis

Respiratory Syncytial Virus

- Asthma
- Bronchiolitis
- Pneumonia
- Foreign body aspiration
- Pertussis

Transport Management

Respiratory Syncytial Virus

- Provide supportive care.
- Administer fluids.
- Administer oxygen.
- Administer a nebulized bronchodilator for wheezing, if indicated by protocols.
- Monitor mechanical ventilator, if applicable.

Necrotizing Fasciitis

Necrotizing fasciitis is a limb-threatening or life-threatening infection of the soft tissue that affects the subcutaneous tissues, fat, and fascia but usually spares the skin and the muscle. Prompt diagnosis and treatment with antibiotics and complete surgical débridement are important to reduce the high mortality rate associated with this disease.

There are two main types of necrotizing fasciitis. Type I is polymicrobial, meaning the infection is the result of a mixture of aerobic and anaerobic microorganisms. This type is commonly seen in postoperative patients with comorbid conditions such as diabetes and peripheral vascular disease. The microorganisms seen in type I necrotizing fasciitis may be a mixture of *Staphylococcus aureus, Streptococcus* species, *Enterococcus* species, *Escherichia coli, Peptostreptococcus* species, *Bacteroides* species, *Vibrio vulnificus*, and *Clostridium* species. Type II is a monomicrobial infection caused by the group A streptococci (GAS) *Streptococcus pyogenes*. This infection was previously known as "streptococcal gangrene." The skin barrier is breached during surgery and is thought to be the portal of entry for the GAS infection.

Patients may present with unexplained pain that increases over time. Initially, the skin may appear

normal or there may be mild erythema. Pain out of proportion to the exam may be the only initial clue that there is a serious problem. Within 24 to 48 hours, the erythema worsens and develops into a dark red to purple color. Blisters or bullae may develop on otherwise normal appearing skin. Fever or tachycardia may also be present. Hypotension may develop as the infection worsens. Gas, as evidenced by crepitus on exam, may develop under the skin. Often this gas can be detected on radiographic imaging as air along the fascial planes.

Aggressive fluid replacement should be initiated in the field. Treatment using antibiotics alone, without surgical intervention, is associated with a nearly 100% mortality rate. Time is of the essence when dealing with a patient with necrotizing fasciitis. Any delay in surgical diagnosis and treatment may increase mortality and cause extension of the disease. Sometimes hyperbaric oxygen therapy is used in the treatment of patients with necrotizing fasciitis. CCTPs may be called on to transport patients to facilities with the resources to provide care for these patients. In any case of suspected or known necrotizing fasciitis, contact precautions must be maintained at all times, and all equipment must be thoroughly disinfected after the transport of this type of patient.

Signs and Symptoms

Necrotizing Fasciitis

- Unexplained pain that increases over time and is out of proportion to the exam
- Mild erythema that develops into a dark red to purple color within 24 to 48 hours
- Blisters or bullae
- Fever or tachycardia
- Hypotension
- Gas, as evidenced by crepitus on exam

Differential Diagnosis

Necrotizing Fasciitis

- Cellulitis
- Gas gangrene
- Toxic shock syndrome

Transport Management

Necrotizing Fasciitis

- Provide supportive care.
- Provide aggressive fluid replacement.
- Continue IV administration of antibiotics that have been started by the sending facility.
- Perform cardiac monitoring.
- Monitor urine output to the indwelling catheter, if one has been placed at the sending facility.
- Provide immediate transport (to be followed by surgical intervention).

Epiglottitis

Epiglottitis is caused from an infected and inflammed epiglottis, aryepiglottic folds, and/or surrounding tissues. In the past, most cases were caused by infection with Hib. Since the introduction of the Hib vaccine in 1991 in the United States, the epidemiology of this disease and others caused by Hib has been changing. The incidence of epiglottitis has been drastically reduced, but because of its severity and rapid progression to airway obstruction, it is important to be familiar with its clinical presentation.

Epiglottitis can occur at any age; however, most cases occur in children aged 1 through 5 years. The agent and median age at presentation are likely to shift as the population continues to be immunized against Hib. Other causes of epiglottitis include *Haemophilus parainfluenzae*, *S pneumoniae*, *S aureus*, and beta-hemolytic *Streptococcus pyogenes*. *H influenzae* type b epiglottitis still may occur, but will likely occur in children who are incompletely immunized or those who are noncompliant with vaccinations. There is no predominate bacterial species found in adults with epiglottitis.

An abrupt onset of symptoms is the common theme for presentation. The symptoms include high fever, severe sore throat, dysphasia, and dysphonia. Often, a child will start drooling as a result of painful or difficult swallowing. The child's speech or voice may be altered so that it is muffled, and has been described classically as the "hot potato voice." As the symptoms progress, the child may assume a distinctive posture: while sitting, the arms extend backwards to support the forward leaning trunk and the chin will be thrust forward to maximize the diameter of the hypopharynx. Unlike croup, stridor is not a predominant sign and increased respiratory effort with use of accessory muscles may not be present, thus the severity of the disease may be underestimated. The symptoms can rapidly progress to airway obstruction, hypoxia, and cardiopulmonary arrest.

As in all resuscitations, diagnostic testing should be deferred for patients in extremis until stabilization is achieved. The diagnosis of epiglottitis can be made by visualization of a fiery-red and edematous epiglottis. The optimal method of visualization is under controlled conditions via direct laryngoscopy by an otolaryngologist. Radiographs are not usually necessary to make the diagnosis of epiglottitis; however, if direct visualization cannot be achieved and doubt remains as to the diagnosis, a lateral soft-tissue neck radiograph may aid in diagnosis. With imaging, a healthy child's epiglottis appears as a thin tall structure, but when epiglottitis is present, the image may reveal the classic "thumb print" sign (ie, the epiglottis appears thick and squatty like a thumb). If a radiograph is obtained, the patient should not go to the radiology department unaccompanied. There should always be an advanced airway cart available, and a person trained in advanced airway management should accompany the patient if the patient leaves the emergency department prior to the procurement of a secure airway. Epiglottitis is a potentially life-threatening infection; as such, patients with epiglottitis must be promptly treated with empirical antibiotics. The antibiotic regimen should be selected to provide coverage for streptococci, pneumococci, staphylococci, and Hib.

CCTPs must maintain a high level of suspicion for epiglottitis for any patient presenting with the abrupt onset of fever, sore throat, muffled voice, and drooling. Agitation of the patient should be minimized because this may lead to worsening of their condition and hasten the progression to complete airway obstruction. Interactions with the child should be conducted in a manner that minimizes his or her anxiety because agitation only makes the situation worse. If airway intervention is necessary in the prehospital environment, the task should be given to the most experienced team member. Orotracheal intubation can be difficult secondary to edema and anatomical distortion. Patients with epiglottitis often require advanced airway management, such as nasotracheal intubation or tra-cheostomy. Getting to the nearest, most appropriate level care facility as quickly as possible is a must.

Signs and Symptoms

Epiglottitis

- High fever
- Severe sore throat
- Dysphasia
- Dysphonia
- Drooling as a result of painful or difficult swallowing
- Alteration of speech or voice so that it is muffled–"hot potato voice"
- Sitting with the arms extended backward to support the forward-leaning trunk, with the chin thrust forward to maximize the diameter of the hypopharynx

Differential Diagnosis

Epiglottitis

- Anaphylaxis
- Croup
- Foreign body aspiration
- Laryngotracheobronchitis
- Mononucleos
- Pertussis
- Pharyngitis
- Pneumonia
- Caustic ingestion

Transport Management

Epiglottitis

- Support the airway.
- Continue IV administration of antibiotics if they have been started by the sending facility.
- Note: When epiglottitis is strongly suspected, transfer of a patient without an artificial airway (endotracheal tube or tracheostomy) is unsafe.

Tuberculosis

Tuberculosis (TB) is caused by the acid-fast bacterium called *Mycobacterium tuberculosis*. The only other genus of pathogenic bacteria that is classified as acid-fast is *Nocardia*. Acid fast refers to the staining process that differentiates these organisms from most other bacteria. Because of the high lipid content (specifically mycolic acid) in the mycobacterial cell membrane, they can be diagnosed by the acid-fast staining technique **Figure 19-4**.

The genus *Mycobacterium* includes other ominous organisms such as the organism that causes leprosy, *Mycobacterium leprae*, and *Mycobacterium avium-intracellulare* complex (called MAC), which causes a tuberculosis-like pneumonia that is usually seen only in immunocompromised patients such as those with advanced HIV/AIDS. MAC is particularly insidious because these organisms are very

resistant to antibiotics and are becoming even more resistant at an alarming rate.

Tuberculosis causes more deaths globally than any other single microbial agent, killing 3 million people per year. In the United States, the combination of deteriorating public health availability, inadequate control of infectious diseases, urban crowding, immigration, and increasing numbers of HIV patients has resulted in a resurgence of TB cases. The risk of this highly infectious disease is increased in persons who live in poor housing conditions, those who are usually malnourished, and those who are co-infected with HIV. With recognition and appropriate treatments, the United States saw approximately 16,377 documented cases in 2000, down from the peak of 67,000 cases reported in 1992. The United States continues to steadily decrease the case rate of TB through education and quick actions taken to control the spread of the disease.

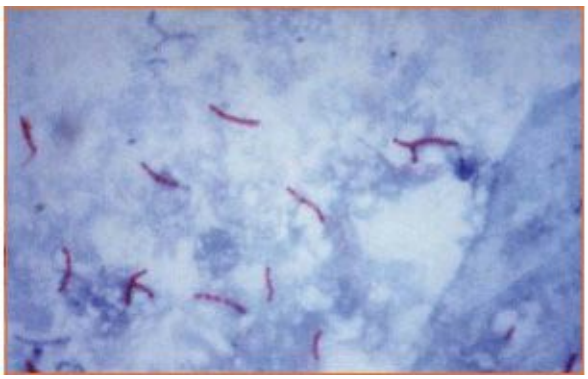

Figure 19-4 Acid-fast staining technique to diagnose tuberculosis.

M tuberculosis is transmitted via respiratory droplets, from the cough of an infected person to the respiratory epithelium of a susceptible person. The first site of infection is the lungs, and the bacterium, being an obligate aerobe, has a propensity to set up infection in the upper lobes of the lungs because that tissue is highly oxygenated. TB can also spread to the kidneys or spine. Most persons infected with *M tuberculosis* are asymptomatic. The immune status of the patient, in large part, determines the clinical symptoms of an infection with *M tuberculosis*. Persons with full-on pulmonary TB have five common symptoms: cough, fever, weight loss, night sweats, and fatigue.

M tuberculosis does not produce any toxins. *Mycobacteria* survive inside the phagosome of the reticuloendothelial cells (specifically, the macrophage). They avoid the normal phagocytic processes of the macrophage by producing a specialized protein that prevents the fusion of the phagosome with the lysosome. The two main types of lesions that *M tuberculosis* produces are exudative and granulomatous, depending on the immune response of the host. The Ghon complex is the pathognomonic lesion seen in the lung. It consists of an exudative lesion and an enlarged lymph node that drains the lesion. As discussed, the primary infection, which frequently goes undiagnosed, is often found in the lower lobes while the lesions of reactivation are found in the upper lobes. Reactivation of a previously contained infection occurs in the immunocompromised or debilitated patient.

After a person recovers from a primary infection, the immune system resists secondary infection and reactivation through cell-mediated immunity. Antibodies to tuberculin are formed and their existence is exploited in the purified protein derivative (PPD) test. The tuberculin antigen is injected into the skin; if positive, it indicates a previous infection, but does not necessarily indicate active disease. If the reaction is large (> 15 mm in diameter), then the person is considered to be actively infected and should receive treatment. As with some other infectious diseases, there is a delay between the primary infection and a positive PPD test result. In this case, it takes 4 to 6 weeks for the TB skin test to be reactive. The induration seen in the PPD is a type IV delayed hypersensitivity reaction, used for diagnostic advantage.

A vaccine is available for the prevention of TB. The BCG vaccine is made from a related bacterium,

Mycobacterium bovis, which usually infects bovines (cows). The BCG vaccine is not used in the United States because it is only effective 70% of the time and its use renders the PPD test useless. Inoculation with the BCG vaccine causes a person to have a positive PPD test result. If a vaccination campaign were to be initiated, the PPD test result of many persons would become positive and would result in loss of the ability to screen for exposure, without the assurance of induced immunity.

Infection with *M tuberculosis* can be difficult to diagnose because it is a slow-growing bacterium, taking weeks to culture. Because treatment must begin as soon as possible, diagnosis is based on clinical signs and confirmed via positive acid-fast staining of three sputum samples from patients suspected of being infected. The emergence of drug-resistant strains of TB has precipitated the use of a multidrug regimen for cure. The individual drugs used may vary slightly according to known sensitivities, but the mainstay of therapy is isoniazid. The drugs are typically given for 18 to 24 months.

Multidrug-resistant tuberculosis (MDR-TB) is TB that is resistant to at least two of the standard anti-TB medications, isoniazid and rifampin. Extensively drug-resistant TB (XDR-TB) is TB that is resistant to isoniazid, rifampin, any fluoroquinolone, and at least one second-line IV drug. Because XDR-TB is resistant to many first- and second-line drugs, treatment options are limited for patients with this form of the disease. Both MDR-TB and XDR-TB are contracted via the same mechanism as TB, through the inhalation of infected droplets from a person who is already infected when that person coughs, sneezes, or spits into the environment. These infected droplets can exist in the air for several hours and anyone who breathes in these particles is at risk for contracting the disease. The spread of MDR-TB is promoted by noncompliance with the full course of prescribed treatment for TB infection because the surviving bacteria develop resistance to the standard antibiotics.

When transporting a patient with a history of fever, weight loss, and night sweats, and anyone with a cough, take proper precaution by placing a mask on the patient (if tolerated); the CCTP should also wear a mask. CCTPs should comply with yearly PPD testing to determine a personal exposure history. If care was provided for a patient known to have TB and exposure was a possibility, providers should undergo PPD testing 6 weeks after exposure to assess his or her status. If the test result is negative, resume routine PPD tests. If a PPD test result is positive, a chest radiograph will be required to determine whether signs of active or previous TB infection exist.

Signs and Symptoms

Tuberculosis

- Cough
- Fever
- Weight loss
- Night sweats
- Fatigue

Differential Diagnosis

Tuberculosis

- Brucellosis
- Chronic cough
- Chronic obstructive pulmonary disease (COPD)

- Histoplasmosis
- Leishmaniasis
- Lung abscess
- Lung cancer
- Mesothelioma
- Myelofibrosis
- *Pneumocystic carinii* pneumonia
- Q fever
- Toxoplasmosis
- Typhoid fever

Transport Management

Tuberculosis

- Provide supportive care.
- Continue administration of IV antibiotics if they have been started by the sending facility.
- Ensure meticulous isolation precautions.

Pneumonia

Pneumonia is the number one cause of death as a result of infectious diseases and is the sixth leading cause of death in the United States for all causes including cancer and heart disease. In the United States, most patients who die of pneumonia are older than 65 years. Pneumonia is an infection of the lung parenchyma that is caused by bacterial, viral, or fungal agents. Physicians normally classify pneumonia as either community acquired or nosocomially acquired (contracted in a medical facility) because the differential acquisition of pneumonia has implications for the most likely infecting agent, treatment, and prognosis. The mortality rate for community-acquired pneumonia ranges from 5% to 14%, whereas the mortality rate for **nosocomial** pneumonia is as high as 40%. Pneumonia is generally diagnosed in a patient presenting with symptoms of sudden-onset fever, chest pain, or dyspnea, and leukocytosis. Patients may also have a cough, either dry or productive, depending on the causative agent. Plain chest radiographs, posterior-anterior and lateral, are the gold standard for diagnosing a consolidated or infiltrative lesion.

There are an estimated 2 to 3 million cases of community-acquired pneumonia in the United States each year. *S pneumoniae* is the most common pathogen for all age groups of patients with community-acquired pneumonia. *S pneumoniae* gains entry to the respiratory tract following aspiration into the lungs from previously colonized nasopharyngeal mucosa. Elderly patients and those with other comorbidities are at a higher risk for succumbing to pneumonia infections. A history of tobacco use, chronic obstructive pulmonary disease, immunosuppression, and obesity are independent risk factors for developing community-acquired pneumonia.

Symptoms of bacterial pneumonias are high fevers, productive cough, and pleuritic chest pain. Many different bacterial species cause pneumonia, the most common being *S pneumoniae*, *H influenzae*, *S aureus*, *Chlamydia pneumoniae*, and *Legionella pneumoniae*.

Mycoplasma pneumoniae and viruses cause atypical pneumonias. They are considered atypical because patients usually present with a slower onset of symptoms, low-grade fever, and a dry cough. *Mycoplasma* pneumonia is often misdiagnosed as viral because of a less severe presentation. The clinical

lung exam result may be normal, but the chest radiograph will demonstrate infiltrates. *Mycoplasma* pneumonias are often preceded by a flu-like prodrome of symptoms, including headache, malaise, and fever.

Viral pneumonia is more common in infants and young children. Respiratory syncytial virus and parainfluenza virus are the most common causes of viral pneumonia in children. The incidence of these viral infections peaks in the fall and winter. Viral pneumonia is often preceded by an upper respiratory tract infection. Like atypical pneumonias, the onset of viral pneumonia can be slower than the onset of bacterial pneumonia.

Hospitalized patients—particularly the elderly—are susceptible to nosocomial pneumonias. The definition of nosocomial or **hospital-acquired pneumonia (HAP)** is a pneumonia that occurs in a hospitalized patient within 48 hours or more after admission to the hospital. (This type of pneumonia was not apparent at the time of admission.) Hospital-acquired pneumonia is the leading cause of death among nosocomial infections and is associated with a mortality rate as high as 50%. **Ventilator-associated pneumonia (VAP)** is a type of HAP that occurs in ventilated patients that appears more than 48 hours after endotracheal intubation. **Health care–associated pneumonia (HCAP)** is a pneumonia that occurs in nonhospitalized patients who have contact with health care facilities or personnel, such as residents of long-term health care facilities, those undergoing hemodialysis, or those who have had a recent admission to an acute care facility.

HAP, VAP, and HCAP can be caused by a number of different pathogenic bacteria and can be polymicrobial. Common pathogens include *E coli, Klebsiella pneumoniae, Enterobacter, Pseudomonas, S aureus*, and *Streptococcus*. Nosocomial bacterial pneumonias secondary to viral or fungal infection are much less common except in patients with severe immunosuppression.

Pneumocystis jiroveci (previously *Pneumocystis carinii*) pneumonia (PCP) is a fungal infection that is seen in patients with immunosuppression, such as those taking immunosuppressive therapy for solid organ transplantation or those infected with HIV, and is included in the list of AIDS-defining illnesses (Table 19-5). In fact, when a patient's CD4 cell count is less than 200 cells/μL, he or she is placed on trimethoprim-sulfamethoxazole prophylactically to prevent the development of PCP. The classic radiographic findings for PCP are bilateral interstitial infiltrates.

As with all respiratory illnesses, CCTPs should use standard precautions when caring for patients suspected of having pneumonia or those diagnosed as having pneumonia. If a patient is actively coughing, place a mask on the patient (if tolerated) and on yourself, wear gloves, and use good handwashing technique between transports. Equipment, including the transport vehicle, must be thoroughly cleaned and decontaminated prior to further transports.

Signs and Symptoms

Pneumonia

- Sudden-onset fever
- Chest pain
- Dyspnea
- Leukocytosis
- Cough, either dry or productive

Differential Diagnosis

Pneumonia

- Asthma
- Bronchiectasis
- COPD
- Lung cancer
- Pulmonary edema
- Pulmonary emboli

Transport Management

Pneumonia

- Provide supportive care.
- Continue administration of IV antibiotics or other medications if they have been started by the sending facility.

Fungal Diseases

Over the last 20 years, serious fungal infections have been increasing in incidence. These infections are not particularly contagious to healthy individuals and their presence in patients is an indication of serious immunosuppression. It is especially important to decontaminate equipment, such as laryngoscopes and suction equipment, after transportation of patients with oral candidiasis (thrush) or patients infected with *P jiroveci*.

Influenza

Influenza virus causes an acute upper respiratory illness lasting between 7 and 14 days depending on the severity of symptoms. It is caused by an orthomyxovirus and has three variants (A, B, and C). Influenza has an incubation period of about 1 to 3 days and is spread by droplet or fomite transmission. Complications in the elderly and the very young include the development of primary or secondary pneumonia. Treatment for influenza is mostly supportive; however, when diagnosed within the first day of symptoms, treatment with an antiviral medication may reduce the severity and duration of symptoms. It is not uncommon for an influenza epidemic to cause illness in 10% to 20% of people; the virus can be responsible for as many as 36,000 deaths and more than 100,000 hospitalizations per year **Table 19-3.**

Influenza A (unlike influenza B and C) is subject to antigenic drift, which is caused by small genetic changes in the virus. This alters the virus just enough so that antibodies generated in previous infections no longer recognize the viral epitopes or surface proteins and therefore do not protect the body from the symptoms of a new infection. Influenza type A is divided into subgroups based on the presence of two surface proteins (also called envelope spikes): hemagglutinin and neuraminidase. In addition to antigenic drift, influenza A is subject to more drastic changes called antigenic shift. This happens when one or both of the spike proteins change the form of their protein structure. Antigenic shift typically occurs only once in several years, whereas antigenic drift occurs more frequently. The phenomenon of antigenic drift is what precipitates the need for yearly preparation of influenza vaccine, thus requiring annual immunization.

TABLE 19-3 Influenza Landmarks in Humans During This Century

Year	Colloquial Name (Subtype)	Source	Impact
Pandemics			
1918	Spanish flu (H1N1 viruses such as swine flu)	Possible emergence from swine or an avian host of a mutated H1N1 virus	Pandemic with > 20 million deaths globally
1957	Asian flu (H2N2)	Possible mixed infection of an animal with human H1N1 and avian H2N2 virus strains in Asia	Pandemic, H1N1 virus disappeared
1968	Hong Kong flu (H3N2)	High probability of mixed infection of an animal with human H2N2 and avian H3Nx virus strains in Asia	Pandemic, H2N2 virus disappeared
1977	Russian flu (H1N1)	Source unknown but virus is almost identical to human epidemic strains from 1950; reappearance detected at almost the same time in China and Siberia	Benign pandemic, primarily involving persons born after the 1950s; H1N1 virus has cocirculated with H3N2 virus in humans since 1977
Incidents With Limited Spread			
1976	Swine flu (H1N1)	United States/New Jersey; virus enzootic in US swine herds since at least 1930	Localized outbreak in military training camp, with one death
1986	(H1N1)	The Netherlands; swine virus derived from avian source	One adult with severe pneumonia
1988	Swine flu (H1N1)	United States/Wisconsin; swine virus	Pregnant woman died after exposure to sick pig
1993	(H3N2)	The Netherlands; swine reassortant between old human H3N2 (1973/1975-like) and avian H1N1	Two children with mild disease; fathers suspected of transmitting the virus to the children after being infected by pigs
1995	(H7N7)	United Kingdom; duck virus	One adult with conjunctivitis
1997	Avian flu (H5N1)	Hong Kong; poultry virus	Since 2003, 421 cases worldwide with 257 deaths
2009	Novel H1N1	Mexico	At publication, ~ 45,000 cases and > 600 deaths in the United States; > 360,000 cases and > 4,000 deaths worldwide

Source: Modified from Snacken R, Kendal AP, Haaheim LR, Wood JM. The next influenza pandemic: lessons from Hong Kong, 1997. *Emerg Infect Dis*. 1999;5(2):195–203.

Influenza is a virus that can also infect species of animals other than humans—in fact, waterfowl are the natural reservoir for the influenza A virus. The influenza A virus is an avian virus that has "jumped species" to infect humans and other mammals. Humans, pigs, ducks, chickens and other animals are susceptible to influenza A. This variability of hosts causes mutations to occur in the virus that facilitate antigenic drift. When animals are infected with swine, human, and avian influenza simultaneously, there can be a mixing of the genes that encode the neuraminidase and hemagglutinin proteins. A large number of variations of these proteins are possible because there are nine known variations of neuraminidase and 16 known variations of hemagglutinin. Variations in influenza A are closely monitored by the World Health Organization (WHO) and the CDC in the United States as well as other global health associations. The subtypes of influenza A are categorized and tracked each year. The predominant subtypes of influenza A circulating through the world in 2009 are called H3N2, meaning that the hemagglutinin is type 3 and the neuraminidase is type 2, and H1N1. The highly pathogenic forms of avian influenza are H5 and H7. When influenza undergoes a major antigenic drift or a novel combination of hemagglutinin and neuraminidase emerges (called an antigenic shift), people have little or no antigenic protection from prior infections. This is when pandemics of influenza break out and spread quickly, causing major illness around the world.

Historically, new strains of influenza A virus arise in the Far East and it is likely that socioeconomic and geographic factors enter into the reasons for this. It is common for farmers in southeast China to live in close proximity with pigs and ducks. This sets up a perfect environment for the avian and human strains to recombine in pigs and then jump species back into humans with an antigenic shift. Each year, the WHO

and the CDC collect samples from the Far East to use as sentinel viruses for preparation of the influenza vaccine offered in the United States and other countries.

Signs and Symptoms

Influenza

- High fever
- Runny nose or congestion
- Cough
- Sore throat
- Muscle aches
- Fatigue
- Headache
- Nausea, vomiting, or diarrhea

Differential Diagnosis

Influenza

- Bacterial infection
- Fungal infection
- Parasitic infection
- Viral infection
- Typhoid

Transport Management

Influenza

- Provide supportive care.

CCTPs should be aware of the signs and symptoms of influenza infection and should use standard and respiratory precautions to avoid infection and spread of infection.

Herpesviruses

The herpesvirus family is a group of double-stranded DNA viruses that are surrounded by a lipoprotein envelope. Herpesviruses are noted for their ability to cause latent infections when the acute viral syndrome is followed by a symptomfree period during which the virus is **quiescent** or inactive. Depending on the species, reactivation of the virus can occur when the patient is immunosuppressed or through inciting agents. There are six important pathogenic herpesvirus members that cause significant disease in humans:

- Herpes simplex types 1 and 2

- Varicella zoster virus
- Cytomegalovirus Epstein-Barr virus
- Human herpesvirus 8

Both Epstein-Barr virus and human herpesvirus 8 are associated with the development of cancer, Burkitt's lymphoma, and Kaposi's sarcoma.

Herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2) cause lesions, but the lesions differ in their location. HSV-1 is transmitted in saliva, whereas HSV-2 is transmitted via sexual contact. Therefore, it follows that HSV-1 causes lesions above the waist, whereas HSV-2 causes lesions below the waist. However, either virus can be isolated from either location through oral-genital contact. HSV-1 causes an acute gingivostomatitis in children and recurrent *herpes labialis* in adolescents or adults, commonly known as cold sores. HSV-1 can also cause keratoconjunctivitis and encephalitis in immunocompromised hosts. HSV-2 commonly causes genital herpes **Figure 19-5** and can cause life-threatening infection in the neonate if delivered vaginally during an outbreak.

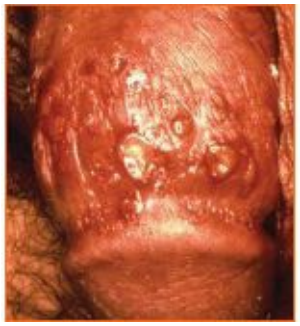

Figure 19-5 Herpes simplex virus type 2.

Viral Hepatitis

Hepatitis is the inflammation of the liver. Hepatitis can be caused by exposure to infectious agents, usually viral, toxins such as alcohol, or drugs such as acetaminophen. The course and severity of the illness range from acute to chronic and mild to life threatening, respectively. The wide range of signs and symptoms of liver disease can range from subclinical symptoms and nonspecific malaise to jaundice, hepatomegaly, bleeding dyscrasias, altered mental status, and multiple organ failure. Damage to the liver can be caused by direct injury to the hepatocytes by the agent or may result indirectly from an inflammatory response or an autoimmune attack.

There are six known hepatotrophic viruses (hepatitis A, B, C, D, E, and G), each having its own characteristic causative agent, mode of transmission, incubation period, associated risk factors, and sequelae. Viral hepatitis is first classified by the duration of the illness into either acute or chronic. Acute viral hepatitis is defined as the presence of signs and/or symptoms of liver inflammation for less than 6 months duration, whereas chronic viral hepatitis is defined as the presence of persistent liver inflammation for 6 months or more. Liver inflammation can also be described by histologic and pathologic findings, which allows for the grading and staging of liver disease. The grade of liver disease describes the severity of the inflammatory process and cell necrosis, and the stage of liver disease describes the severity of the scarring of the liver tissue.

Elevation of liver enzyme levels in the serum is used as evidence of damage to the liver, and the pattern of their elevation provides insight into the nature and probable cause of the insult. An acute viral hepatitis will be accompanied by an acute onset and marked elevation in aspartate aminotransferase (AST) and alanine aminotransferase (ALT), whereas a chronic viral hepatitis produces a more moderate and persistent elevation in these same enzymes. The ratio of serum AST to ALT is commonly greater than

two in liver disease caused by alcohol abuse, whereas the ratio is characteristically less than one in liver disease caused by viral agents.

Alkaline phosphatase (AP) is an enzyme that is present in many different tissues within the body, such as bone, intestine, kidney, liver, and placenta, but when a rise in AP levels is accompanied by a rise in gamma-glutamyl transferase (GGT) levels, the hepatic origin of AP is assumed. Elevations in levels of AP and GGT are seen when there is damage to the bile canaliculi, the cells that line the intrahepatic bile ducts. Damage to the bile canaliculi can be caused acutely by bile ducts that have been blocked by an obstructive process, such as an impacted gallstone, or can develop more slowly from a chronic process. Chronic viral hepatitis may produce swelling within the liver that may result in compression of the pericanalicular cells, leading to a marked rise in the so-called cholestatic enzyme levels, with the rise in the levels of hepatocellular enzymes being more moderate. The resulting rise of GGT and AP levels that occurs as the result of a viral process will occur later in the course of the disease, and the elevation is more moderate.

Signs and Symptoms

Hepatitis

- Nonspecific malaise
- Jaundice
- Abdominal pain
- Loss of appetite
- Intermittent nausea and diarrhea
- Scleral icterus
- Hepatomegaly
- Bleeding dyscrasias
- Altered mental status
- Multiple organ failure

Differential Diagnosis

Hepatitis

- $Alpha_1$-antitrypsin deficiency
- Biliary obstruction
- Drug-induced liver disease
- Sclerosing cholangitis
- Wilson disease

Transport Management

Hepatitis

- Provide supportive care.

Hepatitis A

Hepatitis A virus (HAV), previously known as infectious hepatitis, is a positive single-stranded RNA virus of the Picornaviridae family and is usually transmitted via the fecal-oral route. Sexual and parenteral transmission is possible; however, because the period of **viremia** is brief, fecal-oral transmission is more common. Large-scale outbreaks can occur, usually the result of contaminated food or drinking water. The estimated incidence of HAV is 125,000 to 200,000 cases per year in the United States, but only 25,000 cases are reported because most infections are asymptomatic and go unreported. In fact, 40% of persons living in urban settings have serologic evidence of prior HAV infections, but only 5% report memory of the liver infection. HAV causes approximately 100 deaths per year as a result of fulminant hepatitis. The clinical features of HAV include jaundice, fatigue, abdominal pain, loss of appetite, and intermittent nausea and diarrhea. Those at highest risk of infection are household/sexual contacts of infected persons, international travelers, and persons living in American Indian reservations, Alaskan native villages, and other regions with endemic HAV. During localized outbreaks, those at greatest risk include children who attend day care centers or their employees, men who have sex with men, and IV drug abusers. The incubation period ranges from 2 to 6 weeks. The period of greatest infectivity is the last 2 weeks of incubation, just before the onset of clinical symptoms, but the infected individual continues to be infectious by shedding virus in the feces for 2 to 3 weeks after the onset of clinical symptoms. The diagnosis of HAV is made by detection of anti-HAV IgM antibody. Anti-HAV IgG antibody is detected in the blood after exposure and recovery or immunization, which signifies life-long immunity.

HAV infection can be prevented by active immunization with the HAV vaccine or by passive immunization with immune globulin administered before or after a known exposure. The vaccine is highly effective and is recommended for children starting at the age of 1 year and persons at high risk of HAV infection, such as those planning to travel abroad, persons living in HAV-endemic areas, and children and caregivers at day care centers. The prevention of infection can be accomplished by improving sanitation and by employing good personal hygiene and handwashing.

Hepatitis B

Hepatitis B virus (HBV) is a double-stranded DNA virus of the Hepadnaviridae family, and is transmitted through parenteral routes, or nonoral transmission, with an incubation period of 1 to 6 months. Each year in the United States, more than 200,000 people are infected with HBV, and nearly 5,000 people die from HBV infection. Transmission can occur by sharing needles, having sex with an infected person, or accidental medical exposures, such as needlesticks, blood spray, or touching bloody items with unprotected hands. Although blood is the most effective fluid of transmission, the virus lives in all human body fluids and is present in semen, saliva, urine, and breast milk. Therefore, because health care providers come into contact with patients who are infected with HBV, they are considered an at-risk group for viral infection and should receive the HBV vaccine series with immunity confirmed by titer.

The clinical features of HBV are similar to those of HAV, including jaundice, scleral icterus **Figure 19-6**, fatigue, abdominal pain, loss of appetite, and intermittent nausea or vomiting. However, unlike HAV, HBV can induce other disease states in addition to causing acute infections. HBV can cause acute hepatitis, fulminant hepatitis, and chronic hepatitis. Fulminant hepatitis is a severe inflammation of the liver, that is accompanied by rapid destruction of the liver, leading to liver failure. The liver damage is accompanied by encephalopathy within 8 weeks of the onset of the symptoms. Coagulopathy, electrolyte disturbances, and cerebral edema are common and death is likely if the patient does not receive an urgent liver transplant. Chronic HBV can take on several forms. A patient can be chronically infected yet remain symptomless. This state is termed a chronic asymptomatic carrier. The patient never develops immunity to the virus but harbors the virus and is infectious to others without actually suffering liver damage. There

are an estimated 200 million carriers of HBV worldwide. Chronic-persistent hepatitis is infection with HBV that "smolders," (ie, it is a persistent low-grade infection from which the patient eventually recovers). In addition, there is chronic-active hepatitis, which means the patient has an active hepatitis state and continues without recovery and without severe decline in liver function that continues for more than 6 months.

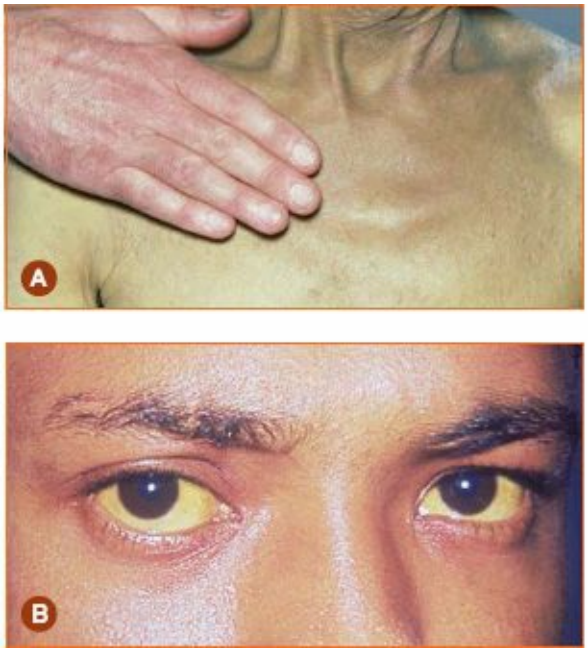

Figure 19-6 **A.** Jaundice. **B.** Scleral icterus.

The key to understanding HBV serology, infectivity, and immunity begins with understanding the viral structure. HBV is an enveloped DNA virus. Immunity to HBV is acquired after exposure to the surface antigen through exposure either to the natural virus or to viral surface components in the vaccine. In either case, immunity is acquired through the development of antibodies to the surface antigen or (anti-HBs). Antibodies to the core proteins, anti-HBc, do not confer immunity because, in order for an individual to ward off the virus, antibodies must attack the intact virion before the virus has a chance to replicate and before the individual is exposed to the core antigen. If the person only has anti-HBc, the immune response will not be mounted until the core protein is unmasked, which is too late to prevent infection. Therefore, antibodies to the core antigen, anti-HBc, are not protective and do not confer immunity, rather their presence in the serum is used as a marker of previous exposure. Only anti-HBs will develop in response to the hepatitis B vaccine. Knowing this helps a provider determine how immunity to HBV was acquired. For example, if the individual has both anti-HBs and anti-HBc, then the individual has been exposed to the wild-type virus (because of the presence of anti-HBc), and is now immune (because of the presence of anti-HBs). The subtype of immunoglobulin yields information about the duration of infection. If the anti-HBc is IgM-type immunoglobulin, then the infection is acute or is from a recent exposure. Anti-HBc IgG means that the exposure occurred more than 3 months prior and the infection is resolving or is chronic.

Those at highest risk for contracting HBV are individuals who participate in high-risk behaviors; live in or travel to disease-endemic areas such as parts of Greenland, northern Canada, southeast Asia, China, and all of Africa; or are exposed to infected body fluids through their lifestyle or occupation. They include IV drug users, sexually active heterosexuals, men who have sex with men, individuals of low socioeconomic status, children of immigrants from disease-endemic areas, infants born to infected mothers, health care workers, and patients receiving hemodialysis.

The treatment and prevention of HBV infection involves screening the blood supply and removing

HBV-contaminated units and donors from the blood pool. The HBV vaccine is recommended for all infants at birth and for children up to the age of 18 years. It is also recommended that adults with a high risk of exposure be vaccinated. All pregnant women should be screened for the disease, and infants born to infected mothers should receive treatment with hepatitis B immune globulin (H-BIG) at birth, in addition to completing the vaccination series during the first 6 months of life. The identification and treatment of individuals with chronic infections and asymptomatic carriers is important for preventing the spread of the disease. The vaccine is given in three separate doses at 0, 1, and 6 months. Passive immunity can be conferred by giving HBV immunoglobulin to persons with known exposures if given within 24 hours of the exposure.

Primary hepatocellular carcinoma is a known complication of chronic HBV infection. The risk of primary hepatocellular carcinoma is 200 times greater for those with chronic HBV infection over those who are not infected. In the United States, 20% of persons with hepatocellular carcinoma are hepatitis B surface antigen positive. Other complications of HBV infection include cirrhosis and fulminant liver failure in less than 1% of cases.

Hepatitis C

Hepatitis C virus (HCV) is an enveloped RNA virus of the Flaviviridae family, which was first known as non-A, non-B hepatitis. HCV causes both acute and nonacute or chronic hepatitis. The virus is transmitted parenterally through transfusion or IV drug use, through sexual contact with infected persons, and from mother to baby; however, the vertical transmission rate of HCV is quite a bit lower than that of HBV. The incubation period of HCV varies from 2 weeks to 6 months. There are 36,000 new cases of HCV infection each year in the United States, of which only 25% to 30% are symptomatic. In cases of acute hepatitis, the symptoms usually consist of nausea, vomiting, and jaundice. In most cases (more than 85% of infections), acute infection leads to chronic infection and spontaneous clearance of the virus is rare once chronic infection is established. Chronic infection typically begins with a long period in which there are no symptoms. The natural history of HCV has been difficult to determine because the disease is so often silent and goes undetected during the early stages, and the interval from infection to the development of cirrhosis can exceed 30 years. Most chronic infections lead to hepatitis and fibrotic liver disease. Cirrhosis develops in 15% to 20% of infected persons, which leads to 10,000 deaths per year. HCV cirrhosis is the leading cause of liver transplantation.

The risk factors most strongly associated with HCV infection include IV drug use and a history of blood transfusion before 1990. Others at risk for HCV infection include patients receiving hemodialysis, health care workers, sexual contacts of infected persons, hemophiliacs, and infants born to infected mothers.

Anti-HCV antibodies develop after exposure to the virus but do not confer immunity. However, their presence in the blood is exploited for serologic assays to screen for contaminated blood products and to identify the carrier of the infection. The antibody is detected through an enzyme-linked immunosorbent assay, a technique that has a sensitivity of 97%, but the positive predictive value in a low-risk population is only 25%. Other techniques, such as a recombinant immunoblot assay, have been developed and are more useful for testing low-risk persons.

There is no cure for HCV and there is no vaccine available to prevent infection. Patients with chronic active HCV infections are candidates for treatment. The goal of treatment is to prevent the development of cirrhosis and liver failure, but success is limited and the side effects of treatment with interferons and antivirals can be difficult to tolerate. Most clinicians agree that treatment should be offered to those patients who have detectable levels of HCV RNA with persistently elevated liver transaminases and who demonstrate evidence of liver damage on biopsy.

Hepatitis D

Hepatitis delta virus (HDV) is another RNA virus that is transmitted parenterally, but it can only replicate with the aid of HBV. HDV is endemic in the Mediterranean region, the Middle East, and parts of South America. Outside of these areas, most HDV infections are a result of transfusion with tainted blood products or IV drug use. The two ways in which a person can be infected with HDV are by coinfection with HBV or superinfection. In coinfection, the two viruses are transmitted together parenterally and cause an acute hepatitis similar to HBV alone. In superinfection, persons with chronic HBV infection are later exposed to HDV and have a superimposition of an acute-on-chronic hepatitis picture. This type of HDV infection is often severe and is associated with a higher incidence of fulminant hepatitis with a greater mortality rate. Therefore, all patients with severe HBV infection should be tested for HDV coinfection or superinfection. There is no treatment for HDV; therefore, the best way to combat the disease is to control or prevent HBV infection.

Hepatitis E

Hepatitis E virus (HEV) is rare in the United States and is limited to travelers to endemic areas of India, Southeast Asia, Africa, and Mexico. The virus is transmitted via the fecal-oral route through the ingestion of contaminated food or water sources. Its transmission and course are most like those of HAV, but HEV is associated with a high fatality rate in pregnant women.

Hepatitis G

Hepatitis G virus (HGV) is another RNA virus of the Flaviviridae family and is transmitted through parenteral routes. It has frequently been detected in patients with chronic liver disease, but a causative link has not yet been established.

Human Immunodeficiency Virus

Human immunodeficiency virus is the virus that causes AIDS and AIDS-related complex (ARC—a syndrome or a group of symptoms associated with AIDS). The first reported case of AIDS occurred in 1981 in Los Angeles. Although the disease was first encountered in homosexual men, it was also seen in IV drug users, transfusion recipients from Haiti, female sexual contacts of infected men, prisoners, and Africans. In the beginning, there were many theories regarding the causative agent of the disease, but the observation that the epidemiology of AIDS was similar to that of hepatitis B infection persuaded researchers to search for a viral component. The cause of AIDS, a novel retrovirus that belongs to the human T-lymphotropic virus group, was isolated in 1983, and was positively linked as the primary cause of AIDS in 1984. At the end of 2004, an estimated 1,147,697 HIV or AIDS cases had been diagnosed and reported to the CDC. The estimated prevalence of HIV in the United States—the total number of people living with HIV infection—is just over 1 million, and as many as 27% of these persons are unaware of their infections. AIDS is the sixth leading cause of death in the United States for those between the ages of 25 and 44 years. AIDS is now pandemic. According to recent estimates, there are 33 million people infected with HIV, with 7,500 new infections occurring daily worldwide.

HIV is a retrovirus that infects T lymphocytes and other cells that display the CD4 surface protein. HIV belongs to a subgroup of retroviruses called the lentiviruses, which cause “slow” infections with long incubation periods. HIV has a bar-shaped core that contains the RNA virus and three essential retroviral enzymes **Figure 19-7**. The core is surrounded by an envelope that contains the virus-specific glycoproteins gp120 and gp41. The viral genome consists of two identical positive single-stranded RNA molecules. HIV has been categorized into subtypes (clades) A through I based on the differences in base-pair sequences that encode the glycoprotein gp120.

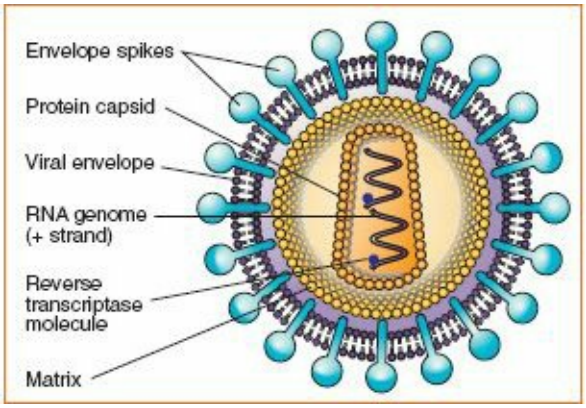

Figure 19-7 The human immunodeficiency virus.

The three essential enzymes enclosed in the retroviral capsid are reverse transcriptase, integrase, and protease. Reverse transcriptase is an RNA-dependent DNA polymerase that gives the retroviruses their name. This enzyme is responsible for converting the RNA viral genome into a DNA provirus. Integrase is the enzyme that catalyzes the integration of the newly transcribed proviral DNA into the host cell genome. Protease is the enzyme that is responsible for producing functional viral proteins. Understanding these specific retroviral enzymes becomes important for understanding how antiretroviral therapy was designed for the treatment of HIV/AIDS.

Transmission of HIV infection occurs by the transfer of infected cells or free virus from one individual to another. The transmission occurs horizontally via sexual contact or by exposure to infected blood, and vertically from mother to neonate across the placenta, during delivery. The infant can also contract HIV from an infected mother through breast milk. The transmission rate of HIV during pregnancy and birth is 25% to 45% and 15% to 25%, with and without breastfeeding, respectively. The transmission rate can be reduced to less than 2% if the mother is treated adequately with zidovudine (AZT) during the pregnancy, the baby is delivered by cesarean section, and the mother does not breastfeed. The HIV transmission rate increases in persons who also have other sexually transmitted infections, especially those that cause disruption of the mucous membrane barrier. The risk of contracting HIV from a blood transfusion has been reduced through aggressive screening of donated blood, and the risk is extremely low but cannot be completely eliminated because there is a small window of time when the virus is not detected in persons with new infections. The CDC, in partnership with 14 countries in Africa and the Caribbean and under the President's Emergency Plan for AIDS Relief (PEPFAR), has worked to reduce the risk of transmitting HIV through blood transfusions. The goal is to deliver an adequate supply of safe blood to these participating countries by collecting blood from low-risk, voluntary, nonremunerated donors and strengthening laboratory and screening capacity.

As a CCTP, exposure to patients with HIV infection is a potential risk for disease contraction. Prevention of exposure (ie, needlesticks) is the most important method of protection for the health care worker. The incidence of needlesticks has been reduced by advances in needleless devices, safety education, and emphasis on proper sharps disposal. Regardless of the stated considerations, needlestick injuries still occur. Fortunately, the risk of becoming infected with HIV after a significant exposure to body fluids from a patient infected with HIV is low. The estimated risk of transmission is 0.33% for hollow-bore needlesticks and 0.09% for mucosal contact; there are no known cases of HIV transmission for intact skin exposures.

HIV infects CD4+ helper T cells and kills them, resulting in the suppression of cell-mediated immunity. There are three stages in HIV infection: acute, latent, and late (or the period of immunodeficiency). The stage of the illness is largely determined by the CD4 cell count and/or symptoms of the disease **Table 19-4.** The initial exposure to the virus starts a sequence of events that goes virtually

unnoticed by the individual, but allows the virus to be permanently established in the individual's immune system. The initial infection begins in the immune cells of the genital tract, and the virus quickly spreads to localized CD4+ helper T cells. However, the virus is not detected in the blood until 4 to 11 days after the initial infection. The acute phase of the disease occurs 2 to 6 weeks after the initial exposure. The patient may present with mononucleosis-like symptoms of fever, lethargy, generalized lymphadenopathy, pharyngitis, and arthralgias. A maculopapular rash on the trunk, arms, and legs, sparing the palms and soles, may also be seen during this time. The acute phase of the disease usually resolves within 2 weeks.

TABLE 19-4 HIV/AIDS Classification of Disease Stage

Stage	Classification	CD4 Cell Count (cells/μL)
1	Asymptomatic	> 500
2	AIDS-related complex	200–499
3	AIDS	< 200

Abbreviations: AIDS, acquired immunodeficiency syndrome; HIV, human immunodeficiency virus.

The disease classification is based on the lowest T-cell count, not the current count. Once a patient has a T-cell count of less than 200 cells/μL, the patient is diagnosed with AIDS regardless of whether the T cell rebounds with therapy. Antibodies to HIV are not detected in the blood until 3 to 4 weeks after the initial infection. Once antibodies are detected in the serum, the patient is said to have seroconverted. The inability to detect antibodies in the blood prior to **seroconversion** results in false-negative serologic test results. This has important implications for the transmission of the virus and for the screening of the blood supply. The person is infected with HIV and is capable of spreading the disease through sexual or blood contacts during this window of time, even though the HIV antibody test result is negative.

After the initial viremia, a viral set point is reached. The viral set point represents the amount of virus present in the blood, is called the viral load, and tends to remain fairly constant for many years. The lower the viral set point at the end of the initial infection, the more likely the patient will remain in the latent stage of the disease. The latent stage of the disease usually lasts for years (the median duration is 10 years), and the patient is generally asymptomatic during this period. While the patient is asymptomatic and the symptoms are latent, the virus is not "latent," but is actively replicating within the lymph nodes of the patient. Toward the end of clinical latency, the patient may begin to experience a syndrome of symptoms called AIDS-related complex. The symptoms of AIDS-related complex include fevers, unexplained weight loss, fatigue, diarrhea, and generalized lymphadenopathy. Often, these symptoms are what persuade the patient to seek help in the health care system. AIDS-related complex may mark the beginning of the final progression toward AIDS.

AIDS is the term that refers to the late stage of HIV infection. The criteria for the diagnosis of AIDS include HIV infection with a CD4+ helper T cell count of less than 200 cells/μL and/or the presence of an AIDS-defining condition. The presence of an AIDS-defining condition supercedes any CD4 cell count **Table 19-5.**

TABLE 19-5 AIDS-Defining Conditions

Candidiasis of the bronchi, trachea, lungs, or esophagus
Cervical cancer, invasive*

~~Coccidioidomycosis, disseminated or extrapulmonary~~ Cryptococcosis, extrapulmonary
Cryptosporidiosis, chronic intestinal (> 1 month's duration)
Cytomegalovirus disease (other than liver, spleen, or nodes)
Cytomegalovirus retinitis (with loss of vision)
Encephalopathy, HIV-related
Herpes simplex: chronic ulcer(s) (>1 month's duration) or bronchitis, pneumonitis, or esophagitis
Histoplasmosis, disseminated or extrapulmonary
Isosporiasis, chronic intestinal (> 1 month's duration)
Kaposi's sarcoma
Lymphoma, Burkitt's (or equivalent term)
Lymphoma, immunoblastic (or equivalent term)
Lymphoma, primary, of brain
Mycobacterium avium complex or *Mycobacterium kansasii*, disseminated or extrapulmonary
Mycobacterium tuberculosis, any site (pulmonary* or extrapulmonary)
Pneumocystis jiroveci pneumonia
Toxoplasmosis of the brain
Abbreviation: HIV, human immunodeficiency virus syndrome. *Added in the 1993 expansion of the acquired immunodeficiency syndome surveillance case definition. *Source*: Data from Castro KG, Ward JW, Slutsker L, et al. 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. Centers for Disease Control and Prevention. *MMWR*. December 18, 1992/41 (RR-17). Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/00018871.htm. Accessed June 29, 2009.

Patients with HIV often present many years after the initial infection or the acute stage of the disease because in the initial stages, the disease is often mild and the symptoms are nonspecific. For many patients with HIV, the illness is noticed when the patient begins to experience symptoms such as generalized lymphadenopathy, weight loss, fatigue, fevers, or night sweats. As the disease progresses, the patient may experience frequent severe herpes infections of the mouth or anus and/or recurrent or persistent yeast infections. The loss of cell-mediated immunity predisposes the host to many opportunistic infections. Symptoms of opportunistic infections commonly seen in patients with AIDS include cough, dyspnea, headache, altered mental status, memory loss, seizures, ataxia, dysphagia, nausea, vomiting, severe persistent diarrhea, and marked weight loss.

The viral load is estimated by determining how much viral RNA is present in the blood. The viral load indicates the magnitude of HIV replication and its associated rate of CD4 cell destruction. It is the most accurate indicator of the risk for disease progression and is used for planning and monitoring anti-retroviral therapy. The CD4 cell count is used to evaluate the extent of HIV-induced immune damage that has occurred, to provide an estimate of the immunologic status of the patient, and to predict the likelihood of the patient developing opportunistic infections. The initiation of antiretroviral therapy is based on the

viral load, the CD4+ T-cell count, and the patient's clinical conditions.

Three general classes of antiretroviral drugs are currently available for the treatment of HIV infection: nucleoside analog reverse transcriptase inhibitors (NRTIs), protease inhibitors (PIs), and non-nucleoside reverse transcriptase inhibitors (NNRTIs). There is a fourth class of drugs in development called fusion inhibitors for which two drugs are currently available.

NRTIs target the enzyme reverse transcriptase. They act to block the transcription of the viral RNA to the proviral DNA by competing with physiologic nucleosides for the enzyme-binding site. Once they are added to the growing DNA sequence, they block the addition of the next nucleoside and, thus, interrupt the synthesis process causing chain termination. The prototype of the NRTIs is AZT.

NNRTIs are similar to the NRTIs in that they target the same viral enzyme, reverse transcriptases. In contrast to the NRTIs, they are not competitive inhibitors of the enzyme, rather they are noncompetitive inhibitors of the enzyme. They bind to the enzyme near the binding site for the nucleoside substrate and alter the configuration of the enzyme, preventing it from carrying out its catalytic function. Nevirapine is an example of an NNRTI.

PIs target the viral enzyme protease that cuts the viral polyprotein into functional individual proteins. Inhibiting the protease prevents the processing of viral proteins, which then prevents the building of mature virion. Indinavir is an example of a typical PI. Because the step that the PIs inhibit is late in the viral replication process, PIs are most often used in combination with other agents.

There are three crucial steps for the entry of HIV into the CD4+ T cell: the binding of HIV to the CD4 receptor, also called attachment; the binding of HIV to the co-receptor; and the fusion of the virus and the cell membranes. All three steps are the targets of a new class of drugs collectively known as entry inhibitors. Enfuvirtide is the prototype of the fusion inhibitors. It binds to gp41, which is thought to be important in the fusion process. It was approved for use in the United States in 2003. Its role in HIV treatment is limited to those in whom the standard therapies fail. It is prohibitively expensive and is only available for parenteral administration.

The goal of current therapies for HIV infection is to prolong life, while maintaining the best possible quality of life. Achieving this requires that the prevention of opportunistic infections and malignancies, and slowing the rate of virus production, be weighed against the side effects of therapy, and therapy must be delivered to the patient in a manner that promotes compliance. The most difficult question in the treatment of HIV is when to start therapy. The current treatment of choice for advanced disease uses a combination of the described agents, usually consisting of two NRTIs and one PI, known as HAART, or highly active antiretroviral therapy. This treatment is very effective in prolonging life and reducing the viral load, but ensuring patient compliance can be challenging because the regimens are often complicated and the side effects can be debilitating. Viral resistance to the antiretroviral drugs is increased when patients use the drugs intermittently or incorrectly. Indications for treatment are based on clinical assessment of the patient's condition, the CD4 cell count, and viral load. The risk of AIDS must be weighed against the risks of long-term toxicity and resistance. The risks of occupational exposures and postexposure prevention protocols are discussed later in this chapter.

Signs and Symptoms

HIV Infection/AIDS

Early symptoms:

- Generalized lymphadenopathy
- Weight loss

- Fatigue
- Fevers
- Night sweats

Symptoms associated with disease progression:

- Frequent, severe herpes infections of the mouth or anus
- Recurrent or persistent yeast infections

Symptoms associated with opportunistic infections:

- Cough
- Dyspnea
- Headache
- Altered mental status
- Memory loss
- Seizures
- Ataxia
- Dysphagia
- Nausea
- Vomiting
- Severe persistent diarrhea
- Marked weight loss

Differential Diagnosis

HIV Infection/AIDS

- B-cell deficiency
- Brucellosis
- Chronic fatigue syndrome
- Dementia
- Epstein-Barr virus
- Fibromyalgia
- Hodgkin disease
- Hypogammaglobulinemia
- Immunodeficiency syndromes
- Influenza
- Lyme disease
- Multiple sclerosis
- Myositis
- Non-Hodgkin lymphoma
- Pelvic inflammatory disease
- Pyoderma gangrenosum
- Severe combined immunodeficiency (SCID)
- T-cell deficiency
- Thrombocytopenic purpura, autoimmune

Transport Management

HIV Infection/AIDS

- Provide supportive care.

Emerging Infectious Diseases

The eradication of infectious diseases has been one of modern medicine's most important achievements. The influenza pandemic of 1918 and 1919, which caused more than 20 million deaths, was thought to be a phenomenon of historic interest. With the advent of effective vaccinations and antibiotics, the death rate from infectious disease declined dramatically during the 20th century, and this success led to complacency. The death rate from infectious disease increased by 58% between 1980 and 1992. Infectious diseases are still the leading cause of death worldwide. Hantavirus, West Nile virus, SARS, and avian influenza have emerged as new threats on the medical landscape. The emergence and re-emergence of drug-resistant infectious agents, such as methicillin-resistant *S aureus*, vancomycin-resistant enterococci, and MDR-TB, have increased dramatically in the last 2 decades.

The term "emerging infectious disease" refers to diseases caused by infectious agents that have increased in incidence within the past 20 years or those that threaten to increase in the near future. The CDC has identified several major factors that contribute to the development or re-emergence of infectious disease. The CDC points to the changes in human behaviors, the changes in economic and technologic advances, and the adaptive changes of the infectious agents. The mobility of modern society has facilitated the movement of people, animals, and foodstuffs across countries and continents. The triangle of transmission between microbe, vector, and host enlarges when access to naïve populations who lack immunity is provided. Selective pressures from overuse of antibiotics (as described earlier when selection was discussed) have precipitated the development of resistant microbes, and abuse of antimicrobials has enhanced the re-emergence of more deadly infections.

Additionally, infectious agents are now thought to be important in the etiology of many diseases previously thought of as noninfectious. *Helicobacter pylori* has long been found in association with peptic ulcer disease, but it is now known to have a causative association. Human papillomavirus is the major cause of cervical cancer (a vaccine that is now available). Hepatitis C virus is a leading cause of chronic liver disease and cirrhosis in the United States and is a major risk factor for the development of hepatocellular carcinoma.

Emerging infectious diseases are particularly damaging to immunocompromised individuals, such as those with HIV disease, those receiving immunosuppressive therapy such as recipients of organ transplants, or those who are immunosuppressed as a result of their treatment for cancer. Public water supplies that are contaminated by emerging infectious agents put entire communities at risk. In 1993, *Cryptosporidium* contaminated a municipal water supply in Wisconsin and caused 4,400 people to be hospitalized from the infection. Large segments of populations may also be exposed to emerging infections through contaminated food sources. *Escherichia coli* O157:H7 was found to have contaminated beef cooked in fast food restaurants, causing a multistate outbreak of hemorrhagic colitis and serious kidney disease and resulting in the deaths of at least four children.

To effectively detect and prevent the threat of emerging infections, the public health sector, health care providers, and emergency medical services must cooperate in an effort to control any potential outbreaks and prevent the spread of potentially deadly diseases.

Severe Acute Respiratory Syndrome

Severe acute respiratory syndrome (SARS) is an emerging lower respiratory tract illness caused by a corona virus. SARS appeared as a highly contagious, deadly virus early in 2003 in the Guangdong Province of China. The virus quickly disseminated via person-to-person droplet transmission to persons in Hong Kong and Vietnam and subsequently to Singapore and Canada. Those at highest risk in the early spread of the disease were health care workers. Ultimately, the epidemic spread to 29 countries, resulting in more than 8,000 infected persons and 780 deaths. The cooperative efforts of scientists and physicians in the WHO and CDC as well as China, Canada, Hong Kong, and other countries proved effective for quick identification of the etiologic agent and isolation and prevention of the spread of the disease. Because of the high morbidity and mortality associated with SARS, the CDC has included it on its list of notifiable diseases to ensure careful monitoring of the US population for further outbreaks.

The virus has an incubation time of 4 to 6 days, and most patients show symptoms within 2 to 10 days after exposure. Early symptoms of fever, headache, and muscle aches are nonspecific and may be mistaken for other viral illnesses. The presenting symptoms are followed within 2 to 7 days by a dry cough and pneumonia. The overall mortality rate (10%) is dramatically higher in the elderly, causing death in 50% of those older than 60 years.

Constant surveillance and early detection of SARS are necessary to prevent future epidemics. One lesson learned in the 2003 outbreak was that international air travel was a key factor in the rapid and global spread of this disease. The WHO quickly recommended screening of international passengers from affected areas and disinfection of aircraft suspected of carrying infected persons. Additionally, the lack of respiratory isolation and respiratory precautions for patients hospitalized early in the disease outbreak permitted nosocomial transmission of the virus, in particular to health care workers. Thus, when transporting persons with a cough, fever, headache, and myalgias of unknown etiology, it is important to place a mask on the patient, keep the patient in respiratory isolation, and sterilize all equipment following transport **Table 19-6. Table 19-7** lists recommended personal protective equipment (PPE), organized by infectious disease.

Signs and Symptoms

Severe Acute Respiratory Syndrome

- Fever
- Headache
- Muscle aches
- Later symptoms: dry cough and pneumonia

Differential Diagnosis

Severe Acute Respiratory Syndrome

- Bacterial septicemia
- *C pneumoniae* infection
- Influenza
- Legionellosis
- Leptospirosis
- *M pneumoniae* infection

- Hantavirus infections (Old World)
- Q fever
- Typhoid and enteric fever

Transport Management

Severe Acute Respiratory Syndrome

- Place a mask on the patient.
- Manage the airway.
- Keep the patient in respiratory isolation.
- Monitor the mechanical ventilator, if applicable.
- Continue IV administration of antivirals or corticosteroids if they have been started by the sending facility.
- Perform high-level disinfection of all equipment after transport.

Escherichia coli O157:H7

Escherichia coli are microorganisms found in the intestinal tract of humans and other mammals. There are hundreds of different strains of *E coli*, most of which are harmless, and a few strains that are pathogenic. The pathogenic strains may have specialized fimbriae (ie, finger-like border), allowing them to adhere to certain intestinal epithelial cells and produce toxins that cause gastrointestinal problems. There are several specific pathogenic groups of *E coli*, including enterotoxigenic, enteroinvasive, enteroaggregative, enteropathogenic, and enterohemorrhagic. *Escherichia coli* O157:H7, which causes hemorrhagic colitis, is emerging as a particularly virulent strain.

Escherichia coli O157:H7 is found in the intestinal tracts of ruminant animals, especially cattle. Individuals have become infected after eating undercooked, contaminated ground beef; drinking unpasteurized milk and fruit drinks; eating unwashed lettuce or sprouts; eating salami; and swimming in or drinking sewage-contaminated water. Transmission can also occur via person-to-person contact, usually from infected persons who have poor hygiene or handwashing technique. It takes fewer than 10 *E coli* bacteria to cause the onset of symptoms.

The Shiga-like toxins produced by these microorganisms are responsible for causing hemorrhagic colitis, an inflammation of the colon with bleeding and abdominal cramps. Most episodes are self-limiting and last only 5 to 10 days. A small number of infected people, usually those younger than 5 years or older than 65 years, experience hemolytic uremic syndrome. This syndrome is manifested when the toxin affects the kidneys, leading to kidney failure and end-stage renal disease that necessitates dialysis or transplant to sustain life.

TABLE 19-6 Recommendations for the Application of Standard Precautions for the Care of All Patients in All Health Care Settings

Component	Recommendations
Hand hygiene	After touching blood, body fluids, secretions, excretions, and contaminated items; immediately after removing gloves; and between patient contacts

Personal protective equipment	Gloves, mask, and gown
Gloves	For touching blood, body fluids, secretions, excretions, and contaminated items; for touching mucous membranes and nonintact skin
Mask, eye protection, and face shield	During procedures and patient care activities likely to generate splashes or sprays of blood, body fluids, and secretions; N95 mask to prevent respiratory transmission
Gown	During procedures and patient care activities when contact of clothing/exposed skin with blood/body fluids, secretions, and excretions is anticipated
Soiled patient care equipment	Handle in a manner that prevents transfer of microorganisms to others and to the environment; wear gloves if visibly contaminated; and perform hand hygiene
Environmental control	Develop procedures for routine care, cleaning, and disinfection of environmental surfaces, especially frequently touched surfaces in patient care areas
Textiles (linen and laundry)	Handle in a manner that prevents transfer of microorganisms to others and to the environment
Needles and other sharps	Do not recap, bend, break, or hand-manipulate used needles; use safety features when available; and place used sharps in a puncture-resistant container
Patient resuscitation	Use mouthpiece, resuscitation bag, and other ventilation devices to prevent mouth contact
Patient placement	Prioritize for the single patient room if the patient is at increased risk of transmission, is likely to contaminate the environment, does not maintain appropriate hygiene, or is at increased risk of acquiring infection or developing an adverse outcome following infection
Respiratory hygiene/cough etiquette (source containment of infectious respiratory secretions in symptomatic patients, beginning at the initial point of the encounter)	Instruct symptomatic persons to cover their mouth/nose when sneezing/coughing; use tissues and dispose in a no-touch receptacle; observe hand hygiene after soiling of hands with respiratory secretions; and place a surgical mask on the patient if tolerated, wear an N95 mask, or maintain spatial separation, greater than 3′ if possible

Source: Centers for Disease Control and Prevention, Department of Health and Human Services, 2004. Severe acute respiratory syndrome: Public health guidance for community-level preparedness and response to severe acute respiratory syndrome (SARS)–version 2, January 8, 2004. Available at: http://www.cdc.gov/ncidod/sars/guidance/I/index.htm. Accessed April 29, 2004.

The diagnosis of *E coli* infection consists of performing stool cultures using a specialized growing medium such as sorbitol-MacConkey agar plates. Treatment is difficult because of emerging antibiotic

resistance. Thoroughly cooking beef, avoiding unpasteurized drinks, and washing hands after handling raw meat can prevent infection with *E coli* O157:H7.

West Nile Virus

West Nile virus (WNV) is an arthropod-borne virus that was first isolated in Uganda's West Nile district in 1937. It was never isolated in the United States until 1999, when the first cases of West Nile virus–associated encephalitis were reported in New York City. Evidence suggests that international travel and commerce have played a significant role in its introduction into the Western hemisphere. In Africa and Asia, disease outbreaks caused by WNV were infrequent and were not associated with serious morbidity or mortality. Since its introduction in the United States, the frequency and apparent severity of WNV cases have increased. The WNV epidemic of 2002, the largest meningoencephalitis epidemic recorded to date, resulted in more than 4,000 cases and 200 deaths. In 2008, there were more than 1,300 documented cases and 44 deaths in the United States.

WNV is a single-stranded RNA *Flavivirus* of the Flavi-viridae family. Virion binding to host cells is mediated by an E-glycoprotein. The E-glycoprotein is a viral hemagglutinin that is highly immunogenic and elicits production of virus-neutralizing antibodies in the immunocompetent host. WNV is a member of the Japanese encephalitis virus serocomplex that contains other medically important viruses, such as the St. Louis encephalitis virus. The close antigenic profiles of the flaviviruses, especially those of the Japanese encephalitis group, cause serologic cross-reactions in the diagnostic laboratory.

WNV is a mosquito-borne pathogen. Adult mosquitoes emerge in the spring. Viral amplification occurs in mosquito-bird-mosquito cycling until early fall when female mosquitoes begin diapause and infrequently bite humans to gain protein for egg development from a blood meal **Figure 19-8**. The virus is found in the salivary glands of the mosquito and is transferred during the bite. Mosquitoes are the bridge vectors that maintain the cycle of infection by feeding off both humans and birds.

TABLE 19-7 Personal Protective Equipment Recommended (by Infectious Disease)

	Standard Precautions				
	Airborne		Droplet	Contact	Eye Protection
Disease	Negative Pressure Transport	N95 Mask	Surgical Mask	Gloves and Gown	Goggles or Face Mask/Shield
Respiratory infections					
Respiratory syncytial virus			Required	Required	Recommended
Influenza			Required	Required	Recommended
Novel respiratory infection (such as SARS or avian flu)	Required	Required	Required if N95 is not available	Required	Required
Pulmonary TB	Recommended	Recommended	Required if N95 is not available	Required	
Meningitis					
			Surgical mask	Required	
Hepatitis					
				Required	Recommended
MDR diseases					
MRSA or VRSA			Recommended	Required	Recommended
VRE			Recommended	Required	Recommended
MDR TB	Recommended	Recommended	Required if N95 is not available	Required	Recommended
Clostridium difficile					
				Required	
Human immunodeficiency virus					
		Required	Required if N95 is not available	Required	Required
Necrotizing fasciitis					
			Required	Required	
Hemorrhagic fevers of unknown etiology					
	Recommended	Recommended	Required if N95 is not available	Required	Required
Any contact with blood, body fluids, or feces, especially if aerosolized					
			Required	Required	Required

Abbreviations: MDR, multidrug resistant; MRSA, methicillin-resistant *Staphylococcus aureus;* SARS, severe acute respiratory syndrome; TB, tuberculosis; VRE, vancomycin-resistant enterococci; and VRSA, vancomycin-resistant S *aureus*.

In temperate climates, human disease outbreaks peak in late summer and continue into fall; however, year-round transmission is possible in the more southern states. WNV is endemic in all of the continental United States and in the District of Columbia (as well as Canada and Mexico). The mosquito responsible for transmission to humans is primarily the *Culex* species; however, other types of mosquito can carry the disease.

The incubation period of WNV ranges from 3 to 14 days. Most infections are subclinical and patients remain asymptomatic. A febrile viral syndrome develops in approximately 20% of infected persons; only 50% of those infected will seek medical attention. The signs and symptoms of WNV infection are variable, but they frequently include sudden onset of fever with malaise, anorexia, nausea, vomiting, eye

pain, headache, and myalgia, which last 3 to 6 days. Symptoms may also include rash, lymphadenopathy, fatigue, and arthralgias. Approximately 1 in 100 infections result in meningitis or encephalitis. Advanced age is the most significant risk factor associated with severe neurologic sequelae following infection. There is no cure for WNV encephalitis. Infections are treated symptomatically, and supportive therapy is important in the more severe illnesses.

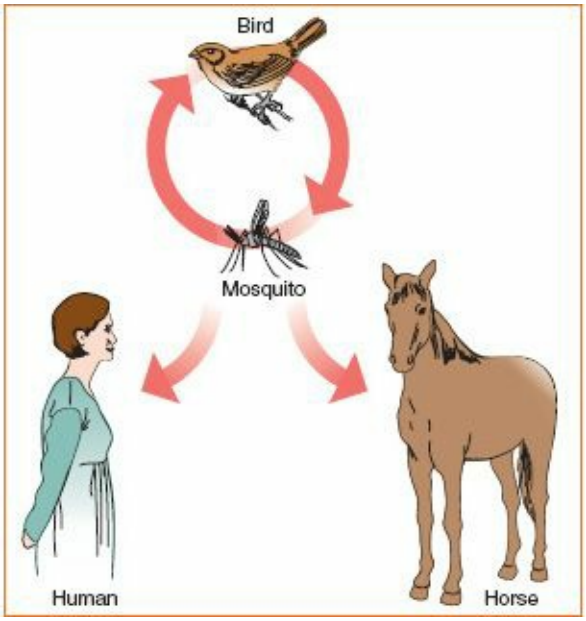

Figure 19-8 Transmission cycle of the West Nile virus.

A high index of clinical suspicion is required for prompt diagnosis. Arboviral disease should be included in the differential diagnosis in any older adults who present with the sudden onset of unexplained encephalitis or meningismus in the late summer or early fall. The presence of local WNV enzootic activity or other human cases should amplify the level of suspicion. Because WNV infections have occurred in all ages and because year-round transmission is possible in southern states, any person presenting with unexplained encephalitis or meningismus should trigger suspicion of WNV infection.

WNV is most efficiently diagnosed by detection of IgM antibody to WNV in the CSF via antibody-capture enzyme-linked immunosorbent assay. WNV-positive IgM antibody present in the CSF strongly suggests WNV infection of the central nervous system because IgM antibody does not cross the blood-brain barrier. The CSF may reveal normal to elevated leukocyte levels, with lymphocytes predominating, an elevated protein level, and normal glucose levels. Serum samples also have a high rate of positive results for WNV IgM antibody.

WNV encephalitis has been added to the nationally notifiable arboviral encephalitides. The timely identification of acute WNV has substantial public health implications, and case reports will likely prompt augmentation of the public health response to reduce the risk of additional human infections.

Integrated vector management programs are the best method for control and prevention of human infection. Mosquito control measures should emphasize the elimination of standing water because mosquito development consists of three aquatic stages: egg, larva, and pupa. Therefore, elimination of their environmental niche is an effective way to reduce the mosquito population without the use of insecticides.

CCTPs working in the outdoors should be instructed to apply an insect repellent while engaging in prolonged extraction or search-and-rescue activities. The CDC recommends repellents that contain the active ingredient *N*,*N*-diethyl-m-toluamide (DEET). Increasing the concentration of DEET in the repellent does not increase the strength, but it does increase the duration of effect. DEET concentrations of 50% or more do not increase the duration of protection significantly and, therefore, little benefit is gained by

using them. DEET does not kill mosquitoes; rather it prevents them from detecting human skin odors and, thus, finding a blood meal. Peak mosquito biting times are dawn, dusk, and early evening. Long pants and long-sleeve clothing provide an added measure of protection.

Signs and Symptoms

West Nile Virus Infection

- Sudden-onset fever
- Malaise
- Anorexia
- Nausea
- Vomiting
- Eye pain
- Headache
- Myalgia
- Rash
- Lymphadenopathy
- Fatigue
- Arthralgias

Differential Diagnosis

West Nile Virus Infection

- *Borrelia burgdorferi* infection
- Brain abscess
- Brain tumor
- Dengue fever
- Guillain-Barré syndrome
- Leptospirosis
- Meningitis
- Paraspinal epidural abscess
- Stroke
- Tick paralysis
- Eastern equine encephalitis
- Other infectious encephalitis

Transport Management

West Nile Virus Infection

- Provide supportive care.

Helicobacter Pylori

Helicobacter pylori bacteria, also discussed in Chapter 16, are spiral-shaped bacteria that infect the stomach and duodenum and are the causative agents of 80% to 90% of peptic ulcers. *Helicobacter pylori* are capable of evading the immune response (largely the result of a protective mucous covering), resulting in the bacteria being able to continue colonization and transmission. Once ingested, the bacteria embed within the mucosa of the stomach to protect themselves from the acidic environment of the gastrointestinal tract. Additionally, these bacteria secrete the enzyme urease (which converts urea and acid into the strong bases ammonia and bicarbonate), thus creating a localized environment favorable for survival and growth. Furthermore, the spiral shape and corkscrew movements of the bacteria enable them to penetrate the stomach's protective lining, resulting in irritation of the lining by stomach acid. The end result is an ulcer that forms at the site of continued irritation. In adults, this is usually a chronic condition that will not heal without specific therapy.

Helicobacter pylori bacteria are acquired via oral ingestion and are transmitted mainly within families during early childhood. Transmission is predominantly direct from person to person via vomitus, saliva, or feces. However, other modes of transmission, such as food, water, nonhuman primates, and other animals, may be responsible for transmission in developing countries. Improved sanitation conditions play an important role in reducing transmission of the bacteria.

Patients infected with *H pylori* may be asymptomatic or may exhibit signs and symptoms of a mild to severe gastritis. The predominant symptom is abdominal discomfort and is described as a dull, gnawing ache. This pain may come and go for several days to several weeks. The pain commonly occurs 2 to 3 hours after eating or in the middle of the night when the stomach is empty and is usually relieved by eating. Other signs and symptoms include weight loss, poor appetite, bloating, burping, nausea, and vomiting.

The diagnosis of *H pylori* infection may include serologic blood tests, a urea breath test, stool antigen assays, and tissue samples obtained through endoscopic biopsy. Testing is important to differentiate whether the ulcer was caused by *H pylori* or by the use of nonsteroidal anti-inflammatory drugs, because the treatments vary based on cause.

The goal of *H pylori* treatment is the complete elimination of the organism from the body. Once this is accomplished, reinfection rates are low. Treatment of *H pylori* requires triple therapy and consists of one antisecretory agent and two antimicrobial agents.

Signs and Symptoms

***H pylori* Infection**

- Abdominal discomfort described as a dull, gnawing ache
- Abdominal pain that occurs 2 to 3 hours after eating or in the middle of the night when the stomach is empty; it is usually relieved by eating
- Weight loss
- Poor appetite
- Bloating
- Burping
- Nausea and vomiting

Differential Diagnosis

H pylori Infection

- Cholecystitis
- Chronic active gastritis
- Drug-induced dyspepsia
- Esophagitis
- Gastroesophageal reflux disease (GERD)
- Ischemic heart disease
- Pancreatitis
- Peptic ulcer disease

Transport Management

H pylori Infection

- Provide supportive care.
- Continue IV administration of any medications started by the sending facility.

Strategies for the Treatment of Infectious Disease

As discussed earlier in this chapter, microorganisms possess unique properties that allow them to invade their human hosts, evade immune detection, and set up infection. These properties are exploited when developing an agent to inhibit their growth or eliminate them from the body. The goal of any such therapy is to eradicate the pathogen without causing undue harm to the host. The following section explores how antibiotics, antivirals, and antifungal agents are designed to target the unique organelles of each class of organism.

Antibiotics

Antibiotics are compounds that are produced by bacteria or fungi that inhibit the growth of or kill other bacterial organisms. Compounds that inhibit bacterial growth are called **bacteriostatic**, and compounds that kill bacteria are termed **bacteriocidal**. Synthetic antimicrobials are designed using the "magic bullet" concept, exploiting the differences between human and microbial cells. The beta-lactam antibiotics include penicillin, ampicillin, amoxicillin, and carbenicillin, among others. These bacteriocidal drugs act on gram-positive bacteria by competitively inhibiting the enzyme, transpeptidase, which catalyzes peptidoglycan cell wall synthesis.

Different families of bacterial cells have unique components that are targeted by different classes of antibacterial drugs. All antibacterial agents follow the same basic principle of targeting bacterial proteins, processes, or cellular components that are not found in human cells. Even though human cellular components are not the targets of antimicrobial therapies, there are adverse effects that must be considered when choosing the appropriate therapy.

The most accurate method for determining sensitivity and resistance of a specific bacterial strain is to perform an antimicrobial susceptibility test. A bacterial isolate is applied to the surface of an agar plate. Then, small disks impregnated with various antibiotics are placed on top of the agar and the plate is incubated for 24 hours. If there is a zone of clearing around the disk, the bacteria is said to be sensitive to the agent. If the growth of the bacteria is unaffected by the disk, the bacteria is said to be resistant to that agent.

Antivirals

Currently, viruses cause 60% of all infectious illnesses, with approximately 90% of the US population experiencing a viral infection during any given year. In light of these numbers, relatively few treatment modalities have been developed to care for patients with a viral illness. The difficulty lies in isolating the steps of the virus replication because they are intimately involved with the normal synthetic processes of the human host cell. The medications that are effective rely on the ability to interrupt the viral replication cycle at a specific step. Virus replication takes place during an incubation period, when the patient is asymptomatic and, thus, not likely to be taking any antiviral medication. The virus can also become latent within the body, rendering it almost impossible to eradicate.

There are three classes of antiviral medications: nucleoside analog, enzyme inhibitors, and interferon. Nucleoside analogs such as acyclovir and AZT are used to treat herpesviruses and HIV infection, respectively. Enzyme inhibitors are used to treat influenza and consist of drugs such as indinavir and zanamivir. Alpha-interferon is used to treat viral hepatitis. Because of the limited number of medications available to treat active viral infections, prevention of viral infections in health care workers is best achieved through available vaccination. Meanwhile, biochemists are working continuously to create novel medications to treat both viral and bacterial infections.

Antifungals

Human and fungal cells are similar in that both are eukaryotic. Because they are similar in makeup, finding agents that affect the fungal cell without harming the human cell is challenging and antifungal agents can produce toxic results when used systemically. A primary difference between human and fungal cells is their membrane components. The fungal cell membrane contains ergosterols, whereas the human cell membrane contains cholesterol. This structure is different enough for it to be a target of antifungal therapy. Polyene compounds bind with the sterol in the fungal cell membrane, making the membrane less fluid and more susceptible to rupture. As a result of the change in fluidity, the cell membrane leaks, loses its integrity, and leads to cell death. There are other antifungal therapies available, but all are a variation on this theme. They target pathways or enzymes that are specific to fungal cells.

The Post-Antibiotic Era

Before the advent of antibiotic agents, bacterial infections were the cause of significant morbidity and mortality. In 1900, the three leading causes of death in the United States were by infectious diseases. In 2006, the leading causes of death were the result of noninfectious diseases: heart disease, cancer, and stroke. Today, the only infectious diseases ranked in the top 10 leading causes of death are lower respiratory tract infections and sepsis. However, many scientists worry that we are entering the "Post-Antibiotic Age" because of the emergence of antibiotic-resistant bacteria. Virtually all important pathogenic bacteria have demonstrated resistance to current Food and Drug Administration–approved antibiotics. Antibiotic resistance is predicted to be one of the world's most pressing public health problems in the 21st century.

Three important factors contribute to the emergence of resistant bacteria within a given population or community:

- Misuse of antibiotics
- Poor infection control
- Importation or intrusion of already resistant strains

The continual use of antibiotics paradoxically leads to declining effectiveness through selective pressures and the development of resistance because increasing resistance causes a concomitant decrease in the therapeutic options for eliminating infections. In the critical care environment, there are many opportunities for contamination and subsequent cross-transmission of resistant bacteria. Infection control measures, such as handwashing, patient isolation, and proper disposal of contaminates, are important in breaking the cycle of transmission. As a CCTP, it is important to always be aware of how your actions can either hinder or hasten the spread of disease.

Antibiotic resistance occurs when the bacteria acquire new properties through mutation, protein alteration, or conjugative transfer of plasmid-encoded resistance factors. Resistant organisms coexist with healthy skin or gastrointestinal flora. When antibiotics are used to treat infection or used inappropriately for viral infections, the normal, protective flora is eliminated and the resistant strains dominate. This relative decrease in the sensitive strains and increase in the resistant strains leads to selective pressure on the weaker protective bacteria. The bacteria that are not eliminated are stronger (ie, more resistant to antibiotics), making it more difficult to cure the patient, and these resistant strains move on to cause infection in the next host. When this theme is applied on a grander scale to the critical care setting where the use of antibiotics is routine and infectious sources are plentiful, the magnitude of the problem becomes apparent.

Infection control is critically important in thwarting the development of new resistant strains and preventing the spread of currently resistant strains. Health care workers are a major source of cross-

infection between critically ill patients. Handwashing is the single most important act that a health care provider can perform to reduce the spread of infection. However, even under the best circumstances, compliance with handwashing is low. It is even more difficult to accomplish in the critical care transport environment, but it is the choice of the CCTP to either practice proper hand sanitation methods when providing care or allow the hands to be vectors for transmission of infectious disease. Another important method for reducing the spread of resistant bacteria is the isolation of infected patients. Methods of isolation include geographic isolation, negative-pressure isolation, contact precautions, and barrier isolation, discussed later in this chapter.

Antibiotics should be prescribed only when they are likely to be beneficial. The choice of antibiotic should be appropriate—using a drug that will eliminate the pathogen most likely to be causing the infection or, better yet, using definitive culture and sensitivity testing to identify the infecting bacterial strain and then combating it with the correct drug. The prescription should be for the appropriate dose and duration of treatment. Using a drug for longer than is required to eliminate the offending bacteria does not offer any additional benefit to the patient.

Each year in the United States, 160 million prescriptions are dispensed for antibiotics, half of which are likely prescribed unnecessarily for viral upper respiratory tract infections. The public must be made aware of the role they play in promoting the emergence of resistant strains and, thus, must be educated to reduce demands for inappropriate prescriptions. Although completely ineffective against viral infections, tens of millions of antibiotics are prescribed for viral illnesses. Time pressure on physicians and patients alike, diagnostic uncertainty, and demand from uninformed patients contribute to the continued inappropriate use of antibiotics. To ensure that patients return to their practice, physicians are under pressure to prescribe, but prescribing for the sole purpose of convenience and patient demand is never appropriate. Patients must be instructed to take antibiotics as directed and for the full duration of the intended treatment. Once symptoms of the illness are relieved, patients are likely to stop taking the drug, but the infection may not be cured. Infections that are treated incompletely kill the weaker strains, leaving the stronger or antibiotic-resistant strains unaffected, which then contributes to the emergence of resistance by selection. Patients should be warned against the dangers of using "leftover" antibiotics prescribed from prior infections to treat their current ailments. First, the symptoms may not be caused by a bacterial infection, and if there is an actual bacterial infection, the drug may be completely ineffective against this causative bacterial strain and may cause the patient to become sicker. Also, partially treating bacterial infections with antibiotics may make it more difficult to isolate, identify, and perform sensitivity testing on the culprit microbe, making treatment decisions all the more difficult for the health care provider.

Antibiotic resistance can cause significant morbidity and mortality in persons infected with previously easily treatable infections. Staphylococci are one of the most common causes of community- and hospital-acquired infections. *Staphylococcus* commonly colonizes on the skin and the nares of healthy people. If *Staphylococcus* causes a simple infection, most people can be successfully treated with beta-lactam antibiotics. However, *Staphylococcus* also causes serious infections, such as postoperative infections or pneumonia; during the past 50 years, treatment of these infections has become increasingly difficult because of the emergence of resistance.

Methicillin-Resistant *S aureus*

Methicillin-resistant *S aureus* (MRSA) is a gram-positive, coagulase-positive, nonmotile coccus that produces an altered penicillin-binding protein that confers resistance to beta-lactam antibiotics, including methicillin. Most strains of MRSA are also resistant to multiple classes of antibiotics, making the infection difficult to control. MRSA can colonize a variety of tissues causing infections such as cellulitis,

cutaneous abscesses, wound infections, osteomyelitis, septic arthritis, endocarditis, pneumonia, and septicemia.

MRSA was first detected in Europe and Australia in the 1960s, and it was detected in the United States 10 years later. MRSA has gradually become a major player in resistant nosocomial infections. Humans are the major reservoir of MRSA. Most carriers are colonized in the nares, pharynx, and skin. High rates of MRSA are found among residents of long-term care facilities, but serious infections with MRSA are more prevalent in the acute care setting. Risk factors for colonization include dialysis, diabetes, use of injectable drugs, chronic skin conditions such as decubitus ulcers, and a history of prior antibiotic use. MRSA is also emerging as a community-acquired pathogen, which is MRSA acquired by persons who have not been hospitalized recently nor undergone a recent medical procedure.

Transient contamination of the hands of health care providers results in the transmission of resistant organisms to other patients. MRSA is spread by direct contact with patients who are either colonized or infected with MRSA or by contact with contaminated objects. Patients with known MRSA infections should be placed on contact precautions until they have been successfully treated and the results of their nasal and rectal swabs are negative. As always, CCTPs should avoid direct contact with wounds by wearing gloves for all dressing changes and avoiding direct contact with a patient with a known MRSA infection

Vancomycin is the drug of choice for treating MRSA infection. An alternative to vancomycin for the treatment of MRSA skin infections and nosocomial pneumonia is linezolid. The advantage to using linezolid is that it is available in an oral form, whereas vancomycin is not absorbed in the gastrointestinal tract. Until recently, all strains of MRSA have been sensitive to vancomycin; however, vancomycin-resistant *S aureus* has also emerged and resulted in deaths in the United States.

The emergence of vancomycin-resistant MRSA poses a serious threat to patients and health care workers; if encountered, the patient should be isolated and the infection should be reported to public health authorities. Implement contact precautions, minimize the number of persons with access to the colonized/infected patient, and dedicate one-on-one care for the colonized patient. Avoid transferring infected patients between or within facilities. If a transfer is necessary, fully inform the receiving institution of the patient's status.

Vancomycin-Resistant Enterococci

Enterococcus is a gram-positive bacterium normally found in the intestinal tract (normal flora); however, antibiotic-resistant species of *Enterococcus* have become a major cause of nosocomial infection in the United States. These bacteria rarely cause illness in healthy people but can cause serious infections in immunocompromised, postoperative, and other seriously ill persons. Because enterococcal bacteria colonize the intestinal tract, transmission is largely the result of direct person-to-person contact or indirect contact (eg, door knobs and toilet seats) from inadequate handwashing after evacuation of feces.

Two types of antibiotic resistance are found in enterococci—intrinsic and acquired. Intrinsic resistance is a natural resistance found in the bacteria and is caused by the vanC gene. Acquired resistance is genetically transferred from another resistant organism. The two responsible genes are transmissible to other bacteria by plasmids and transposons, and they confer resistance to much higher levels of vancomycin. Another critical consideration is that the vancomycin-resistant genes present in VRE have the potential to be transmitted to other gram-positive organisms, such as *S aureus*. For these reasons, health care facilities should test all persons at high risk for VRE and, if found positive, patients should be isolated and standard precautions should be used. Patients can carry VRE organisms for long periods of time, particularly in their stool. Therefore, the CDC and the Hospital Infectious Control Practices Advisory Committee recommend that isolation should continue until the patient has three sets of negative cultures from all appropriate sites taken at least 1 week apart.

In the critical care environment, there are many opportunities for contamination and subsequent cross-transmission of resistant bacteria. When transporting VRE-positive patients, infection control measures such as handwashing, patient isolation, use of protective barriers such as gloves and gowns, and the proper disposal of contaminates are important in breaking the cycle of transmission.

Rickettsial Diseases

Rickettsial diseases are transmitted via tick bite and, therefore, patients with these diseases are not considered infectious by human-to-human contact (however, known cases of transmission have occurred via blood transfusion from an infected but asymptomatic donor). There are three major diseases classified as tick-borne rickettsial diseases—Rocky Mountain spotted fever, ehrlichiosis, and anaplasmosis. Of these diseases, the highest mortality rate is seen with Rocky Mountain spotted fever. Early signs and symptoms of these tick-borne diseases are generally mild and nonspecific, making diagnosis problematic; however, Rocky Mountain spotted fever should be considered a possible diagnosis in all patients presenting with fever and having suspected or known tick exposure (outdoor activities during April through September in North America). In particular, children and young adults are at risk because of their increased outdoor activity. Despite its name, Rocky Mountain spotted fever is seen mostly in the southeastern United States (especially North Carolina) followed by two midwestern states (Missouri and Tennesee). **Table 19-8** compares initial signs and symptoms of anaplasmosis, ehrlichiosis, and Rocky Mountain spotted fever.

Rocky Mountain spotted fever begins acutely with fever accompanied by one or more of the following: headache (usually severe), rash, muscle aches, nausea and vomiting, and malaise. Patients may also exhibit neurologic symptoms resembling meningitis. Children may present with abdominal pain, altered mental status, and conjunctival injection (bloodshot eyes). Within a few days after the initial fever, a petechial rash develops on the hands and feet, and may spread to the rest of the body in about half of the adults and most of the children. The skin on the hands and feet of some patients will slough off about a month after a severe petechial rash occurs. Of patients with tick-borne rickettsial diseases, 50% or more require hospitalization, and some patients will experience long-term complications such as renal failure, myocarditis, meningoencephalitis, hypotension, ARDS, thrombocytopenia, and multiple organ failure.

Diagnosis is determined using a serum antibody test or polymerase chain reaction test or by obtaining a biopsy specimen of the rash. If rickettsial disease is suspected, treatment with doxycycline (or a similar antibiotic) should be initiated immediately and not delayed by waiting for results from diagnostic tests.

TABLE 19-8 Initial Signs and Symptoms of Tick-Borne Rickettsial Diseases

Anaplasmosis	Ehrlichiosis		Rocky Mountain Spotted Fever
Anaplasma phagocytophilum (anaplasmosis)	*Ehrlichia chaffeensis* (ehrlichiosis)	*Ehrlichia ewingii* (infection)	*Rickettsia rickettsii*
Fever	Fever	Fever	Fever
Headache	Headache	Headache	Headache
Malaise	Malaise	Malaise	Malaise
Muscle aches	Muscle aches	Muscle aches	Muscle aches
Vomiting		Vomiting	Vomiting
		Nausea	Nausea
			Loss of appetite
Rare rash	Rash in < 30% of adults and approximately 60% of children	Rare rash	Maculopapular rash approximately 2-4 d after onset of fever in 50%-60% of adults (and > 90% of children); might involve the palms and soles

Source: Centers for Disease Control and Prevention. Available at:

http://www.cdc.gov/ticks/symptoms.html. Accessed July 13, 2009.

Prevention of tick-borne diseases includes use of insect repellents containing DEET and wearing light-colored clothing (to make ticks more apparent). If possible, long sleeves and long pants should be worn and pants should be tucked into socks when outside—particularly when walking through grassy areas. The body should be inspected for ticks after being outdoors.

Removal of embedded ticks should be done with tweezers or gloved hands (avoid removal with bare hands). Grasp the tick as close to the skin as possible and lift the tick with enough force to "tent" the skin surface. Hold the tick in this position for a minute or until the tick lets go and either save the tick for analysis (in a plastic bag placed in the freezer) or dispose of the tick by flushing it down a drain. To avoid contamination from body fluids, do not crush or squeeze the tick. After removal, thoroughly wash the area of the tick bite with soap and warm water and apply rubbing alcohol. Wash hands with soap and water.

Bioterrorism

Bioterrorism (biological weapons) is an emerging threat to the health and safety of the community. There are many countries that currently have or are developing offensive biological weapons programs. With the events of September 11, 2001, and the continued threat of terrorism in the United States, there is increasing concern over the possibility of terrorists using biological agents against civilian populations. Therefore, local, state, and federal officials have implemented education and training of first responders in preparation for potential intentional biological disasters **Figure 19-9**.

The goals of a bioterrorism emergency response should be to contain the agent, protect critical personnel, and manage the health consequences of the event. Containment of a contagious disease resulting from deliberate release of an infectious agent requires that it has to be recognized as a non-natural event based on a presentation that differs from the usual background of the endemic infectious diseases that affect a population. The first cases are likely to go unrecognized because most agents likely used present with initial nonspecific symptoms such as fever, malaise, and headache; these types of findings make the biological attack difficult to recognize in the initial stages of infection.

Figure 19-9 Homeland security or other government agency training responders work to prepare for bioterrorism.

The covert release of any agent could take place over a period of days to weeks and it may be even longer before the initial cases of infection are identified, depending on the mode of transmission and incubation period of the agent. As the duration of time between exposure and symptoms increases, so does the likelihood that the agent will be distributed over a wide geographic area and result in an epidemic. It

is also possible that the deliberate release of a biologic agent will be announced as such and, therefore, poses a different set of problems for the emergency response system. In this case, there would likely be large-scale panic with many individuals calling on emergency services without actual need, overwhelming health care systems and preventing the efficient use of medical resources. The challenge would then be to differentiate those actually exposed to the agent from those whose fear of the agent has caused them to seek care. To date, a number of announced events have occurred in the United States since 1998, but these events were later determined to be hoaxes. In any event, the CCTP must be prepared to deal with either scenario. Knowledge of epidemiologic principles is crucial in differentiating between the presentation of a naturally occurring disease and an unusual, non-natural event that should raise concern that a bioterrorist event has been initiated and that local emergency response systems should be activated.

The CCTP must maintain an index of suspicion regarding a potential threat because early symptoms may be nonspecific, but early treatment will be required to prevent severe injury or death. Before approaching a patient, CCTPs must always take steps to protect themselves first, using standard precautions and additional precautions outlined in **Table 19-7.** In addition, immunologic protection is the responsibility of the CCTP. Stay current on required and recommended vaccines, keeping in mind what is appropriate for a particular patient population, and consider the appropriateness of being vaccinated against agents that may be exploited in bioterrorism attacks. For the most updated information, visit the Web site of the Centers for Disease Control and Prevention at http://www.cdc.gov and search for the Infection Control Guidelines.

Exposure and Protection

CCTPs have unique occupational exposures to a variety of biologic, chemical, and physical hazards. CCTPs are in contact with critically injured and very sick patients, often in uncontrolled settings under emergent conditions. Resuscitating the critically ill is associated with being in contact with potentially infected body fluids 31% of the time. CCTPs also provide care to trauma patients with open wounds that may be actively bleeding. Managing trauma patients is associated with exposure to body fluids 80% of the time. In the chaos of providing care for life-threatening diseases in unpredictable or dangerous conditions, well-established exposure prevention procedures are essential for protecting the health of the CCTP.

Figure 19-10 All sharps must be placed into containers that are puncture-resistant, closable, and leakproof, and that contain the biohazard symbol.

The health risks associated with being a CCTP are not simply limited to blood or body fluid exposures; infectious agents pose a significant risk. The greatest risk comes from patients who are actively infectious but are undiagnosed. Activities and tasks during which exposure may take place include the following:

- Patient assessment
- Airway management
- Establishing IV access
- Clean-up of the vehicle and equipment
- Assisting in childbirth
- Contact with blood or body fluids

Prophylactic measures must be taken to prevent infection prior to exposure. There are several effective ways to reduce the risk of contracting an infectious agent or transmitting infections to patients, but disease prevention is best achieved when the CCTP is fully prepared and properly equipped.

The use of protective clothing and PPE reduces exposure risks. Employers are required to provide their employees with the proper protective equipment at no cost to the employee. The proper handling of contaminated medical devices, such as sharps, soiled bandages, linens, and reusable medical equipment, is essential in reducing the risk of indirect transmission of infectious diseases **Figure 19-10**. In addition, the consistent implementation of isolation precautions is required to control the spread of infections to others.

Immunizations

The CDC recommends that all emergency medical care providers be vaccinated against preventable infectious diseases. The immunization recommendations are based on the risk of exposure. All new employees should not begin taking an active role in caring for patients until their immunization status is reviewed and up to date.

The current recommendations for emergency medical care providers include the following:

- Measles
- Mumps
- Rubella
- Varicella (immunization or proof of prior infection)
- Hepatitis B
- Annual influenza
- Pertussis
- Tetanus
- Polio
- Diphtheria
- Pneumococcal vaccine
- Meningococcal vaccine

All employers of health care providers exposed to blood or other infectious materials during the course of performing work-related duties are required to provide the hepatitis B vaccine to their employees at no cost. Ideally, the vaccination should start during the training period, before patient contact is initiated. The vaccine is administered at 0, 1, and 6 months, with 95% of healthy adults developing protective titers on completion of the three scheduled immunizations. If an employee has already received the full series of vaccinations, an antibody titer should be drawn to verify immunity. The duration of protection is unknown, but it lasts at least 7 years in healthy young adults. If there is a medical contraindication to the vaccine, this should be documented and readdressed if the medical condition

changes. Any employee who declines the hepatitis B vaccine must sign a waiver releasing the employer from responsibility for the work-related exposure.

The influenza vaccine is recommended for CCTPs because the influenza virus easily spreads through close contact with infected individuals via small aerosolized particles. Although most healthy adults are able to recover from influenza without significant morbidity, sick and elderly patients do not have the same immunologic reserve. On that basis, the influenza vaccine is recommended for CCTPs to prevent spread of the virus to their patients and thereby prevent significant morbidity and mortality.

Measles, mumps, and rubella—although rare—can cause significant disease in susceptible individuals. The risk of contracting such infections is increased 13 times for the CCTP over that of the general public. Most adults in the United States have been immunized, but immunity should be confirmed via a serologic titer. This is especially important for women of childbearing age to prevent the advent of rubella syndrome in pregnancy.

The varicella vaccine is important for CCTPs for two main reasons. First, varicella is highly contagious, and varicella infection can cause serious complications in normally healthy adults. Second, vaccination of CCTPs is important to prevent the transmission of the disease to their patients. Individuals with a positive history of chickenpox as children are considered immune, but if one's medical history of varicella is in doubt, he or she should undergo immunologic confirmation of serologic status. If the person is seronegative and has no contraindications to the vaccine, the vaccine should be administered.

All CCTPs are encouraged to discuss immunizations with employer health personnel or their own private physicians before vaccinations are given to ensure that there are no contraindications to receiving specific vaccines.

Needlestick Injuries

Needlestick injuries do occur. By law, all needlesticks and other injuries with sharps contaminated by blood must be reported to the employer. Reporting sharps injuries is necessary when the injury occurs in the course of performing work-related duties and when the object causing the injury is contaminated with blood or other potentially infectious materials. It is not necessary to report sharps injuries that occur with clean objects. When reporting the injury, it is important to characterize it by type of injury (ie, splash, spill, stick, or cut) and by the type and amount of contaminating fluid (eg, saliva, semen, ascites, or blood). It is also important to determine the infectious status of the source patient. The Ryan White Act enacted in 1990 mandated that all EMS personnel could request testing of the source patient to determine if they had been exposed to a life-threatening disease and allowed the results to be given to the affected employee. When the act was renewed in 2006, the clauses protecting emergency personnel were removed by Congress, making it much more difficult for the CCTP to gain access to this critical information.

The risk of a CCTP acquiring an infectious disease from a contaminated sharps exposure is proportional to infectivity of the source and type or severity of the exposure. The larger the number of viral particles exposed to, the greater the risk. The estimated risk of HIV transmission is 0.33% for hollow-bore needlesticks and 0.09% for mucosal contact; there are no known cases of HIV transmission for intact skin exposures. The following factors increase the risk of acquiring HIV after a needlestick injury:

- Deep injury
- Needle placed directly in a vessel (artery or vein)
- Visible contamination with source patient's blood
- Terminal illness in the source patient

Universal Precautions

Universal precautions are recommended to prevent transmission of bloodborne pathogens such as HIV and hepatitis B and C. In these cases, all blood and body fluids are considered to be a source of infectious material. These precautions stipulate the use of gloves, gowns, or aprons (or uniforms) and masks or other eye protection to reduce exposure of the skin (especially abraded or broken skin) and mucous membranes. CCTPs should be especially cautious with needles and sharps used on infected patients to prevent parenteral exposure.

The first response to a percutaneous exposure should be to wash the wound thoroughly with soap and water. If water is not available, an alcohol-based hand cleanser is a reasonable alternative. After a significant exposure, the source patient and CCTP both should be tested for baseline testing and the exposed CCTP should receive follow-up testing at 6 and 12 weeks and at 6 months. During the period following exposure, those potentially exposed to an infectious agent such as HIV should refrain from donating blood products, refrain from sexual intercourse, or use condoms to prevent sexual transmission and avoid pregnancy. If breastfeeding, this should be discontinued. All CCTPs who have been exposed to potentially infectious body fluids should seek prompt medical attention. The decision to start postexposure prophylaxis should be discussed with your health care provider, and the risks and benefits of such treatment should be considered. The risk of contracting HIV from exposure to contaminated body fluids is very low, about 3 per 1,000 for needlesticks. The risk of seroconversion is reduced by approximately 80% by starting postexposure prophylaxis in a timely fashion. The side effects, complications, drug interactions, and proper dosing instructions should be discussed prior to initiating treatment.

Isolation Precautions

There are two levels of isolation precautions. The first level of precaution is designed for the care of all patients, regardless of their diagnosis, presumed infections, or immune status. The main goal of the first level is the successful control of nosocomial infection by facilitating the safe and proper handling of blood and other potentially infectious materials. These are the standard precautions, the major features of which are designed to reduce the risk of transmission of bloodborne pathogens from both known and unknown sources of infection. They should be universally applied to all patients. They protect against contact with blood, body fluids, and secretions, regardless of the presence of visible blood, and contact with nonintact skin and mucous membranes.

The second level of isolation precautions is the transmission-based precautions that consist of isolation precautions designed to be employed during the transport of all patients with documented infections or those suspected to be infected with highly transmissible or epidemiological significant infectious agents for which additional precautions above the first tier of standard precautions are warranted. The three means of providing transmission-based precautions are airborne, droplet, and contact precautions. These are to be used over and above the universal precautions and can be used singly or in conjunction, as the circumstances require.

Airborne precautions include use of an N95 mask by CCTPs, a negative pressure room (airborne infection isolation room) or transport vehicle, or the use of the exhaust fan during transport. Airborne precautions are designed to reduce the risk of transmitting infectious agents that are contained in airborne droplet nuclei or dust particles. Examples of agents carried in droplets, equal to or less than 5 μm, are tuberculosis (either pulmonary or laryngeal), measles, chickenpox, viruses that cause hemorrhagic fevers, and smallpox. Microbes found in droplet nuclei can remain viable suspended in the air for long durations, and can be dispersed over a wide area where they may be inhaled by susceptible hosts. Specialized air handling equipment is required to provide airborne precautions.

Droplet precautions include isolation of the patient and use of surgical masks by CCTPs (or keeping at a distance of 3′). Droplet precautions are designed to reduce the risk of transmitting infectious agents that are found in large-particle droplets (> 5 μm). Transmission via large-particle droplets requires close contact between the source of the infection and the susceptible recipient. Droplets are generated from infectious persons during coughing or sneezing, and during invasive procedures such as suctioning of the airway, intubation, or bronchoscopy. Unlike airborne droplet nuclei, large-particle droplets only travel short distances and are airborne only for a short time after being generated. Examples of infectious agents found in large-particle droplets include *S pneumoniae*, *Corynebacterium diphtheriae*, *Bordetella pertussis*, *N meningitidis*, *H influenzae*, the influenza virus, and *Yersinia pestis* (pneumonic plague). Droplet precautions should be implemented when caring for persons suspected or known to be infected with epidemiologically important pathogens known to be transmitted by infectious droplets. Eye protection (goggles, face shields, and safety glasses) is recommended for use while transporting patients known to carry infectious agents that can be spread via mucous membranes of the eye (conjunctiva), including *S aureus*, rhinovirus, adenovirus, herpes simplex virus, and any bloodborne agents such as hepatitis B and C viruses and HIV. These can be spread via respiratory droplets or by touching the patient and then touching the eyes with contaminated hands/fingers. Eye protection prevents introduction of infectious agents into the conjunctiva. (Note: Full-face respirators also provide eye protection.)

Contact precautions for CCTPs include isolation of the patient, use of a gown (or uniform) and gloves, and, if warranted, face protection. Contact precautions are designed to decrease the risk of transmission of infectious agents that can be transmitted by direct or indirect contact. Direct contact transmission is defined as skin-to-skin contact and subsequent transfer of an infectious agent from an infected person to a susceptible host. Contact transmission can occur during any patient care activity that requires physical contact between the CCTP and the infectious patient. Direct contact transmission can also occur between two patients, and between a patient and a family member. Indirect transmission is the transfer of infectious agents from an infected patient through an intermediate, most often inanimate, object touched by or used during the caring of the infected patient. Objects that transmit infectious agents to susceptible hosts are called fomites. Examples of epidemiologically important infectious agents that can be transmitted via contact include MRSA, VRE, *Clostridium difficile*, scabies, smallpox, and viruses that cause hemorrhagic fevers, and *Bacillus anthracis*. Contact precautions must be employed during the transfer of patients known or suspected to be infected with microorganisms that can be transmitted through direct or indirect contact. Equipment used on these patients during the course of a transport must be discarded or disinfected to interrupt the cycle of indirect transmission.

There should be a regimen in place for donning and removing PPE as established by the infection control staff **Figure 19-11**. In general, never touch your eyes, nose, or mouth with contaminated gloves or hands. After handling contaminated equipment, always wash your hands. If handwashing is not possible, use a waterless hand sanitizer and then wash hands as soon as it is practical.

It is likely that during the transport of critically ill patients from the scene to the tertiary facility, the etiology of any infectious disease will not be known. The risk of transmission is greatest before definitive diagnoses are made. Standard precautions should be implemented without fail on all transported patients. The use of transmission-based precautions should be implemented if there are any known risk factors or if suspicious symptoms or syndromes are observed. It is much better to err on the side of prevention, until the definitive diagnosis can be determined, than to try to contain an infectious disease outbreak after the fact. Certain clinical syndromes and conditions are red flags for which the empiric use of the second layer of precautions is warranted. In the case of interfacility transfers, the infectious agent may have already been identified. If so, it is imperative that this information be known and contact precautions implemented accordingly.

Handwashing

Handwashing is the single most important measure employed to reduce the person-to-person transmission of infectious agents **Figure 19-12**. However, washing your hands between patient transports is logistically difficult because sinks are not available on transport units. Therefore, the conscientious CCTP must make a concerted effort to wash between patients and after touching any body fluid. Waterless antiseptic agents can be used as a substitute for washing under certain conditions. The use of alcohol-based waterless handwashing antiseptic has been shown to improve rates of handwashing among health care workers. Therefore, transport units should be equipped with easily accessible dispensers of hand antiseptic to facilitate compliance in handwashing. Alcohol-based hand antiseptics are not effective against all infectious agents. *C difficile*, for example, is not killed by hand sanitizers. Therefore, handwashing with soap and water between transports is of paramount importance.

Personal Protective Equipment

Personal protective equipment is specialized clothing or equipment worn by an individual that provides protection from a potentially hazardous agent. PPE is required to be provided by the employer at no cost to the employee. It should be readily available and used consistently by the CCTP.

Clean, nonsterile, disposable gloves are standard equipment on critical care transport units. They must be donned by all personnel prior to initiating patient care. Gloves are worn by CCTPs for three important reasons. First, gloves provide a protective barrier to prevent gross contamination of the hands when exposed to blood and body fluids, and while working on patients with open injuries or nonintact skin. Second, gloves protect patients from the microbial agents that exist on the hands of CCTPs while performing invasive procedures. Third, gloves decrease the likelihood of transmission of microorganisms between patients or from fomites to patient, but only if the gloves are changed each time a new patient is encountered. Gloves should be removed if torn, punctured, or contaminated with gross blood. They should be promptly discarded after each use and before touching noncontaminated objects or surfaces. Once the gloves are removed, hands should be washed or sanitized with an alcohol-based waterless handwashing antiseptic.

SEQUENCE FOR DONNING PERSONAL PROTECTIVE EQUIPMENT (PPE)

1. GOWN

- Fully cover torso from neck to knees, arms to end of wrists, and wrap around the back
- Fasten in back of neck and waist

2. MASK OR RESPIRATOR

- Secure ties or elastic bands at middle of head and neck
- Fit flexible band to nose bridge
- Fit snug to face and below chin
- Fit-check respirator

3. GOGGLES OR FACE SHIELD

- Place over face and eyes and adjust to fit

4. GLOVES

- Extend to cover wrist of isolation gown

SEQUENCE FOR REMOVING PERSONAL PROTECTIVE EQUIPMENT (PPE)

1. GLOVES

- *Outside of gloves is contaminated!*
- Grasp outside of glove with opposite gloved hand; peel off
- Hold removed glove in gloved hand
- Slide fingers of ungloved hand under remaining glove at wrist
- Peel glove off over first glove
- Discard gloves in waste container

2. GOGGLES OR FACE SHIELD

- *Outside of goggles or face shield is contaminated!*
- To remove, handle by head band or ear pieces
- Place in designated receptacle for reprocessing or in waste container

3. GOWN

- *Gown front and sleeves are contaminated!*
- Unfasten ties
- Pull away from neck and shoulders, touching inside of gown only
- Turn gown inside out
- Fold or roll into a bundle and discard

4. MASK OR RESPIRATOR

- *Front of mask/respirator is contaminated—do not touch!*
- Grasp bottom, then top ties or elastics and remove
- Discard in waste container

Figure 19-11 An example of a regimen for donning and removing personal protective equipment. Courtesy of CDC.

Figure 19-12 Handwashing is critical to preventing the spread of infection.

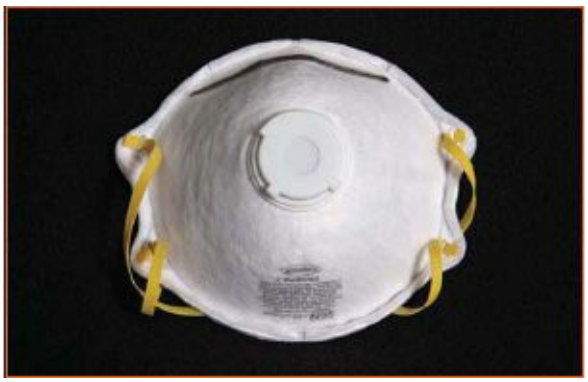

Figure 19-13 The N95 mask.

Masks, face shields, and eye protection are worn to protect mucous membranes of the eyes, nose, and mouth during procedures and patient care activities that are likely to generate splashes or sprays of blood, body fluids, or excretions. Not all masks offer sufficient protection from airborne infectious agents. The N95 mask must be worn when patients are known (or suspected) to be infected with agents that are transmitted by droplet nuclei. The N95 mask requires proper fitting, so the CCTP should be fit tested before the need arises **Figure 19-13**.

Gowns are used to protect the skin and prevent the soiling of clothing during procedures that are likely to generate splashes of blood or body fluids. However, they may not be appropriate garb for the CCTP because they may increase the risk of injury to the provider if trying to don the gown while in a moving vehicle. Also, if working in a small space, the gown may catch on equipment. If clothing or the gown becomes soiled, it should be removed as quickly as possible and disposed of properly, and the provider's hands should be washed promptly after removal to avoid the transfer of contaminants to the patient or the environment.

The two types of allergic reactions to latex are type I and type IV hypersensitivity reactions. Type IV is the more common and less debilitating of the two reactions. It is a cellmediated delayed reaction causing redness, chafing, and blisters to develop in areas of contact. Type I hypersensitivity is the more dangerous allergic reaction. It is antibody-mediated, specifically IgE, and causes the immediate and rapid development of symptoms of rhinitis, pruritus, and urticaria. If this reaction is not immediately recognized and treated, anaphylaxis may develop.

The prevalence of type I latex allergy in the general population is estimated as 4% to 8%. The prevalence of type IV latex allergy is not known. The risk factors for latex allergy are a history of atopy, spina bifida, and frequent exposure. Latex allergy can be devastating to CCTPs because it interferes with their ability to work safely among patients, thus these persons should wear nonlatex gloves such as nitrile or vinyl.

Other Exposure Prevention Practices

It is important to notify the accepting institution of the nature of the patient's illness or potential infectious diseases so that the receiving facility can arrange for proper isolation. This should be communicated by the transferring facility, but it is always a good policy to notify the receiving facility. Patients with epidemiologically important infections should be sufficiently isolated from other patients.

Patients themselves should be enlisted in the effort to prevent transmission by educating them on methods to prevent spreading infectious organisms to other vulnerable individuals. They can wear masks while being transported between or within medical facilities. They can cover their mouths while coughing, frequently wash their hands, and refrain from sharing drinking cups or utensils with family and friends.

Work Practice Controls

Control of the work environment and practices, when used consistently, reduces the risk of contamination and subsequent transmission of infectious agents. The prohibition of eating, drinking, smoking, applying cosmetics or lip balm, and handling of contact lenses in the patient work areas helps to reduce the risks of uncontrolled exposures. Actions that cause splattering of fluids should be avoided. All single-use items should be disposed of immediately after they are used. Contaminated gloves must be removed before driving the ambulance and touching the steering wheel.

The proper handling of sharps is another method for controlling exposure risk in the work environment. Do not bend, remove, or recap needles. If there is no choice but to recap a needle (ie, no sharps receptacle is available, which in practice, should never occur), the one-handed "scoop" technique should be used. Shearing or breaking of needles is prohibited. Dispose of sharps in an appropriate container as soon as possible after use. Sharp containers should be easily accessible, puncture-resistant, labeled or color-coded, and leakproof. They should not be filled beyond capacity and should be disposed of as medical waste.

Contaminated work surfaces must be decontaminated with an Environmental Protection Agency–registered disinfectant on completion of the transport. All equipment used during the course of the transport must also be decontaminated. All bins, cans, and reusable receptacles must be decontaminated on a regular schedule and emptied after each use. Contaminated laundry should be placed into leakproof bags, labeled, and stored until proper disposal is possible. Regulated waste must be placed in closeable, leakproof containers designed for disposal. Regulated wastes include semiliquid blood or other potentially infectious materials, contaminated items that would release blood if compressed, and items that are caked with dry blood, contaminated sharps, and biological wastes. If droplet precautions are required, the transport vehicle should be equipped with negative-pressure exhaust in the patient compartment. If this is not possible, the transport vehicle windows should remain open with the ventilation system set on "high" to move the air from within the vehicle to the outside.

Table 19-9 lists resistance of infectious organisms to disinfectants. Low resistance to disinfectant does not mean the organism is not dangerous. It simply means some organisms can be killed easier than other organisms. Assume that *all* patients transported carry infectious pathogens and take the necessary steps to decontaminate the equipment and vehicle prior to the next transport. There are three levels of disinfection:

- **High-level disinfection**, used on inanimate objects to kill all microorganisms. This does *not* kill bacterial spores.
- **Intermediate-level disinfection**, used on inanimate objects to kill *M tuberculosis*, vegetative bacteria, most viruses, and most fungi. This does *not* kill bacterial spores.

- **Low-level disinfection**, used on inanimate objects to kill most bacteria, some viruses, and some fungi. This does *not* kill *M tuberculosis* and does *not* kill bacterial spores.

According to the CDC, handwashing is the *single most important means* of preventing the spread of infection. CCTPs should strictly and properly decontaminate vehicles and equipment, change uniforms (including shoes/boots) or don new protective gowns over their uniforms and shoe covers, and perform handwashing between patient transports. Do not allow time constraints between transports to deter proper disinfection. Improper, cursory, or no **decontamination** of equipment and vehicles allows transfer of organisms to susceptible hosts and spreads disease. Transport equipment, PPE, and personnel can become reservoirs for disease organisms.

TABLE 19-9 Resistance of Infectious Organisms to Disinfectants

Resistance to Disinfection	Class of Organism	Organism Example	Class of Disinfectant	Example of Disinfectant
Most resistance	Spore formers	*Clostridium difficile*	EPA-registered sporicidal	Glutaraldehyde; household chlorine bleach (1:10 dilution)
High resistance	Mycobacteria	TB	EPA-registered tuberculocidal	Combinations of high-percentage hydrogen peroxide (*not* household hydrogen peroxide) and peracetic acid; chlorine dioxide; various phenolics
Medium resistance	Nonenveloped viruses	Norovirus, poliovirus, adenovirus, papilloma viruses	EPA-registered effective agent against norovirus	Household chlorine bleach; Quats; high-percentage hydrogen peroxide (*not* household hydrogen peroxide)
	Cationic detergent (Quats)-resistant bacteria	*Pseudomonas aeruginosa* and *Acinetobacter baumannii*		Household chlorine bleach; high-percentage hydrogen peroxide (*not* household hydrogen peroxide); note: do not use Quats (Pseudomonads are resistant to Quats)
Low resistance*	Fungi	*Trichophyton* and *Aspergillus*	EPA-registered fungicidal	Quats
	Vegetative bacteria	*Staphylococcus aureus* (including MRSA, VRSA, and VRE)	Germicidal, EPA-registered anti-MRSA and anti-VRE	Quats; high-percentage hydrogen peroxide (*not* household hydrogen peroxide); various phenolics
Least resistance*	Enveloped viruses	Influenza, hepatitis B, and HIV	EPA-registered anti-hepatitis B and anti-HIV	Most environmental cleaning agents, including bleach; Quats; phenolics

Abbreviations: EPA, Environmental Protection Agency; HIV, human immunodeficiency virus; MRSA, methicillin-resistant *Staphylococcus aureus;* Quats, quaternary ammonium compounds; TB, tuberculosis; VRE, vancomycin-resistant enterococci; VRSA, vancomycin-resistant *Staphylococcus aureus*.
*Note that low resistance to disinfectant does not mean the organism is not dangerous. It simply means some organisms can be killed easier than other organisms. Assume all patients transported carry infectious pathogens and take necessary steps to decontaminate the equipment and vehicle prior to the next transport.
Data source: Selected EPA-registered disinfectants. US Environmental Protection Agency. January 9, 2009. Available at: http://www.epa.gov/oppad001/chemregindex.htm. Accessed June 29, 2009.

Use barrier protection (eg, plastic wrap) for surfaces of equipment touched frequently with gloved hands or those difficult to clean with disinfectants (such as computer keyboards) or more likely to be contaminated with blood or body fluids. Do not attempt to clean and reuse disposable materials. Place contaminated disposable materials (dressings, gloves, syringes, tubing, gowns, surgical masks, and disposable N95 respirators) into appropriately marked biohazard containers. Always place reusable contaminated PPE (eye protection, uniforms, and shoes/boots) into appropriately marked storage

containers until it can be decontaminated. Do not reuse contaminated PPE until it has been disinfected. All biohazardous waste and uniforms should be handled with gloved hands. Uniforms should first be autoclaved and then laundered after transporting patients with highly virulent and communicable diseases, such as anthrax or avian influenza (H5N1). CCTPs should decontaminate the transport vehicle, shower and scrub with antibacterial soaps, and don street clothes prior to leaving for home after the transport of patients with highly virulent and communicable diseases. It is important to consider treatment of footwear worn during transport or to cover shoes during transport. Shoes should be considered part of the uniform and treated with disinfectant. Uniforms and shoes or boots should not be worn home routinely after patient transport—particularly after transporting patients ill with *C difficile* who had active diarrhea during the transport or patients who were ill with an antibiotic-resistant infection, necrotizing fasciitis, or pulmonary TB.

Postexposure Risks and Protocols

After a blood exposure, the appropriate actions differ depending on the type of exposure. Generally, the appropriate actions taken should be immediately employed: wash the area with soap and water, flush mucous membranes with copious amounts of water, and irrigate eyes with sterile solutions of saline or water. Exposure should be reported immediately to supervisory personnel and those managing blood exposure. Prompt reporting is paramount to ensure that any applicable postexposure treatment begins as soon as possible.

CCTPs who have received the hepatitis B vaccine are protected against contracting HBV from a blood exposure. CCTPs who have not been vaccinated or have not yet achieved full immunity have a 6% to 30% risk of contracting HBV from exposure to HBV-infected blood. The wide range of risk varies depending on the severity of exposure and the hepatitis B antigen status of the source. Blood that is positive for the antigen is more likely to transmit infection. The risk of infection after direct nonintact skin exposure to blood infected with HCV is approximately 1.8%. The average risk of contracting HIV after a needlestick or cut exposure to HIV-infected blood is 0.33% or approximately 1 in 300. The risk decreases for exposures to the eye, nose, or mouth. A small amount of blood splashed on intact skin poses no risk, except if the skin is damaged.

Postexposure prophylaxis is available for the prevention of seroconversion after a known exposure to HBV or HIV. If the exposed worker has not been immunized against hepatitis B, he or she should receive HBIG within 24 hours, followed by the hepatitis B vaccine within 7 days of the exposure. If the CCTP has been vaccinated against hepatitis B, but the anti-HB titer is unknown, an assay for anti-HB antibodies should be drawn to determine the person's immune status, followed by the administration of HBIG and a booster dose of hepatitis B vaccine, if necessary.

If exposed to HCV, there is no prophylactic treatment available to the exposed CCTP. The current recommendations are for the individual to receive interferon, if and when signs of an acute infection develop. Acute infection is diagnosed by increasing liver enzyme levels and detecting viral RNA in the serum.

If the source patient is HIV negative, nothing needs to be done. If the patient's status is unknown, the risks and benefits of postexposure prophylaxis should be discussed with employer health personnel, and the exposed person should be informed of his or her options. The basic regimen is administration of two drugs for 1 month for small exposure risks. Prophylaxis treatment for exposure to a hollow-bore needle contaminated with the blood of a patient known to be HIV positive should consist of three different antiretroviral drugs for 1 month. The exposed individual should be tested immediately following the injury and again at 6, 12, and 24 weeks after exposure. The employee should be aware of and report any febrile illnesses that occur during the 12 weeks following the exposure. The employee should also refrain from blood donation and unprotected sexual encounters during that same postexposure 12-week period,

until the employee's negative HIV status can be confirmed.

Postexposure prophylaxis is the treatment of persons with a known exposure to an infectious agent and is undertaken to prevent contracting the disease. HBIG is effective against HBV infection after an exposure has occurred. However, use of HBIG is not without risk; therefore, the risks and benefits of treatment should be considered. Treatment is appropriate for unvaccinated CCTPs who have been exposed to a source individual known to be positive for the hepatitis B antigen. In order for it to be effective, treatment should begin within 24 hours of exposure. HBV immunity should be confirmed by drawing an HB antibody titer (IgG). Any person whose antibody titer is negative, with a blood exposure regardless of the source person's HBV status, should receive the hepatitis B vaccine. Follow-up after postexposure prophylaxis for HBV is not recommended because the treatment is highly effective. However, any symptoms associated with hepatitis should be reported to the CCTP's health care provider.

There is no effective postexposure prophylaxis for HCV. Immune globulin is not recommended for exposures to HCV-tainted blood. As soon as possible after exposure, baseline laboratory studies should be performed for liver function test results and an HCV antibody titer should be drawn. These tests should be repeated at 4 to 6 months after exposure.

HIV postexposure prophylaxis is recommended for only a few types of occupational exposures to HIV-tainted blood because most exposures do not lead to contracting HIV, and the drugs used to prevent seroconversion are not benign. Common adverse effects include nausea, vomiting, diarrhea, lethargy, and headache. More serious adverse effects include kidney stones, hepatitis, and suppression of bone marrow. The risks and benefits of postexposure prophylaxis for HIV exposures must be weighed before starting treatment. The dangers associated with the antiviral drugs for a low-risk exposure may outweigh the reduction in risk in an already low-risk situation.

Treatment should begin as soon as possible after the exposure, as soon as within the first few hours. Animal studies have demonstrated the treatment is not effective after 24 to 36 hours have passed since the exposure event. The recommended postexposure protocol for HIV exposures is a 4-week course of two nucleosides. For those exposures associated with a higher rate of seroconversion, a protease inhibitor is added to the regimen. HIV antibody titers should be drawn immediately following exposure and periodically for at least 6 months after exposure. Any flu-like symptoms that occur with an abrupt onset during the follow-up period should be reported. If in doubt about the appropriate prophylactic treatment, consult the CDC Web site for the most current treatment regimen.

Transmission of HIV to health care providers following an occupational exposure has been documented in 57 patients. Of the 57 individuals, 26 have gone on to develop AIDS. An additional 139 cases of HIV have occurred in health care workers who have no reported risk factors for HIV other than occupational exposure, but for whom seroconversion after an occupational exposure was not documented. None of the 57 documented cases are EMS personnel; however, 12 of the 139 undocumented seroconversions were EMS providers.

The risk of seroconversion from an occupational exposure is related to percutaneous injury as it occurs when the patient is HIV positive. The risk of seroconversion is 0.33% from a deep puncture wound from a hollow-bore needle, visibly contaminated with blood from an HIV-positive patient with a high viral load. The risk of contracting HIV from such an injury is lower than the risk of contracting HBV or HCV from the same type of injury, if the health care provider is unvaccinated (the risk of contracting HBV from this type of injury is reported to be between 6% and 30%).

Flight Considerations

There are no special patient care considerations specific to air medical transport of patients with infectious diseases. Follow the same precautions and management strategies discussed in this chapter.

One important air medical service consideration when receiving a transport request is the length of out-of-service time required to properly disinfect or decontaminate the aircraft afterwards.

Summary

CCTPs must be aware of the risk to exposures from a variety of infectious agents. Their job makes them inherently at risk to both contagious agents and those transferred by needlesticks or body fluids. A solid knowledge of microorganisms and the diseases they cause is necessary to ensure their protection. An understanding of the fundamental principles of the immune system and infectious disease is an essential tool for medical care professionals. This knowledge, when applied to the critical care arena, will enable one to assess risk, minimize exposure, and prevent transmission of infection.

Case Study

YOU ARE DISPATCHED TO A RURAL HOSPITAL INTENSIVE CARE UNIT (ICU) for an emergency transfer to a tertiary hospital facility. En route to the hospital, dispatch relays the following limited patient information. Your patient is a 58-year-old man who weighs 72 kg. He was admitted to the ICU from the emergency department with respiratory failure. The patient has been diagnosed with postinfluenza methicillin-resistant *Staphylococcus aureus* (MRSA) pneumonia and he requires further consultation and treatment in a tertiary facility. The transport time for this patient typically would be only 30 minutes via air, but weather issues necessitate that the patient be transported via a ground unit. The ground transport time is approximately 2 hours with the inclement weather conditions.

On arrival to the hospital ICU, you are informed that the staff nurse and referring physician are at the patient's bedside. Both are dressed in full personal protective equipment (PPE) and are attempting to suction the patient's endotracheal tube with assistance from the respiratory therapist. You and your partner note the droplet precaution signage posted on the patient's door and the isolation cart placed outside the patient's room. After you and your partner don the appropriate PPE (gloves, gown, and surgical mask with a faceshild), the referring physician reports to you that the patient arrived at the hospital emergency department approximately 6 hours ago in acute respiratory distress and was electively intubated successfully with rapid sequence intubation medications. Prior to arrival at the emergency department, EMS performed multiple intubation attempts without success. They were able to assist the patient's ventilations with a bag-mask device but achieved an oxygen saturation level of only 86%. EMS reported that the patient was discharged from this same hospital just 2 days ago, after being treated for an upper respiratory infection. He has a medical history of hypertension, type II diabetes mellitus, chronic obstructive pulmonary disease, seizures, and gastroesophageal reflux disease. He has no known drug allergies to report, which is consistent with his recent admission record obtained by the emergency department physician. The patient is homeless and is a "frequent flyer" known to the EMS and emergency department staff. He admits to smoking a pack-a-day of cigarettes and drinks about 3 to 4 beers a day. He is not compliant with his daily prescription regimen to control his blood pressure, high glucose levels, and seizure disorder.

Your initial assessment of this patient reveals a Glasgow Coma Scale (GCS) score of 8. The patient appears anxious with restlessness noted in his lower extremities. The patient's upper extremities are secured with padded restraints because it was reported that he tried to pull out his endotracheal tube prior to your arrival. No sedation has been administered because his blood pressure was so labile. His airway is secured with an 8.0 endotracheal tube at the 24-cm mark (lip). The patient is being ventilated with synchronized intermittent mandatory ventilation (SIMV) with the following settings: tidal volume, 600 mL; rate, 26 breaths/min; FIO_2, 60% with 10 cm H_2O of positive end-expiratory pressure (PEEP). After

the previous suctioning of the endotracheal tube, it was noted that his secretions were thick, rusty colored (blood tinged), and had a foul odor. The patient has two peripheral IV lines in his left arm with normal saline running at 30 mL/h and a dopamine drip infusing at 10 μg/kg/min. The other IV line has a vancomycin drip (15 mg/kg) infusing over 1 hour. Prior to placing the transport monitoring equipment on this patient, the following vital signs are noted on the patient's ICU monitor: ECG, sinus tachycardia with occasional unifocal PVCs; heart rate, 138 to 145 beats/min; blood pressure, 88/40 mm Hg (confirmed with manual blood pressure to right arm); respiratory rate, 26 (SIMV); Spo_2, 88%; $ETCO_2$, 58 mm Hg; and a reported temperature of 101°F (rectal). After receiving the transfer chart, the following laboratory values are noted: WBC count, 26,000/mm^3; BUN, 58 mg/dL; creatinine, 2.3 mg/dL; Na+, 148 mEq/L; K+, 4.5 mEq/L; Cl^-, 109 mEq/L; glucose, 212 mg/dL; pH, 7.30; PCO_2, 54 mm Hg; PaO_2, 68 mm Hg; and HCO^-, 16 mEq/L. A chest radiograph reveals bilateral lobular infiltrates consistent with pneumonia. A gram stain shows gram-positive cocci in a cluster and the patient has a prior history of MRSA infection. The staff nurse reports his daily input/output as 600 mL/50mL/h with amber-colored urine.

On completion of your assessment of this patient, you note the following findings. The skin is "hot" to the touch, dry, and pale. The pupils are equal and reactive to light at 5 mm (bilateral/brisk), and your patient remains at a GCS score of 8 with eyes opening to tactile stimulus. The endotracheal tube remains secured with tape at the 24-cm mark at the lip line. The trachea is midline with noted "mild" jugular venous distention to the right neck with the head of the bed at 30°. The remainder of the head/eyes/ears/nose/throat examination is within normal limits for this patient. The chest wall has an equal symmetrical rise and fall with mechanical ventilations, but shallow effort is noted with patient-initiated ventilations. The lungs sounds reveal bilateral coarse rhonchi, and the abdomen is soft with no distention noted. The pelvis is within normal limits with an indwelling urinary catheter to gravity drainage. The lower extremities are pale and cool to the touch without edema. Pedal pulses have been only obtained via Doppler, and a weak, thready radial pulse is noted with delayed capillary refill of greater than 3 seconds. There is no swelling or redness noted to the two peripherally inserted IV lines being used with no signs of infiltrates.

Before you move the patient to the transport stretcher, your partner transfers the dopamine and vancomycin drip to your transport pumps with the normal saline at a keep vein open rate of 30 mL/h. The patient is placed on your unit transport monitor with ECG, Spo_2, $ETCO_2$, and noninvasive blood pressure with alarms on and functioning. The patient remains on the hospital ventilator while your partner duplicates the above ventilator settings from the referring hospital. The patient is moved to your stretcher without incident as the transport ventilator is attached (alarms are on and functioning). You depart the ICU with the transfer documentation and radiology studies to your awaiting ground unit. All PPE is properly disposed. The patient is loaded and secured to the ground unit with all power and oxygen requirements transferred to the ground unit source. You depart the referral hospital en route to the receiving facility with routine traffic due to the weather conditions.

While en route to the receiving hospital, the patient's condition remains the same without any change or improvement in the hemodynamic status (manual BP, 90/38; heart rate, 140 beats/min with occasional unifocal PVCs). The neurologic status remains the same with a GCS score of 8 with periods of noted agitation and restlessness to his lower extremities. The Spo_2 remains at 88% to 92% with the above ventilator settings, and an $ETCO_2$ of 45 to 54 mm Hg. Your partner contacts your dispatch center to relay an updated report and an estimated time of arrival to the receiving hospital. No orders are requested or received from the receiving facility, but your partner advises that you will be following your standing protocols for this patient's care.

1. Is there anything that you would want to change (ie, ventilator settings) or initiate on this patient?

2. What other interventions or medications would be beneficial for this patient?
3. Is this a suitable mode of transfer based on the patient's condition?

Analysis

This is a complex patient scenario that should have been corrected in a more stable environment, such as the referring hospital ICU with a multidisciplinary team approach. The incidence of postinfluenza MRSA pneumonia is rare, and demands critical thinking skills and rapid interventions by the CCTP. This patient was recently discharged after being treated for an upper airway infection, and he is also noncompliant with his daily medication regimen. He is homeless with admitted alcohol and tobacco abuse that exacerbates his history of other medical issues (COPD, diabetes, hypertension). Treatment at the ICU bedside or en route to the receiving hospital should include, but not be limited to, the following interventions:

- Administration of a chemical paralytic such as vecuronium (Norcuron) at 0.1 mg/kg.
- Administration of a sedative medication such as etomidate (Amidate) at 0.3 mg/kg or half dose at 0.15 mg/kg for this patient's declining hemodynamic status.
- Changes to the ventilator settings could include the following:
  - Increase the FIO_2 from 60% to 100%.
  - Decrease the rate from 26 to 14 to 16 breaths/min, which decreases the potential for increased air trapping.
  - Lengthen the inspiratory time, which would allow for exhalation of retained CO_2 (already has a history of COPD and hypercapnia).
- Decrease the PEEP setting from 10 to 5 cm H_2O, which has inadvertently increased the intrathoracic pressure and decreased the venous return to the right atrium, causing hypotension. The unstable hemodynamic status (hypotension) is also the result of the patient's septicemic shock state.
- Administration of an additional fluid bolus (3 to 4 liters) because the patient remains hypotensive. The CCTP must reassess breaths sounds frequently and be cautious of pulmonary edema. After fluid resuscitation has taken place, the dopamine dosage can be increased.
- Administration of an additional infusion of another vasopressor agent such as vasopressin or norepinephrine.

After the following interventions have taken place en route to the receiving hospital, the patient responds well to the treatment and his hemodynamic status is within normal limits with noted clear yellow urine in the indwelling urinary catheter. Though it would have been a more expedient transport via air, the weather conditions hindered this mode of transport. The patient tolerated the ground transport and was subsequently transferred to the ICU nursing and medical staff's care.

Prep Kit

Ready for Review

- Patients, health care workers, and members of the community are at risk for contracting infectious diseases from their interactions with one another, and through contact with infectious agents that are found in body fluids, on solid surfaces, and in the air.

- A solid understanding of the fundamental principles of infectious disease is an essential tool that will enable CCTPs to assess risk, minimize exposure, and prevent transmission of infection.
- For an organism to contract a disease, the host must be susceptible. Susceptibility is determined by factors such as nutritional status, immune status, genetic makeup, living conditions, and exposure.
- Disease can be transmitted directly through inhalation, ingestion, or direct contact with infectious agents, or indirectly via vectors, fomites (eg, contaminated fluids or equipment), or some other complex cycle of interactions.
- For the immune system to protect the body from invading microorganisms, it must be able to distinguish "self" components or cells from "nonself" components or cells; must be both highly specific and general; and must provide protection that consists of several layers so that once one level of immunity has been breached, additional methods of fighting infection remain available.
- Immunity can be either innate (the type of immunity one is born with, which provides protection from invading pathogens despite a lack of prior exposure) or acquired (the type of immunity that develops as a result of interactions between components of the immune system and the invading microbe, which provides improved protection with repeated exposures to specific agents).
- Innate immunity is nonspecific, is activated immediately on invasion, and has no memory. The primary components of innate immunity are the skin, mucous membranes, and their secretions, which provide mechanical and chemical barriers to invasion.
- Nonspecific factors of the immune system help limit growth of microorganisms within the body and include cells such as natural killer cells, neutrophils, and macrophages; proteins such as complement or transferrin; and the body's general responses to invasion with actions such as fever or inflammation.
- Acquired immunity includes both humoral and cellmediated immunity: humoral immunity is provided by B cells through the production of antibodies and complement, and cell-mediated immunity is provided by T cells.
- Cytokines and macrophages are modulators of both cellmediated and humoral immunity.
- Acquired immunity results from the interaction of the agent with the host, is specific, improves with repeated interactions, allows for the development of immunologic memory, and can be either active or passive.
- Active immunity (an ongoing process of developing antibodies and activating T cells in response to invading agents) occurs when an individual either is exposed to microbes through the natural process of infection or artificially stimulated through a vaccine.
- Passive immunity is the process of giving an individual a preformed antibody (from a donor) in the event of an exposure during which the individual has not yet developed immunity.
- Antigens are a complex of proteins and/or sugars that are anchored into the cell membrane and displayed on the cell's surface. An antigen is recognized by the immune system as either self or nonself; in the latter case, it will induce a humoral or cell-mediated response.
- The immune system includes the central lymphoid organs (the thymus and the bone marrow), which are the site of synthesis and differentiation of immunocompetent cells.
- The peripheral lymphoid organs are where immunocompetency is expressed; they include the spleen, lymph nodes, tonsils, intestinal Peyer's patches, and mucosa. Lymph nodes are made up of lymphoidal tissue located throughout the body as single units or in chains of units and are prominent in the neck, axilla, and groin.
- T lymphocytes mature to express phenotypic markers or clusters of differentiation called CD markers.

CD4+ cells (helper T cells) are regulators of the immune system; CD8+ cells (cytotoxic suppressor T cells) kill virus-infected and tumor cells.

- B cells either differentiate into plasma cells that produce specific antibodies on encountering an antigen or become memory cells.
- Antibodies are globulin proteins (immunoglobulins) that react to antigen. There are five classes of antibodies: IgA, IgM, IgG, IgE, and IgD.
- The humoral (antibody-mediated) response is effective against agents that produce toxins, against bacteria that have polysaccharide capsules, and against some viral infections.
- In the primary immune response, antibodies do not appear until 7 to 10 days after the initial encounter. On a second encounter with the same antigen or a closely related antigen, a more rapid secondary response occurs, with antibody being detected in the serum in only 3 to 5 days, and antibody titers being produced that are much higher than those seen in the primary response.
- Cell-mediated immunity plays an important role in defending against intracellular infections, such as TB or gonorrhea, and viral infections, fungal infections, parasites, and tumors. Antibody is not involved in CMI reactions except in the antibody-dependent cellular cytotoxic reactions.
- The process of phagocytosis is performed by macrophages, neutrophils, and monocytes, which engulf and destroy bacteria, foreign antigen, and cellular debris.
- The remaining fragments of the invading cells, known as epitopes or antigen, are displayed by the phagocytic cell in close association with specialized proteins on the exterior of the cell; these proteins are called major histocompatibility complex.
- An inappropriate or exaggerated immune response known as hypersensitivity (allergic) reaction occurs when a first encounter sensitizes an individual to the antigen and induces an antibody response, and then subsequent encounters with the same or closely related antigen elicit the allergic response.
- There are four major types of hypersensitivity reactions: types I, II, and III are antibody-mediated responses, whereas type IV is a cell-mediated response.
- Anaphylaxis is a serious allergic reaction that has a rapid onset of symptoms, has a variable clinical presentation, and, if unrecognized, can lead to respiratory arrest, circulatory collapse, and death. Common triggers for anaphylaxis include foods, drugs, antibiotics, and *Hymenoptera* venom.
- Most cases of anaphylaxis have cutaneous involvement, including flushing, itching, urticaria, or angioedema. Upper airway involvement may include changes in phonation, the sensation of throat closure or choking, cough, wheezing, and shortness of breath.
- The treatments for anaphylaxis focus on early recognition and the use of epinephrine to prevent progression to life-threatening symptoms. Airway management, including intubation, if necessary, and fluid resuscitation are key components of care for a patient experiencing anaphylaxis.
- In biphasic anaphylaxis, the initial resolution of symptoms is followed by a reappearance of symptoms and, if not anticipated, can be the cause of a life-threatening recurrence.
- Immunodeficiency occurs when one or more of the components of the immune system is damaged or is nonfunctioning as the result of illness or lack of functioning immune cells, or when the immune system is intentionally suppressed by various immunosuppressant therapies.
- Critically ill patients have little reserve to fight infections as the result of a decrease in host defenses and loss of innate immunity.
- Features of chronic illness that decrease innate immunity include stress, malnutrition, invasive procedures, loss of physical barriers of skin and mucosal surfaces, and pathogenic bacterial

overgrowth or bacterial growth in normally sterile tissue.

- Management of illnesses and injuries during transport of a transplant patient may be complicated by the side effects of immunosuppressive medications (eg, hypertension, hyperglycemia, hyperkalemia, nephrotoxicity, and neurotoxicity) or by the patient's immunosuppressed condition.
- Normal flora is comprised of colonies of nonpathogenic species that assist the immune system by preventing the overgrowth of pathogenic strains by using up available nutrients, maintaining a certain pH level, and creating bac-teriocins (antibacterial toxins).
- Many individuals requiring critical care transport are immunocompromised, and CCTPs need to be keenly aware that these very sick patients can be inadvertently infected with opportunistic infections (ie, diseases caused by normally nonpathogenic agents in patients with an abnormally functioning immune system).
- Pathogenic organisms cause disease because they possess unique factors that help them gain entry into the body, colonize and overcome host defenses, produce toxins that cause cytopathic effects, or do mechanical damage to the body.
- Properties of microbes that influence their virulence (ie, degree of pathogenicity) include host and tissue specificity (the ability of the organism to gain entry into a host), adherence to specific host cells, invasion of host tissue, evasion of host defenses, and toxicity.
- Virulence can be experimentally quantified by two measures: the ID_{50} and the LD_{50}.
- Typical portals of entry into the body for pathogens are through broken skin, hair follicles, or sweat glands and through the mucous membranes of the respiratory, digestive, and genitourinary tracts, or the conjunctiva of the eye. Once inside the body, the pathogenic organism must be able to adhere to specific cells to begin colonization.
- Bacteria are capable of producing two types of toxins: exotoxins, which are produced inside the cell and are released into surrounding tissues or fluids; and endotoxins, which are part of the gram-negative cell wall and are not released until the bacteria are destroyed.
- The DNA of various bacteria encodes both virulence factors and antibiotic resistance genes. Many virulence factors are carried on circular, extrachromosomal, self-replicating pieces of DNA called plasmids; the uptake of fragments of the chromosomal DNA released on lysis of a nearby cell may also lead to proliferation of the harmful DNA once it becomes part of the recipient cell's genome.
- Bacterial cells are subject to infection by bacterial viruses called bacteriophages. Bacteriophages can cause destruction of a host bacterial cell by growth and lysis of the host bacterium; sometimes, the bacteriophage DNA becomes integrated into the host chromosome and remains there as the bacteria undergo replication.
- Viruses that enter the body bind to host cell surface receptors using viral proteins found on the surface of the virions, leading to receptor-mediated endocytosis that brings the virus directly into the cell.
- Once inside a cell, the virus particle may release its viral nucleic acid, allowing the viral DNA or RNA to direct synthesis of viral-encoded protein and nucleic acid molecules and stimulate the process of new virion assembly.
- Viruses cause illness through three mechanisms: (1) production of viral-specific molecules that deplete the host cell's resources and lead to eventual cell death; (2) induction of programmed cell death (apoptosis); and (3) induction of large inflammatory responses.
- Epidemiology is the study of how disease is distributed within populations and the factors that influence that distribution.

- The incidence of a disease is the number of new cases of disease within a defined population over a defined period of time.
- The prevalence of a disease is the number of cases of disease detected in a specified period of time, regardless of when the illness was contracted.
- Factors such as the incubation period of a disease or the presence of different carrier states may cause the disease to go undetected and unaccounted in an individual, but can place others at risk for contracting the disease. For this reason, CCTPs must use appropriate disease control and prevention practices when providing care to all patients, regardless of whether the patient is currently exhibiting symptoms.
- For a microorganism to infect and reinfect a host, it must have the ability to survive, duplicate or reproduce, and spread to new hosts. Reservoirs of infection provide a supply of nutrients and an environmental niche that enable long-term microbial survival.
- Persons who are colonized by pathogenic species but do not have symptoms of the infection (ie, asymptomatic or latent carriers) can serve as sources of infection during extended hospital admissions.
- Health care providers unwittingly act as a major source of cross-infection when they attend different patients without proper intervening antisepsis.
- The health care environment itself may serve as a reservoir of infection. Some organisms can survive on inanimate objects, including durable medical equipment, for an extended duration of time.
- Direct contact involves person-to-person transmission of a pathogen (ie, no intermediate carrier is involved in the transfer).
- Horizontal transmission (ie, the transmission of infection between members of the same generation) may occur through sexual transmission, exchange of respiratory droplets or secretions, and transmission by contact.
- Vertical transmission is the exchange of an infectious agent between mother and fetus in utero or during the birthing process.
- To guard against direct transmission of infection, health care workers can use protective barriers such as gloves, gowns, masks, and eye shields, which prevent direct contact with infectious materials during invasive procedures or when providing care for an infectious patient.
- Indirect transmission occurs when there is an intervening step in the transmission from reservoir to susceptible host. The intermediate step involves an inanimate object or fomite, such as towels, bedding, thermometers, and contaminated syringes.
- Many infectious diseases are transmitted through the use of a medium, such as water, food, air, or contaminated body fluids. Water can be either the reservoir of disease or a vehicle for the transmission of that disease.
- Vector transmission is the exchange of a pathogen from an infected organism to a susceptible host via an insect or other animal.
- Meningitis (ie, the inflammation of the leptomeninges) is a serious medical condition that can be caused by a bacterial, viral, or fungal infectious agent.
- Bacterial meningitis has an acute onset and is a medical emergency that requires prompt diagnosis and treatment to preserve life and neurologic function. The classic triad of symptoms observed in patients with this disease includes fever, nuchal rigidity, and altered mental status.
- Most cases of bacterial meningitis are observed in adults and can be caused by a variety of bacterial species, including *N meningitidis, S pneumoniae*, and *H influenzae*.

- The severity of the symptoms and the disease is less serious with viral meningitis than with bacterial meningitis, and complete recovery can occur in 2 to 7 days without medical intervention.
- Respiratory syncytial virus causes seasonal outbreaks of acute respiratory tract infections.
- Necrotizing fasciitis is a limb- or life-threatening infection of the soft tissue that affects the subcutaneous tissues, fat, and fascia but usually spares the skin and muscle. Prompt diagnosis and treatment with antibiotic and complete surgical débridement are important to reduce the high mortality rate associated with this disease.
- Epiglottitis (ie, the result of an infected and inflamed epiglottis, aryepiglottic folds, and/or surrounding tissues) typically occurs in children between the ages of 1 and 5 years. It is a potentially life-threatening infection, and patients must be treated promptly with empiric antibiotics.
- Tuberculosis is caused by infection with *M tuberculosis*, which is transmitted via respiratory droplets from the cough of an infected person to the respiratory epithelium of a susceptible person.
- In those with TB, the first site of infection is the lungs, but the pathogen can also spread to the kidney or spine. Most persons infected with *M tuberculosis* are asymptomatic. Persons with full-blown pulmonary tuberculosis typically exhibit five symptoms: cough, fever, weight loss, night sweats, and fatigue.
- The emergence of drug-resistant strains of TB has precipitated the use of a multidrug regimen for cure. The individual drugs used may vary slightly, but the mainstay of therapy is isoniazid.
- Multidrug-resistant TB is TB that is resistant to at least two of the standard anti-TB medications, isoniazid and rifampin. There are limited treatment options for patients with this form of the disease.
- When transporting a patient with suspected or known TB, CCTPs should place a mask on the patient (if tolerated) and wear a mask themselves.
- CCTPs should comply with yearly PPD testing (which identifies antibodies to TB) to determine a personal exposure history to TB.
- If care was provided for a patient known to have TB and exposure was a possibility, providers should get a PPD test 6 weeks after exposure to assess their personal status.
- Pneumonia is an infection of the lung parenchyma that is caused by bacterial, viral, or fungal agents; it is a major cause of death in the United States.
- Many different bacterial species cause pneumonia, including *Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus, Chlamydia pneumoniae*, and *Legionella pneumoniae. Mycoplasma pneumoniae* and viruses cause atypical pneumonias.
- Nosocomial or hospital-acquired pneumonia is a pneumonia that occurs in a hospitalized patient within 48 hours or more after admission to the hospital that was not apparent at the time of admission.
- Ventilator-associated pneumonia is a type of hospital-acquired pneumonia that occurs in ventilated patients and appears more than 48 hours after endotracheal intubation.
- Health care–associated pneumonia is a pneumonia that occurs in nonhospitalized patients who have contact with health care facilities or personnel, such as residents of long-term health care facilities, those undergoing hemodialysis, or those who have had a recent admission to an acute care facility.
- *Pneumocystis jiroveci* pneumonia is a fungal infection that is seen in patients with immunosuppression, such as those taking antirejection therapy for solid organ transplantation or those infected with HIV. It is included in the list of AIDS-defining illnesses.
- CCTPs should use standard precautions when caring for patients suspected of or diagnosed as having

pneumonia. If a patient is coughing, place a mask on the patient (if tolerated) and on the CCTP; wear gloves; use good handwashing technique between transports; and clean and decontaminate equipment, including the transport vehicle, prior to undertaking further transports.

- It is especially important to decontaminate equipment such as laryngoscopes and suction equipment after transportation of patients with oral candidiasis (thrush) or patients infected with *P jiroveci*.
- There are few treatments available for influenza except supportive therapy; however, when diagnosed within the first day of symptoms, treatment with an antiviral medication may reduce the severity and duration of symptoms.
- When influenza undergoes a major antigenic drift or a novel combination of hemagglutinin and neuraminidase emerges (antigenic shift), people have little or no antigenic protection from prior infections; pandemics of influenza may then break out and spread quickly.
- Herpesviruses are noted for their ability to cause latent infections, during which the acute viral syndrome is followed by a symptom-free period and the virus is inactive. Reactivation of the virus can occur when the patient is immunosuppressed or through inciting agents.
- Six pathogenic members of the herpesvirus family cause significant disease in humans: herpes simplex types 1 and 2, varicella zoster virus, cytomegalovirus, Epstein-Barr virus, and human herpesvirus 8. Epstein-Barr virus and human herpesvirus 8 are associated with the development of Burkitt's lymphoma and Kaposi's sarcoma, respectively.
- Hepatitis (inflammation of the liver) can be caused by exposure to infectious agents (usually viral), toxins (eg, alcohol), or drugs (eg, acetaminophen). The course of the illness ranges from acute to chronic, and the severity ranges from mild to life threatening.
- Hepatitis A virus (HAV) is usually transmitted via the fecal-oral route, although sexual and parenteral transmission is possible. Large-scale outbreaks can occur, usually the result of contamination of food or drinking water.
- HAV infection can be prevented by active immunization with the HAV vaccine or by passive immunization with immune globulin administered before or after a known exposure.
- Hepatitis B virus (HBV) is transmitted through parenteral routes, or nonoral transmission. Health care providers are considered an at-risk group for this infection and should receive the HBV vaccine series, with immunity confirmed by titer.
- Unlike HAV, HBV can induce other disease states in addition to causing acute infections (namely, acute hepatitis, fulminant hepatitis [severe inflammation of the liver, accompanied by rapid destruction of the liver leading to liver failure], and chronic hepatitis [chronic asymptomatic carriers]).
- The treatment and prevention of HBV infection involves screening the blood supply and removing HBV-contaminated units and donors from the blood pool.
- Hepatitis C virus (HCV) is transmitted parenterally through transfusion or IV drug use, through sexual contact with infected individuals, and from mother to baby. In most cases, acute infection leads to chronic infection, with the disease eventually progressing to hepatitis and fibrotic liver disease (cirrhosis).
- There is no cure for HCV, and there is no vaccine available to prevent infection; the goal of treatment is to prevent the development of cirrhosis and liver failure.
- Hepatitis delta virus (HDV) is transmitted parenterally, but can replicate only with the aid of HBV. All patients with severe HBV infection should be tested for HDV coinfection or superinfection.
- HIV causes AIDS and AIDS-related complex. This retrovirus infects T lymphocytes and other cells that

display the CD4 surface protein, and is notable for causing "slow" infections with long incubation periods.

- Transmission of HIV infection occurs by the transfer of infected cells or free virus from one individual to another. The transmission occurs both horizontally (via sexual contact or by exposure to infected blood) and vertically (from mother to neonate across the placenta or during delivery and from an infected mother through breast milk).
- As a CCTP, exposure to patients with HIV infection is a potential risk for disease contraction. Prevention of exposure (eg, with needlesticks) is the most important method of protection for the health care worker.
- The three stages in HIV infection—acute, latent, and late (period of immunodeficiency)—are largely determined by the CD4 cell count and/or symptoms of the disease.
- Antibodies to HIV are not detected in the blood until 3 to 4 weeks after the initial infection. The person is infected with HIV and is capable of spreading the disease through sexual or blood contacts during this window of time, even though the HIV antibody test result will be negative prior to seroconversion.
- Even while the HIV-infected patient is asymptomatic and symptoms remain latent, the virus continues actively replicating within the lymph nodes.
- Toward the end of the clinical latency period, the HIV-infected patient may begin to experience ARC syndrome, a constellation of symptoms including fevers, unexplained weight loss, fatigue, diarrhea, and generalized lymphadenopathy.
- ARC-related symptoms are often what persuade the patient to seek help in the health care system. ARC syndrome may mark the beginning of the final progression toward AIDS.
- The criteria for the diagnosis of AIDS include HIV infection with a CD4+ helper T-cell count of less than 200 cells/μL and/or the presence of an AIDS-defining condition.
- Patients with HIV often present many years after the initial infection or acute stage of the disease, because in the initial stages the disease is often mild and the symptoms are nonspecific.
- For many patients with HIV, the illness is noticed when the patient begins to experience symptoms; eventually, loss of cell-mediated immunity predisposes the host to many opportunistic infections.
- In HIV-infected patients, the viral load is estimated by determining how much viral RNA is present in the blood. It indicates the magnitude of HIV replication and its associated rate of CD4 cell destruction; is the most accurate indicator of the risk for disease progression; and is used for planning and monitoring antiretroviral therapy.
- Three classes of antiretroviral drugs are currently used to treat HIV infection: nucleoside analog reverse transcriptase inhibitors, protease inhibitors, and non-nucleoside reverse transcriptase inhibitors. A fourth class of drugs, fusion inhibitors, is also under development.
- The goal of current therapies for HIV infection is to prolong life (by preventing opportunistic infections and malignancies, and slowing the rate of virus production) while maintaining the best possible quality of life.
- Emerging infectious diseases (diseases whose incidence has increased dramatically in the last 20 years) are particularly damaging to immunocompromised individuals, such as those living with HIV disease, those receiving immunosuppressive therapy such as the recipients of organ transplants, or those who are immunosuppressed as a result of their treatment for cancer.
- Severe acute respiratory syndrome is an emerging lower respiratory tract illness that is associated with high morbidity and mortality.

- Infection with *Escherichia coli* O157:H7 may occur after eating undercooked, contaminated ground beef; drinking unpasteurized milk and fruit drinks; eating unwashed lettuce or sprouts; eating salami; swimming in or drinking sewage-contaminated water; or having person-to-person contact.
- West Nile virus is a mosquito-borne pathogen that occasionally leads to meningitis or encephalitis; there is no cure for West Nile virus encephalitis.
- *Helicobacter pylori* bacteria infect the stomach and duodenum and are the causative agents of most peptic ulcers. The goal of *H pylori* treatment is the complete elimination of the organism from the body.
- Antibiotics are compounds that are produced by bacteria or fungi that inhibit the growth of bacteriostatic organisms or kill other (bacteriocidal) bacterial organisms.
- All antibacterial agents follow the same basic principle: target bacterial proteins, processes, or cellular components that are not found in human cells.
- The relatively few antiviral treatments available work by interrupting the viral replication cycle at a specific step.
- Antifungal therapies typically bind with components in the fungal cell wall, making the membrane less fluid and more susceptible to rupture and, therefore, leading to cell death.
- Virtually all important pathogenic bacteria have demonstrated resistance to currently available Food and Drug Administration–approved antibiotics.
- Three important factors that contribute to the emergence of resistant bacteria within a given population or community are misuse use of antibiotics, poor infection control, and importation or intrusion of already resistant strains.
- Health care workers are a major source of cross-infection between critically ill patients, and handwashing is the single most important step that a health care provider can take to reduce the spread of infection.
- Methicillin-resistant *Staphylococcus aureus* (MRSA) can colonize a variety of tissues, causing infections such as cellulitis, cutaneous abscesses, wound infections, osteomyelitis, septic arthritis, endocarditis, pneumonia, and septicemia.
- High rates of MRSA are found among residents of long-term care facilities, but serious infections with MRSA are more prevalent in the acute care setting.
- Risk factors for colonization with MRSA include dialysis, diabetes, use of injectable drugs, chronic skin conditions such as decubitus ulcers, and a history of prior antibiotic use.
- The emergence of vancomycin-resistant MRSA poses a serious threat to patients and health care workers. Any patient with this type of infection should be isolated, and the infection should be reported to public health authorities.
- Although antibiotic-resistant species of *Enterococcus* rarely cause illness in healthy people, they can cause serious infections in immunocompromised, postoperative, and other seriously ill individuals.
- When transporting patients infected with vancomycin-resistant enterococci, providers should implement infection control measures such as handwashing, patient isolation, use of protective barriers such as gloves and gowns, and proper disposal of contaminants.
- Rickettsial diseases are transmitted via tick bite and include Rocky Mountain spotted fever, ehrlichiosis, and anaplasmosis.
- Rocky Mountain spotted fever should be considered a possible diagnosis in all patients presenting with

fever and having suspected or known tick exposure. Rocky Mountain spotted fever begins acutely with fever accompanied by one or more of the following: headache (usually severe), rash, muscle aches, nausea and vomiting, and malaise. Treatment is with doxycycline (or a similar antibiotic).

- Knowledge of epidemiologic principles is crucial in differentiating between the presentation of a naturally occurring disease and an unusual, non-natural event that should raise concern that a bioterrorist event has been initiated and that local emergency response systems should be activated.
- In bioterrorism scenarios, the CCTP must maintain an index of suspicion regarding a potential threat because early symptoms may be nonspecific, but early treatment will be required to prevent severe injury or death. Providers must always take steps to protect themselves before approaching patients and should take responsibility for implementing immunologic protection.
- The health risks associated with the job of the CCTP are not limited to blood or body fluid exposures; rather, the greatest risk comes from patients who are actively infectious but are undiagnosed.
- Steps to reduce exposure risks include the use of appropriate protective clothing and personal protective equipment; proper handling of contaminated medical devices; and implementation of isolation precautions to control the spread of infection to others.
- The Centers for Disease Control and Prevention recommends that all emergency medical care providers be vaccinated against the following preventable infectious diseases: measles, mumps, rubella, varicella, hepatitis B, influenza (annual), pertussis, tetanus, polio, diphtheria, pneumococcal vaccine, and meningococcal vaccine.
- By law, all needlesticks and other injuries with sharps contaminated by blood must be reported to the employer.
- Universal precautions are recommended to prevent transmission of bloodborne pathogens such as HIV and hepatitis B and C; they stipulate use of gloves, gowns, or aprons (or uniforms) and masks or other eye protection to reduce exposure of the skin (especially abraded or broken skin) and mucous membranes.
- The first response to a percutaneous exposure should be to wash the wound thoroughly with soap and water or, if water is not available, an alcohol-based hand cleanser.
- After a significant exposure, the source patient and health care worker both should be tested for baseline values, and the exposed health care worker should receive follow-up testing at 6 and 12 weeks and at 6 months.
- Standard precautions are used with all patients and protect against contact with blood, body fluids, and secretions, regardless of the presence of visible blood, and with nonintact skin and mucous membranes.
- Transmission-based precautions are isolation precautions that are designed to be employed during the transport of all patients who have documented infection or who are suspected to be infected with highly transmissible or epide-miological significant infectious agents.
- Airborne precautions include use of an N95 mask by CCTPs, use of a negative-pressure room (airborne infection isolation room) or transport vehicle, or use of the exhaust fan during transport.
- Droplet precautions include isolation of the patient and use of surgical masks by CCTPs (or keeping at a distance of 3′). Contact precautions include isolation of the patient, use of a gown (or uniform) and gloves, and, if warranted, face protection by CCTPs.
- Clean, nonsterile, disposable gloves are standard equipment on critical care transport units and must be donned by all personnel prior to initiating patient care.

- Masks, face shields, and eye protection are worn to protect mucous membranes of the eyes, nose, and mouth during procedures and patient care activities that are likely to generate splashes or sprays of blood, body fluids, or excretions.
- Gowns are used to protect skin and to prevent the soiling of clothing during procedures that are likely to generate splashes of blood or body fluids.
- Proper handling of sharps includes not attempting to bend, remove, or recap needles; not shearing or breaking needles; and disposing of sharps into an appropriate container as soon as possible after use.
- Contaminated work surfaces and all equipment used during the course of the transport must be decontaminated with an Environmental Protection Agency–registered disinfectant on completion of the transport.
- Contaminated laundry should be placed into leakproof bags, labeled, and stored until proper disposal is possible; likewise, regulated waste must be placed in closeable, leak-proof containers designed for disposal.
- Do not allow time constraints between transports to deter proper disinfection.
- In case of a blood exposure, wash the area with soap and water, flush the mucous membranes with copious amounts of water, irrigate the eyes with sterile solutions of saline or water, and report the exposure immediately to supervisory personnel and those managing blood exposure issues.
- Postexposure prophylaxis is available for the prevention of seroconversion after a known exposure to hepatitis B or HIV, but not after exposure to hepatitis C.

Vital Vocabulary

active immunity Production of specific antibody in response to an infection or antigen.

acquired immunity The immunity the body develops as part of exposure to an antigen.

antigen An agent that, when taken into the body, stimulates the formation of specific protective proteins called antibodies.

bacteriocidal Capable of killing bacteria.

bacteriostatic Capable of inhibiting bacterial growth.

decontamination Use of chemical, physical, or other means to remove, inactivate, or eradicate harmful microorganisms from persons, surfaces, or objects.

epidemiology The study of disease distribution within populations and the factors that determine that distribution.

epitope The specific portion of the antigen that is recognized by the antibody or B cell.

etiology The cause of a disease.

fecal-oral route The route of transmitting infectious organisms from one individual to another; commonly, enteric bacteria or viruses are shed in the feces, spread via contamination of food or water sources, and ingested by other individuals.

fomite An inanimate object that is capable of transmitting infectious organisms from one individual to another.

hapten A small-molecular-weight nonprotein molecule that is capable of reacting with a specific antibody but cannot elicit the formation of antibodies unless bound to a carrier protein; also called a

partial or incomplete antigen.

health care–associated pneumonia (HCAP) A pneumonia that occurs in nonhospitalized patients who have contact with health care facilities or personnel.

high-level disinfection Treatment used on inanimate objects to kill all microorganisms but which does *not* kill bacterial spores.

hospital-acquired pneumonia (HAP) Pneumonia that occurs in a hospitalized patient within 48 hours or more after admission to the hospital that was not apparent at the time of admission.

hypersensitivity Occurs when a patient reacts with exaggerated or inappropriate allergic symptoms after coming into contact with a substance the body perceives as harmful.

iatrogenic Any adverse physical condition that results from medical treatment.

immunocompromised Unable to mount a normal immune response as the result of disease (such as AIDS) or chemotherapy treatment.

immunodeficiency An abnormal condition in which some part of the body's immune system is inadequate, and consequently resistance to infectious disease is decreased.

indicator conditions Rare or unusual diseases associated with an immunocompromised state. Also called AIDS-related diseases.

innate immunity Immunity to diseases inherent in the genetic makeup of an individual.

intermediate-level disinfection Treatment used on inanimate objects to kill *M tuberculosis*, vegetative bacteria, and most viruses and fungi, but which does *not* kill bacterial spores.

low-level disinfection Treatment used on inanimate objects to kill most bacteria, some viruses, and some fungi, but which does *not* kill *M tuberculosis* and does *not* kill bacterial spores.

necrotizing fasciitis A limb- or life-threatening infection of the soft tissue that affects the subcutaneous tissues, fat, and fascia.

nosocomial Infections acquired during hospitalization or a nursing home stay.

opsonize To make bacterial cells more susceptible to the action of phagocytosis by the action of binding an antibody to the pathogen's cell membrane.

palpable purpura Larger areas of bleeding under the skin (greater than 3 mm).

passive immunity Short-lived immunity acquired from placental transfer, antibodies in the mother's milk, or antiserum administered IV.

pathogen Any microbe capable of causing a disease state.

petechial lesions Small areas of bleeding under the skin (pinpoint).

prophylaxis Treatment of disease before it occurs (ie, to prevent the disease).

quiescent A latent state as in a dormant virus or bacterium, in which an infected patient will exhibit little to no symptoms.

respiratory syncytial virus (RSV) A labile paramyxovirus that produces its characteristic fusion of human cells in a tissue culture known as the syncytial effect; can affect both upper and lower respiratory tracts but is more prevalent with the lower, causing pneumonias and bronchiolitis.

rickettsial diseases Diseases transmitted via tick bite; the three major diseases in this class include Rocky Mountain spotted fever, ehrlichiosis, and anaplasmosis.

seroconversion Development of antibodies, measured in the serum, in response to an infection or

vaccine.

severe acute respiratory syndrome (SARS) A highly contagious, potentially life-threatening lower respiratory tract illness that usually starts with flu-like symptoms, including fever, headache, and muscle aches, followed within 2 to 7 days by a dry cough and pneumonia.

vector An organism that carries a disease-causing microorganism from one organism to another.

ventilator-associated pneumonia (VAP) A type of hospital-acquired pneumonia that occurs in ventilated patients and that appears more than 48 hours after endotracheal intubation.

viremia The presence of viruses in the blood.

virulence Physical or biochemical properties of a disease agent that determine its pathogenicity.

West Nile virus (WNV) An arthropod-borne flavivirus that is spread to birds and humans by mosquitoes, which may be characterized by sudden onset of fever with malaise, anorexia, nausea, vomiting, eye pain, headache, myalgia, rash, lymphadenopathy, fatigue, and arthralgias.

Bibliography

Ahya SN, Flood K, Paranjothi S, eds. *The Washington Manual of Medical Therapeutics,* 30th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2001.

Anderson RR, Harrington LC. West Nile virus. Cornell University Medical Entomology Extension Web page. 2006. Available at: http://www.entomology.cornell.edu/MedEnt/WestNileFS/WestNileFS.html. Accessed July 10, 2009.

Bischoff WE, Reynolds TM, Sessler CN, et al. Handwashing compliance by health care workers: the impact of introducing an accessible, alcohol-based hand antiseptic. *Arch Intern Med.* 2000;160:1017–1021.

Bolyard EA, Tablan OC, Williams WW, et al; the Hospital Infection Control Practices Advisory Committee. Guidelines for infection control in healthcare personnel, 1998 [published correction appears in *Infect Control Hosp Epidemiol.* 1998;19:493]. *Infect Control Hosp Epidemiol.* 1998;19:407–463.

Bolyard EA, Tablan OC, Williams WW, et al; Hospital Infection Control Practices Advisory Committee. Guideline for infection control in health care personnel. *Am J Infect Control.* 1998;26:289–354.

Brocato CE, Miller GT. The next agent of terror? Understanding smallpox and its implications for prehospital crews. *J EMS.* 2002;27:44–50, 52–55.

Burnet M. *Natural History of Infectious Disease.* Cambridge, England: Cambridge University Press; 1962.

Cardo DM, Culver DH, Ciesielski CA, et al; Centers for Disease Control and Prevention Needlestick Surveillance Group. A case-control study of HIV seroconversion in health care workers after percutaneous exposure. *N Engl J Med.* 1997;337:1485–1490.

Centers for Disease Control and Prevention. Community-associated MRSA information for the public. Last updated June 30, 2008. Available at: http://www.cdc.gov/ncidod/dhqp/ar_mrsa_ca_public.html. Accessed July 27, 2009.

Centers for Disease Control and Prevention, Department of Health and Human Services. Severe acute respiratory syndrome (SARS): in the absence of SARS-CoV transmission worldwide: guidance for surveillance, clinical and laboratory evaluation, and reporting version 2. May 3, 2005. Available at:

http://www.cdc.gov/ncidod/sars/absenceofsars.htm. Accessed May 2009.

Centers for Disease Control and Prevention, Department of Health and Human Services. Recommendations for Isolation Precautions in Hospitals. Updated: February 18, 1997. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf. Accessed May 2009.

Centers for Disease Control and Prevention, Department of Health and Human Services. Severe acute respiratory syndrome (SARS): fact sheet: basic information about SARS. May 3, 2005. Available at: http://www.cdc.gov/ncidod/sars/factsheet.htm. Accessed May 2009.

Centers for Disease Control and Prevention, Department of Health and Human Services. Severe acute respiratory syndrome (SARS): public health guidance for community-level preparedness and response to severe acute respiratory syndrome (SARS) version 2: supplement I: infection control in healthcare, home, and community settings. IV. Infection Control for Prehospital Emergency Medical Services (EMS). May 3, 2005. Available at: http://www.cdc.gov/ncidod/sars/guidance/I/prehospital.htm. Accessed May 2009.

Centers for Disease Control and Prevention, Division of Vector-Borne Infectious Diseases. West Nile virus: questions and answers. Last updated April 26, 2004. Available at: http://www.cdc.gov/ncidod/dvbid/westnile/qa/overview.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control. 3rd Revision. 2003. Available at: http://www.cdc.gov/ncidod/dvbid/westnile/resources/wnv-guidelines-aug-2003.pdf. Accessed July 24, 2009.

Centers for Disease Control and Prevention. Epidemiology of HIV/AIDS: United States, 1981–2005. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5521a2.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Get smart: know when antibiotics work. Last updated June 30, 2009. Available at: http://www.cdc.gov/getsmart. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Immunization of health-care workers: recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Hospital Infection Control Practices Advisory Committee (HICPAC). Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/00050577.htm. Accessed July 24, 2009.

Centers for Disease Control and Prevention. The influenza (flu) viruses. Available at: http://www.cdc.gov/flu/about/fluviruses.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Meningitis questions and answers. Updated June 24, 2009. Available at: http://www.cdc.gov/meningitis/about/faq.html. Accessed July 27, 2009.

Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Healthcare Quality and Promotion, and Division of Viral Hepatitis. Exposure to blood: what healthcare personnel need to know. July 2003. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/bbp/Exp_to_Blood.pdf. Accessed May 2009.

Centers for Disease Control and Prevention. Progress toward strengthening blood transfusion services: 14 countries, 2003–2007. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5747a2.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Protecting the nation's health in an era of globalization: CDC's global infectious disease strategy. Available at: http://www.cdc.gov/globalidplan/global_id_plan.pdf. Accessed July 24, 2009.

Centers for Disease Control and Prevention. Recognition of illness associated with the intentional release of a biologic agent. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5041a2.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Surveillance of healthcare personnel with HIV/AIDS, as of December 2002. Available at: http://www.cdc.gov/ncidod/dhqp/bp_hiv_hp_with.html. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Surveillance of occupationally acquired HIV/AIDS in healthcare personnel, as of December 2006. Available at: http://www.cdc.gov/ncidod/dhqp/bp_hcp_w_hiv.html. Accessed July 24, 2009.

Centers for Disease Control and Prevention. TABLE II. (Part 5) Provisional cases of selected notifiable diseases, United States, week ending June 28, 2008. Available at: http://wonder.cdc.gov/mmwr/mmwr_reps.asp?mmwr_table=2E&mmwr_year=2008&mmwr_week=26. Accessed July 10, 2009.

Centers for Disease Control and Prevention. Tickborne rickettsial diseases. Available at: http://www.cdc.gov/ticks/index.html. Accessed July 10, 2009.

Centers for Disease Control and Prevention. Vancomycin-resistant enterococci (VRE) and the clinical laboratory. December 1999. Available at: http://www.cdc.gov/ncidod/dhqp/ar_lab_vre.html. Accessed July 27, 2009.

Centers for Disease Control and Prevention. Viral hepatitis. Last updated April 25, 2009. Available at: http://www.cdc.gov/hepatitis/index.htm. Accessed July 27, 2009.

Centers for Disease Control and Prevention. VISA/VRSA: vancomycin-intermediate/resistant *Staphylococcus aureus*. Last updated April 21, 2004. Available at: http://www.cdc.gov/ncidod/dhqp/ar_visavrsa.html. Accessed July 27, 2009.

Chapman AS, Bakken JS, Folk SM, et al. Diagnosis and management of tickborne rickettsial diseases: Rocky Mountain spotted fever, ehrlichioses, and anaplasmosis—United States. A practical guide for physicians and other health care and public health professionals. Available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5504a1.htm. Accessed July 10, 2009.

Craven R, Roehrig J. West Nile virus. *JAMA.* 2001;286:651–653.

Dailey MW, Dunn T. The prehospital needlestick. *Emerg Med Services.* 2000;2968–76.

de Gans, J, van de Beek, D; for the European Dexamethasone in Adulthood Bacterial Meningitis Study Investigators. Dexamethasone in adults with bacterial meningitis. *N Engl J Med.* 2002;347:1549–1556.

Dorevitch S, Forst L. The occupational hazards of emergency physicians. *Am J Emerg Med.* 2000;18:300–311.

Drazen J. SARS: looking back over the first 100 days. *N Engl J Med.* 2003;349:319–320.

Drotman DP. Emerging infectious diseases: a brief biographical heritage. Last updated December 29, 2005. Available at: http://www.cdc.gov/ncidod/eid/vol4no3/drotman.htm. Accessed July 27, 2009.

Durand ML, Calderwood SB, Weber DJ, et al. Acute bacterial meningitis in adults: a review of 493 episodes. *N Engl J Med.* 1993;328:21–28.

Ellis JS, Alvarez-Aguero A, Gregory V, et al. Influenza AH1N2 viruses, United Kingdom, 2001–2002 influenza season. *Emerg Infect Dis.* 2003;9:304–310.

English JF, Cundiff MY, Malone JD, et al; APIC Bioterrorism Task Force, and CDC Hospital Infections

Program Bioterrorism Working Group. Bioterrorism Readiness Plan: A Template for Healthcare Facilities. April 13, 1999. Available at: http://www.cdc.gov/ncidod/dhqp/pdf/bt/13apr99APIC-CDCBioterrorism.PDF. Accessed July 27, 2009.

Flowers LK, Mothershead JL, Blackwell TH. Bioterrorism preparedness, II: the community and emergency medical services system. *Emerg Med Clin North Am.* 2002;20:457–476.

Foster DA, Heller ST, Young JK. Multidrug-resistant *Streptococcus pneumoniae* [letter]. *N Engl J Med.* 2001;344:1329.

Fridkin SK, Gaynes RP. Antimicrobial resistance in intensive care units. *Clin Chest Med.* 1999;20:303–316.

Fridkin SK. Increasing prevalence of antimicrobial resistance in intensive care units. *Crit Care Med.* 2001;29(4 suppl):N64–N68.

Gates RH. *Infectious Disease Secrets,* 2nd ed. Philadelphia, PA: Hanley and Belfus; 2003.

Gerberding JL. Occupational exposure to HIV in health care settings. *N Engl J Med.* 2003;348:826–833.

Gladwin M, Tattler B. *Clinical Microbiology Made Ridiculously Simple,* 3rd ed. Miami, FL: MedMaster Inc; 2001.

Henderson DK, Fahey BJ, Willy M, et al. Risk for occupational transmission of human immunodeficiency virus type 1 (HIV-1) associated with clinical exposures: a prospective evaluation. *Ann Intern Med.* 1990;113:740–746.

Hoffmann C, Rockstroh JK, Kamps BS, eds. *HIV Medicine 2007,* 15th ed. Paris, France: Flying Publisher; 2007. Available at: http://HIVMedicine.com/hivmedicine2007.pdf. Accessed July 9, 2009.

Kamps BS, Hoffmann S, eds. *SARS Reference,* 3rd ed. 2003. Available at: http://www.sarsreference.com. Accessed July 27, 2009.

Kemp SF, Lockey RF, Simons FE; World Allergy Organization ad hoc Committee on Epinephrine in Anaphylaxis. Epinephrine: the drug of choice for anaphylaxis: a statement of the World Allergy Organization. *Allergy.* 2008;63:1061–1070.

Komar N, Panella N, Burns J, et al. Serologic evidence of West Nile virus infection in birds in the New York City vicinity during an outbreak in 1999. *Emerg Infect Dis.* 2001;7:621–625.

Ksiazek TG, Erdman D, Goldsmith CS, et al; SARS Working Group. A novel coronavirus associated with severe acute respiratory syndrome. *N Engl J Med.* 2003;348:1953–1966.

Lauer GM, Walker BD. Hepatitis C virus infection [published correction appears in *N Engl J Med.* 2001;345:1425]. *N Engl J Med.* 2001;345:41–52.

Levinson W, Jawetz E. *Medical Microbiology and Immunology,* 7th ed. New York, NY: Lange Medical Books/McGraw-Hill Companies; 2002.

Ligon BL. Robert Kock: Nobel laureate and controversial figure in tuberculin research. *Semin Pediatr Infect Dis.* 2002;13:289.

Louie JP, Bell LM. Appropriate use of antibiotics for common infections in an era of increasing resistance. *Emerg Med Clin North Am.* 2002:20.

Maryland Department of Health and Mental Hygiene. Informed consent and agreement to HIV testing. DHMH AIDS Administration Form 98-2. Available at: http://74.125.95.132/search?q=cache:ehJOENj3qBIJ:www.dhmh.state.md.us/AIDS/ProviderResources/CTR/English%2520Consen2&cd=2&hl=en&ct=clnk&gl=us&client=safari. Accessed July 27, 2009.

Morse DL. West Nile virus: not a passing phenomenon. *N Engl J Med.* 2003;348:2173–2174.

Nash D, Mostashar F, Fine A, et al. The outbreak of West Nile virus infection in the New York City area in 1999. *N Engl J Med.* 2001;344:1807–1814.

National Institute for Occupational Safety and Health. What Every Worker Should Know: How to Protect Yourself From Needlestick Injuries. July 1997. NIOSH publication 2000-135. Available at: http://www.cdc.gov/niosh/docs/2000-135. Accessed May 2009.

Petersen L, Marfin A. West Nile virus: a primer for the clinician. *Ann Intern Med.* 2002;137:173–179.

Petersen L, Roehrig J. West Nile virus: a reemerging global pathogen. *Emerg Infect Dis.* 2001;6:611–614.

Peterson JW. Bacterial pathogenesis. Available at: http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=mmed.chapter.537. Accessed July 10, 2009.

Pumphrey RSH. Lessons for management of anaphylaxis from a study of fatal reactions. *Clin Exp Allergy.* 2000;30:1144–1150.

Rollins DM, Briken V, Joseph SW. Pathogenic microbiology: the study of disease-causing bacteria. NSCI home page. Revised September 2006. Available at: http://life.umd.edu/classroom/bsci424. Accessed July 24, 2009.

Roos KL. Acute bacterial meningitis. *Semin Neurol.* 2000;20:293–306.

Rosenstein NE, Perkins BA, Stephens DS, et al. Meningococcal disease. *N Engl J Med.* 2001;344:1378–1388.

Schrag SJ, Brooks JT, Beneden CV, et al. SARS surveillance during emergency public health response, United States, March–July, 2003. *Emerg Infect Dis.* 2004;10:185–194.

Schultz CH, Mothershead JL, Field M. Bioterrorism preparedness, I: the emergency department and hospital. *Emerg Med Clin North Am.* 2002;20(2):437–455.

Sepkowitz KA. AIDS: the first 20 years. *N Engl J Med.* 2001;344:1764–1772.

Simons FER, Frew AJ, Ansotegui IJ, et al. Risk assessment in anaphylaxis: current and future approaches. *J Allergy Clin Immunol.* 2007;120(1 suppl):S2–S4.

Small PM, Fujiwara PI. Management of tuberculosis in the United States. *N Engl J Med.* 2001;345:189–200.

Snacken R, Kendal AP, Haaheim KR, et al. The next influenza pandemic: lessons from Hong Kong, 1997. *Emerg Infect Dis.* 1999;5:195–203.

Suerbaum S, Michetti P. *Helicobacter pylori* infection. *N Engl J Med.* 2002;347:1175.

Thomas KE, Hasbun R, Jekel J, et al. The diagnostic accuracy of Kernig's sign, Brudzinski's sign, and nuchal rigidity in adults with suspected meningitis. *Clin Infect Dis.* 2002;35:46–52.

Todar K. Todar's Online Textbook of Bacteriology. 2003. Available at: http://www.textbookofbacteriology.net. Accessed July 27, 2009.

Tortora GJ, Funke BR, Case CL. *Microbiology: An Introduction,* 10th ed. San Francisco, CA: Benjamin Cummings; 2009.

Tyler KL. West Nile virus encephalitis in America [editorial]. *N Engl J Med.* 2001;344:1858–1859.

Update: West Nile virus activity: United States, 2001. *Morb Mort Wkly Rep.* 2002;51:497–501.

US Army Medical Research Institute of Infectious Diseases. *USAMRIID's Medical Management of Biological Casualties Handbook,* 5th ed. Fort Detrick, Frederick, MD: USAM-RID;2004. Available at:

http://www.usamriid.army.mil/education/bluebookpdf/USAMRIID%20Blue%20Book%205th%20Edi Accessed July 10, 2009.

US Department of Health and Human Services. National Vaccine Program Office. Last revised February 12, 2004. Available at: http://www.hhs.gov/nvpo/pandemics/fluprint.htm. Accessed July 27, 2009.

US Department of Labor, Occupational Safety and Health Administration. Occupational Exposure to Bloodborne Pathogens: Precautions for Emergency Responders. 1998. OSHA publication 3130. Available at: http://www.nccer.org/documents/safety/OccupationalExposureToBloodbornePathogens1998OSHA31 Accessed July 10, 2009.

Vu HT, Leitmeyer KC, Le DH, et al. Clinical description of a completed outbreak of SARS in Vietnam, February–May 2003. *Emerg Infect Dis.* 2004;10:334–338.

Warren DK, Fraser VJ. Infection control measures to limit antimicrobial resistance. *Crit Care Med.* 2001;29 (4 suppl):N128–N134.

Webster RG. Influenza: an emerging disease. *Emerg Infect Dis.* 1998;4:436–441.

Weir E. West Nile fever heads north. *CMAJ.* 2000;163:878.

Wenzel RP. Airline travel and infection [editorial]. *N Engl J Med.* 1996;334:981–982.

Wenzel RP, Edmond MB. Managing antibiotic resistance [editorial]. *N Engl J Med.* 2000;343:1961–1963.

Wenzel RP, Edmond MB. Managing SARS amidst uncertainty. *N Engl J Med.* 2003;348:1947–1948.

West K. AIDS update: occupational exposure and postexposure treatment of HIV/AIDS. *JEMS.* 2002;27:48–60, 62–63.

WHO/SEARO. Emerging infectious diseases. Broadcast on World Health Day, 7 April 1997, on the theme of control of infectious diseases. Available at: http://www.searo.who.int/en/Section980/Section1162/Section1167/Section1171_4750.htm. Accessed July 27, 2009.

Willams WW, Preblud SR, Reichelderfer PS, et al. Vaccines of importance in the hospital setting: problems and developments. *Infect Dis Clin North Am.* 1989;3:701–722.

Wong RSM, Hui DS. Index patient and SARS outbreak in Hong Kong. *Emerg Infect Dis.* 2004;10:339–341.

Toxicologic Emergencies

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Objectives

1. Identify issues that may adversely affect the health and safety of critical care transport professionals during the treatment and transport of patients experiencing a toxicologic emergency (p 800–803).
2. Describe priority interventions and considerations common to all patients following a poisoning or overdose (p 806–809).
3. Discuss the assessment findings associated with the various toxidromes and medication reaction syndromes encountered in the critical care environment (p 804–806).
4. Describe the management of a patient experiencing a toxicologic emergency (p 806–809).
5. Discuss situations in which decontamination is required or indicated following a toxic exposure or overdose (p 806, 829).
6. Identify which chemicals or medications require enhanced elimination (removal of absorbed toxins) from the body (p 812).
7. Discuss the risks and benefits of and the techniques for administration of various antidotes (p 812–828).
8. Identify the clinical presentation, mechanism of toxicity, and treatment of poisoning or overdose situations commonly encountered or requiring specialized management by critical care transport professionals (p 812–828).
9. Discuss hazardous materials response and critical care transport considerations (p 828–830).
10. Discuss radiation emergencies and critical care transport considerations (p 830–831).

Introduction

CCTPs may be asked to transport patients experiencing a toxicologic emergency following a vast array of possible events. Poisons, chemicals, medications, and other toxic substances are found in dangerous quantities in homes, schools, agriculture, industry, commercial establishments, on all modes of transportation, and naturally in the environment. Through accidents, carelessness, intentional misuse, abuse, and the intentional acts of others, people may be exposed to these toxins and experience potentially lethal consequences. CCTPs can dramatically improve patient outcomes through prompt recognition and effective treatment of toxicologic emergencies.

The safety of the transport team and other health care or emergency responders is the highest priority in the approach to a poisoned or overdosed patient. Patients present hazards to rescuers in a variety of ways. Bizarre, aggressive, or violent behavior may accompany or follow a medication overdose. Suicidal patients often jeopardize the safety of others during a suicide attempt. Patients exposed to hazardous

chemicals may contaminate and injure unprotected rescuers if thorough decontamination is not performed before treatment is initiated.

Care for a poisoned patient or one with an overdose is based on maintaining a patent airway, supporting respirations, and promoting effective circulation. Additional steps involve protecting the patient from additional injury through decontamination, prevention of absorption, antidote administration, enhanced elimination, and general patient safety and comfort measures.

Assessment

Safety

Assessment of a patient after poisoning or an overdose ideally should begin well in advance of the first face-to-face contact with the patient. In many situations, if rescuers or health care providers do not use the correct personal protective equipment (PPE) before initial patient contact, it will be too late for this equipment to provide adequate protection **Figure 20-1**. As the transport team receives the initial dispatch or patient report, safety precautions and potential hazards should be considered. CCTPs may encounter hazardous situations during anticipated patient transports between health care facilities, while responding to an emergency in the community, or by happening on a vehicular crash without any warning.

Cues to the presence of toxic substances may include the following:

- The presence of unusual odors, smoke, or vapors
- Signs, placards, or markings indicating chemicals or hazardous materials
- Vehicles known to carry chemicals (such as tank cars and fuel trucks)
- Industrial, manufacturing, agricultural, or laboratory facilities
- Bystanders or rescuers becoming unexpectedly ill or unconscious
- Multiple patients with similar unexpected signs and symptoms

Figure 20-1 Adequate use of personal protective equipment during contact with patients limits the risk to rescuers and health care providers. The four levels of protection include levels A through D. **A.** Level A protection is fully encapsulating, including the self-contained breathing apparatus (SCBA). **B.** Level B protection is worn with an SCBA but is not fully encapsulating. **C.** Level C protection is for a known agent. **D.** Level D protection is sometimes worn in the cold zone, an area where no environmental hazards

should be present.

In addition to chemical hazards posed to health care providers and emergency responders, patients may exhibit violent, bizarre, or aggressive behavior. Hospital security staff, law enforcement personnel, or a sufficient number of trained health care providers may be necessary to restrain an aggressive or confused patient before any real assessment or treatment can begin. Intoxicated patients and patients with psychological or emotional disorders pose a significant risk to health care providers and emergency responders. All patients should be evaluated for the presence of weapons before transport. Physical and chemical restraints should be considered for intoxicated patients and for patients with a psychological or emotional disorder. CCTPs should not approach an unsecured scene without law enforcement involvement when there is an increased likelihood of aggression or violence.

The following are cues to an increased likelihood of aggression or violence:

- Reported suicide attempt or significant mental illness
- History of violent behavior
- Involvement of alcohol, illicit drugs, or medication or chemical abuse
- Altered or confused mental status
- Patients with escape risk (such as prisoners and inpatient psychiatric patients)

Once the safety concerns for health care providers and emergency responders have been adequately addressed, the transport team should evaluate and manage any problems with the patient's ABCs. The overall patient assessment should be focused on identifying immediate threats to life and health, yet be comprehensive enough to disclose the subtle cues that may assist in the diagnosis of a specific type of toxic exposure.

Airway

Maintaining or establishing a patent airway remains the top priority in the management of a patient with a toxic exposure. Transport team members must evaluate whether the patient currently has a patent airway and whether the patient is at significant risk for developing airway compromise during the transport. Patients who are awake, alert, and speaking without difficulty demonstrate a patent airway. Unusual sounds from the airway, such as stridor, snoring respirations, and gurgling noises, indicate that an airway is at least partially compromised. In addition, visual signs such as secretions, blood, edema, and foreign substances in the airway indicate a potentially compromised airway. CCTPs should continually monitor patients, particularly patients exposed to a vaporous toxin, such as anhydrous ammonia. Vaporous corrosives continue to react and may produce progressive airway occlusion over several minutes.

The presence of an endotracheal (ET) tube does not guarantee a patent airway. An ET tube can become clogged, can become dislodged, or may not have been placed correctly. Auscultation of lung sounds and epigastric sounds, continuous observation of end-tidal carbon dioxide ($ETCO_2$), and observation of the clinical status of a patient are necessary to confirm that an ET tube is functioning correctly. Other nontracheal airway devices provide less protection and require greater scrutiny during patient assessment.

A previously patent airway may become compromised during patient transport for a variety of reasons. Any subsequent alteration in mental status or seizure activity may compromise an airway in a poisoned patient or a patient with an overdose. Evolving structural abnormalities from trauma or increasing edema may also compromise a previously patent airway. Obstructions may occur from emesis (especially unrecognized), secretions, and a displaced foreign body during the transport, leading to airway compromise.

Patients who received activated charcoal or syrup of ipecac (agents used for gastrointestinal [GI] decontamination) present an unusual risk of subsequent airway compromise. Syrup of ipecac may induce vomiting as long as 3 to 4 hours after administration. In supine or sedated patients, aspiration may easily occur. Aspiration of activated charcoal from an inadequately protected airway is often lethal. In patients with unprotected airways who have been given ipecac or charcoal, some protection from aspiration can be afforded by maintaining a minimum of 30° elevation of the head of the stretcher at all times. Aspiration is often silent, so if there is any doubt about the patient's ability to protect the airway during transport, the CCTP should secure the airway with an ET tube.

Breathing

Breathing is second only to airway in importance during patient assessment. Patients require effective oxygen delivery and ventilation to supply the body with oxygen and to remove carbon dioxide. Without effective oxygenation and ventilation, tissue hypoxia, acidosis, and cell death will occur.

The CCTP must evaluate whether a patient's oxygenation and ventilation are adequate to meet metabolic needs. Patients who are at risk for subsequent respiratory compromise must also be identified so the appropriate interventions may begin.

A patient's intrinsic or ventilator-controlled rate and tidal volume must provide adequate gas exchange for his or her clinical status. Physiologic stress, acidosis, hypermetabolic states (such as in pediatrics), and many toxic substances require greater oxygenation and ventilation than would normally be required. The CCTP must consider these factors when evaluating a patient's respiratory status.

Clues in the patient's history of present illness and physical examination will alert the CCTP to the increased likelihood of impending respiratory compromise. Many toxic exposures will affect a patient's respiratory status directly or indirectly. Direct toxins, including asphyxiates, organophosphates, and neuromuscular blockers, affect gas exchange, airway diameter, lung tissue, and ventilation. Indirect toxins, such as opiates, benzodiazepines, tricyclic antidepressants (TCAs), and ethanol, inhibit a patient's central respiratory drive. Hypoxia may occur from gases that displace oxygen in the lungs, substances that block cellular oxygenation, and agents that cause cardiogenic or noncardiogenic pulmonary edema. Respiratory compromise may also develop as a result of associated trauma, burns, or aspiration.

Cyanosis, fatigue, or dyspnea indicates that a patient's respiratory status may be compromised. Lung auscultation revealing wheezing, pulmonary edema, and decreased, absent, or coarse breath sounds further indicates unstable respiratory status.

Circulation

The evaluation of a patient's circulation requires assessment of end-organ tissue perfusion and overall cardiovascular functioning. Previously healthy people may experience vast alterations in hemodynamic parameters without losing end-organ tissue perfusion. Conversely, patients with longstanding cardiovascular disease may experience devastating consequences to modest hemodynamic alterations that are normally well tolerated in healthier people.

Patients with intact circulatory functioning will have adequate end-organ tissue perfusion demonstrated by normal mental status and adequate urine output for their age or weight. Patients will have strong distal pulses and "normal" heart rate, blood pressure, and other hemodynamic values for their age and weight. Skin will be warm and dry with a capillary refill time of less than 2 seconds. The electrocardiogram (ECG) will show a normal sinus rhythm (or the patient's baseline rate and rhythm) without signs of conduction abnormality, unusual ectopy, or ischemia. Any deviation from these findings indicates potentially compromised circulation.

Numerous mechanisms may impair a patient's cardiovascular functioning following an exposure to a

toxic substance. During the evolution of a toxic exposure, a patient's circulatory status may decline because of any of the following:

- Direct exposure to cardiotoxic or vasoactive substances
- Fluid volume loss or redistribution
- Electrolyte disturbance
- Airway or respiratory compromise with secondary cardiac dysfunction
- Manifestations of preexisting cardiovascular disease
- Altered oxygen-carrying capacity

A history of exposure to agents with the potential to cause any of the aforementioned events provides a cue that a patient's circulatory status may become impaired before, during, or after transport.

Additional Assessment

Patient assessment is not limited to safety and the ABCs. Every patient transported should ideally be assessed with a thorough history and physical examination. The depth and detail of the additional assessment should be balanced against the needs for prompt transportation and/or immediate clinical interventions.

The history of present illness should include the following:

- The medication(s) or substance(s) involved (including multiple substances, particularly with intentional poisonings)
- Time, quantity, and/or duration of exposure
- Route(s) of exposure
- The patient's initial and subsequent clinical status
- Initial decontamination, stabilization, and treatment performed before arrival of the transport team
- Other contributory information, such as accidental vs intentional event, multiple patients, and the presence of environmental hazards

Interviews of patients, family, and bystanders may provide valuable clues to the nature and severity of a toxic exposure. Through interviews, the CCTP may become aware of **prodromal** symptoms not otherwise discoverable during the physical examination (such as nausea, headache, and dizziness). The interview may be abbreviated or completed during the transport if interventions or transport cannot be delayed.

Knowing a patient's medical and social history, personal medications, and allergies will assist health care providers treating and transporting a patient. Preexisting medical problems may have a profound effect on the severity of a toxic exposure or overdose. Long-term exposures to various substances often present much differently from a single, brief, acute exposure to the same substance. Other factors such as a history of a psychiatric disorder or substance abuse will impact the present treatment plan and the discharge and follow-up options.

The physical examination must be appropriate for the clinical situation. Transport teams must balance the usefulness of a complete head-to-toe physical examination with concerns for patient thermoregulation (hyperthermia or hypothermia), privacy, patient access in confined transport vehicles, and the clinical needs of that patient. It is inappropriate to delay transport of a critically ill patient by performing a time-consuming comprehensive history and head-to-toe examination; it may be equally inappropriate to initiate rapid transport before adequately assessing the patient.

Vital Signs

A vast array of toxic substances will cause altered vital signs. Hemodynamic and respiratory alterations occur from direct action on various organs and tissues or through effects on the central nervous system, which regulates the cardiovascular and respiratory systems. The examples that follow provide only a sample of medications and substances causing alterations in vital signs.

Tachycardia following a toxic exposure may occur from several different classes of medications and chemicals. Sympathomimetics, such as amphetamines, caffeine, and cocaine, produce an increase in sympathetic tone and atrioventricular node conduction, increasing the heart rate. Medications with anticholinergic effects (such as atropine, TCAs, and antihistamines) antagonize acetylcholine and enhance sinus node discharge, causing sinus tachycardia. In addition, any substance causing cellular hypoxia (such as carbon monoxide and cyanide) will cause responsive tachycardia. Various medications and chemicals may also induce ventricular tachyarrhythmias or myocardial ischemia with related tachycardia. The list of chemicals and medications with the potential for causing tachycardia is extensive.

Bradycardia may occur early following exposure to a toxic substance or immediately preceding asystole as the exposure becomes fatal. Central nervous system (CNS) depression causing bradycardia occurs with toxicity from ethanol, benzodiazepines (for example, lorazepam, diazepam, and midazolam), clonidine, and others. Cardiac (digitalis) glycosides, organophosphates, and carbamates cause bradycardia as a result of an increase in vagal tone. Bradycardia may also occur from direct action on myocardial cell membranes produced by calcium channel blockers, beta-adrenergic blockers, tricyclic antidepressants, household poisonings, and others.

Hypertension accompanies tachycardia and bradycardia following exposure to various medications and chemicals. The sympathomimetic agents and anticholinergic agents described in the preceding paragraphs each produce hypertension with tachycardia. Norepinephrine and phenylephrine are examples of agents that can cause hypertension with bradycardia or an atrioventricular block.

Hypotension may also be accompanied by tachycardia or bradycardia. Hypotension with tachycardia may result from fluid loss, third spacing, or blood vessel dilation. Cell membrane depressant agents and agents that depress the CNS can cause hypotension and bradycardia. These include opioids, sedative-hypnotic agents, beta-blockers, calcium channel blockers, and various others.

Temperature regulation is impaired by many chemicals and medications. Hyperthermia occurs from increased muscle activity or seizures, an increased metabolic rate, impaired heat dissipation, and disrupted thermoregulation. Drug withdrawal and reactions to medications at therapeutic doses also precipitate hyperthermia in specific high-risk populations. Examples of medications that may cause hyperthermia include cocaine, salicylates, and phenothiazines. Examples of medications that may cause hypothermia include beta-blockers, benzodiazepines, ethanol, and hypoglycemic agents.

Vasodilators and CNS depressant agents cause hypothermia. In addition to alterations in metabolism, vasodilation, and impaired shivering response, a patient exposed to CNS depressants may lose consciousness in a cold environment, leading to exogenous hypothermia.

Laboratory Assessment

Laboratory analysis can provide great assistance to the CCTP during diagnosis and treatment of a patient with an actual or potential toxic exposure. Analysis of blood and urine samples is routinely available at sending hospitals and clinics. Advances in **point-of-care testing** technology allow CCTPs to measure blood chemistry values, arterial and venous blood gases, and other assorted values during the transport and obtain results almost immediately. CCTPs should be aware, however, that not all chemicals are included in standard toxicology screens.

Certain toxic exposures require a specific approach to laboratory evaluation once the diagnosis is

made. In these circumstances, the toxic substance has a predictable effect on various laboratory values, and the tests are used to guide treatment or monitor patient status. CCTPs do not often have the luxury of a definitive diagnosis before transport and must use available laboratory testing as a screening or an assessment tool.

Patients who are exposed to a chemical or medication and who arrive unresponsive or with altered mental status cannot provide a reliable history and require a thorough screening for life-threatening conditions. In addition, suicidal and psychiatric patients may intentionally provide misleading or incomplete information to health care providers. The laboratory assessment should include the following:

- Urine drug screen, which must include TCAs unless tested for separately
- Acetaminophen level 4 hours after exposure when time of ingestion is known, or immediately when time of ingestion is not known
- Salicylate level (drawn immediately for a baseline and then at least 1 to 2 hours after exposure; repeated testing needed for positive value or a possibility of delayed absorption)
- Serum electrolytes, including glucose and potassium (particularly if paralysis with succinylcholine [Anectine] is a possibility)
- Evaluation of acid-base status through arterial blood gas or serum carbon dioxide testing
- Ethanol level
- Serum levels of any chemicals and medications known to be available to the patient for which tests can be done (for example, digoxin, lithium, phenytoin [Dilantin], and valproic acid [Depakote])
- Anion gap (serum sodium level minus the sum of chloride and bicarbonate; normal level, 8 to 16 mEq/L)
- Osmolar gap if toxic alcohol poisoning is suspected

The listed tests are merely for screening purposes. If any abnormal or positive results are obtained, further investigation is essential. Known exposure to a particular medication or chemical or profound clinical instability may require a more comprehensive laboratory assessment.

Toxic Syndromes (Toxidromes) and Medication Reaction Syndromes

Exposure to certain classes of chemicals and medications will produce a readily identifiable pattern of clinical signs and symptoms, called a **toxidrome**. These patterns aid in diagnosis when the exact nature of the exposure is unclear and permit health care providers to initiate the correct lifesaving interventions. CCTPs should use caution not to overlook other possible causes when making treatment decisions based solely on identification of a toxidrome.

Patients may also experience life-threatening reactions to medications taken at therapeutic doses. Prompt recognition of these rare events is essential and will happen only if health care providers recognize the unusual clues or at-risk patients. Misdiagnosis of these events will adversely affect patient care, with possibly lethal consequences.

Table 20-1 lists some of the most common toxidromes and medication reaction syndromes.

Toxic Syndromes (Toxidromes)

Anticholinergic syndrome occurs following excessive exposure to medications such as antihistamines, atropine, and benztropine (Cogentin), or other substances such as Jimson weed. Poisoning results in muscarinic receptor blockade at the neuromuscular junction. This syndrome is characterized by tachycardia, hyperthermia, dilated (mydriatic) pupils, warm (or hot) dry skin, ileus, delirium, seizures,

psychosis, and urinary retention. A common phrase to remember this syndrome is: "Mad as a hatter (psychosis), red as a beet (hot, flushed skin), dry as a bone (dry mucous membranes and urinary retention), blind as a bat (pupil dilation), and hot as a hare (hyperthermia)." Seizure activity is treated with benzodiazepines (such as lorazepam, diazepam, and midazolam) and phenobarbital. Severe toxicity is treated with physostigmine (Antilirium, Eserine), administered following consultation with a toxicologist.

Cholinergic syndrome (**cholinesterase** inhibitor toxicity) typically occurs following exposure to organophosphate and carbamate insecticides or to certain chemical nerve agents. Cholinesterase inhibitors may affect nicotinic receptors, muscarinic receptors, or both and alter the function of the neurotransmitter acetylcholine. Patients may have two distinct patterns of toxicity, depending on which receptor(s) are involved. Nicotinic receptor toxicity produces tachycardia, hypertension, fasciculations, weakness, hyperglycemia, and dilated pupils. Muscarinic receptor toxicity produces the classic **SLUDGEM** syndrome (Salivation, Lacrimation, Urination, Diarrhea, Gastroenteritis, Emesis, and Miosis) plus bronchorrhea, sweating, and bradycardia. The mnemonic **DUMBELS** can also be used to remember these symptoms (Diaphoresis/diarrhea, Urination, Miosis, Bradycardia/bronchospasm/bronchorrhea, Emesis, Lacrimation, and Salivation). Nicotinic and muscarinic toxicity produce altered mental status, coma, and seizure activity in severe exposures. It is essential that health care providers and rescuers use adequate PPE during evaluation and treatment of exposed patients. Atropine, in doses much higher than in typical Advanced Cardiac Life Support protocols, and pralidoxime are the antidotes for cholinesterase inhibitor toxicity. Seizure activity is treated with benzodiazepines.

TABLE 20-1 Toxidromes and Medication Reaction Syndromes

Toxidromes	Anticholinergic syndrome Cholinergic syndrome Opioid syndrome Sympathomimetic syndrome
Medication Reaction Syndromes	Malignant hyperthermia Neuroleptic malignant syndrome Serotonin syndrome

Signs and Symptoms

Anticholinergic Syndrome

- Tachycardia
- Hyperthermia
- Dilated (mydriatic) pupils
- Warm (or hot) dry skin
- Ileus
- Delirium
- Seizures

- Psychosis
- Urinary retention

Opioid syndrome is frequently encountered by prehospital and emergency department health care providers. This toxicity commonly develops following illicit use or abuse of opioids in the community, or as an adverse consequence of a therapeutic error or accidental ingestion of opioids (for example, morphine, heroin, and fentanyl [Duragesic]). Signs include lethargy and sedation, hypoventilation or apnea, pinpoint pupils, and noncardiac pulmonary edema. Treatment is focused on airway and ventilation support and the prompt administration of an opioid antagonist such as naloxone (Narcan).

CCTPs should be aware that administration of an opioid antagonist may precipitate severe, violent withdrawal symptoms. In addition, many opioids may exert effects much longer than the duration of opioid antagonists, allowing the return of life-threatening symptoms. It is often necessary to use repeated doses of opioid antagonists in severe overdose situations.

Sympathomimetic syndrome involves overstimulation of the adrenergic nervous system, resulting in tachycardia, hypertension, agitation, seizures, hyperthermia, dilated pupils, and diaphoresis. An isolated alpha-adrenergic or beta-adrenergic syndrome may cause bradycardia or hypotension, respectively, but the classic (mixed) syndrome involves the listed symptoms. Amphetamines, caffeine, cocaine, and MDMA (ecstasy) cause sympathomimetic syndrome. Treatment is primarily supportive, with benzodiazepines and aggressive cooling measures. Avoidance or the use of extreme caution is needed with beta-blockers because of the risk of unopposed alpha-adrenergic stimulation causing worsening hypertension.

Signs and Symptoms

Nicotinic Receptor Toxicity

- Tachycardia
- Hypertension
- Fasciculations
- Weakness
- Hyperglycemia
- Dilated pupils

Signs and Symptoms

Muscarinic Toxicity

- SLUDGEM symptoms (Salivation, Lacrimation, Urination, Diarrhea, Gastroenteritis, Emesis, and Miosis)
- DUMBELS symptoms (Diaphoresis/diarrhea, Urination, Miosis, Bradycardia/bronchospasm/bronchorrhea, Emesis, Lacrimation, and Salivation)
- Bronchorrhea
- Sweating
- Bradycardia

Signs and Symptoms

Opioid Toxicity

- Lethargy and sedation
- Hypoventilation or apnea
- Pinpoint pupils
- Noncardiac pulmonary edema

Signs and Symptoms

Sympathomimetic Toxicity

- Tachycardia
- Hypertension
- Agitation
- Seizures
- Hyperthermia
- Dilated pupils
- Diaphoresis

Medication Reaction Syndromes

Malignant hyperthermia is a condition that occurs following administration of succinylcholine or certain inhaled anesthetic agents to genetically susceptible patients. CCTPs may encounter this condition following a rapid sequence intubation (RSI) procedure in the prehospital setting or when responding to a health care facility to perform a patient transport. Patients have muscle spasms, profound muscle rigidity, acidosis (metabolic or respiratory), hyperthermia, tachycardia, tachypnea, myoglobinuria, rhabdomyolysis, and hyperkalemia, beginning up to 12 hours after exposure. Treatment involves aggressive cooling, correction of acidosis, and administration of dantrolene sodium, 2 to 3 mg/kg. Larger patients may require massive quantities of dantrolene for severe reactions; some reports have described the need for more than 36 vials, each containing 20 mg of dantrolene, with 3 g of mannitol. The Malignant Hyperthermia Association of the United States recommends that facilities stock 36 vials of dantrolene to provide initial treatment for a patient weighing 100 to 110 kg, who may require 8 to 10 mg/kg for stabilization.

Neuroleptic malignant syndrome (NMS) is a potentially fatal reaction to antipsychotic and antischizophrenic medications (such as haloperidol [Haldol], prochlorperazine [Compazine], promethazine [Phenergan], and risperidone [Risperdal]), which is not dose dependent and has a 5% to 11% mortality rate overall. Patients often have hyperthermia, profound muscle rigidity, metabolic acidosis, and confusion. Severe manifestations include renal failure, respiratory failure, arrhythmias, and cardiovascular collapse. Supportive treatment involves aggressive cooling and muscle relaxation. Prompt, effective muscle relaxation with nondepolarizing neuromuscular blocking agents, dantrolene sodium, and benzodiazepines will prevent many catastrophic consequences of NMS.

Signs and Symptoms

Malignant Hyperthermia

- Muscle spasms
- Profound muscle rigidity
- Acidosis (metabolic or respiratory)
- Hyperthermia
- Tachycardia
- Tachypnea
- Myoglobinuria
- Rhabdomyolysis
- Hyperkalemia

Signs and Symptoms

Neuroleptic Malignant Syndrome

- Hyperthermia
- Profound muscle rigidity
- Metabolic acidosis
- Confusion

Serotonin syndrome is an unusual response to serotonin-altering medications causing hyperserotoninergic symptoms. Selective serotonin reuptake inhibitors (SSRIs; such as fluoxetine [Prozac], sertraline [Zoloft], and paroxetine hydrochloride [Paxil]), meperidine (Demerol), and monoamine oxidase inhibitors (also called MAOIs; such as phenelzine sulfate [Nardil] and tranylcypromine [Parnate]) are the usual culprits. Amphetamines, TCAs, and lithium are implicated less frequently. Serotonin syndrome may occur following an SSRI (or similar) overdose or when the aforementioned medications are inadvertently combined. Patients have irritability, muscle rigidity, hyperthermia, diaphoresis, headaches, seizures, coma, tachycardia, and hallucinations. Treatment includes aggressive cooling, IV hydration, and seizure control. Muscle relaxation is achieved with sedation and nondepolarizing neuromuscular blocking agents. **Rhabdomyolysis** is treated with sodium bicarbonate and aggressive IV fluid administration. Serotonin antagonist medications have been tested but are not currently recommended for widespread use.

Signs and Symptoms

Serotonin Syndrome

- Irritability
- Muscle rigidity
- Hyperthermia
- Diaphoresis
- Headaches
- Seizures

- Coma
- Tachycardia
- Hallucinations

General Toxicologic Emergency Management

Introduction

Management of toxicologic emergencies includes many interventions common to other areas of critical care transport. Interventions to maintain and enhance the ABCs are discussed in greater depth in Chapter 6. In many toxicologic emergencies, decontamination, enhanced elimination, and antidote administration will not become effective rapidly enough to correct immediately life-threatening problems with the ABCs, although there are a few notable exceptions (such as naloxone for opioid overdose and atropine for acetylcholinesterase toxicity). CCTPs are strongly discouraged from delaying basic ABC interventions while locating, administering, or waiting for a possible antidote to work. Wasted time will prove fatal in many cases.

There are two distinct types of decontamination below, each with a unique purpose. **Primary decontamination** usually is done on a scene or outside a health care facility or transport vehicle to protect rescuers and health care providers from exposure to a toxic substance during patient care or transport. **Secondary decontamination** still requires health care providers to use PPE but is directed at minimizing patient absorption or injury from a toxic substance. The risk to health care providers during secondary decontamination is typically much less than before or during primary decontamination. Gastric decontamination or eye irrigation should be performed well after the ABCs are addressed. Decontamination of a hazardous chemical should occur before an unprotected health care provider makes any contact whatsoever. Both types of decontamination are beneficial to patients, but each must be considered separately to avoid confusion in the following text. Enhanced elimination is the practice of augmenting the body's removal of a toxic substance (or metabolite) once systemic absorption has occurred.

The term *antidote* may be confusing. Certain substances have an identified antidote, which counteracts toxicity through a mechanism clearly related to the particular toxic substance (for example, acetylcysteine [Mucomyst, Acetadote] for acetaminophen overdose). Other toxic exposures cause signs and symptoms amenable to treatment by a variety of medications that treat these symptoms in other situations (for example, benzodiazepines treating seizures caused by a vast array of toxic chemicals or other causes). The line between antidote and other pharmacologic interventions is not important to the outcome but is mentioned to avoid confusion. For many toxic substances, there is a preferred medication to treat a particular sign or symptom, but the term antidote implies a complete reversal of toxicity, which does not usually occur.

Safety

The transport of a patient exposed to a toxic substance should not pose an increased risk to critical care transport personnel if safety considerations are adequately addressed. Potentially suicidal, combative, aggressive, violent, intoxicated, and confused patients should be restrained before transport. Physical restraint includes the spectrum from soft limb restraints, to four-point leather or nylon restraints with additional cross-straps, to handcuffs and shackles for transport of prisoners (prisoners are almost always handcuffed unless they are sedated, intubated, or paralyzed). CCTPs should follow local agency policies when applying or transporting patients in physical restraints.

In addition to physical restraints, CCTPs should consider chemical restraint for patients in the

aforementioned circumstances. Chemical restraint may involve a mild oral sedative and, for extremely high-risk patients, intubation. Local guidelines should be followed for the use of chemical restraint. Air medical crews should consider alternative ground transport if patients cannot be adequately restrained. Ground transport crews should consider taking extra personnel or delaying transport if crew safety cannot be ensured. CCTPs should also be aware of family members, visitors, and prospective passengers who might be carrying weapons or are exhibiting dangerous behavior.

CCTPs risk illness or injury if PPE is not used and adequate decontamination has not occurred before they treat patients exposed to a vast array of toxic substances. Depending on the profile of the critical care transport agency, providers may or may not be actively involved in extrication and decontamination at hazardous material scenes. All transport providers must assess the adequacy of PPE and decontamination before making patient contact or placing the patient in the transport vehicle. Contaminated patients may expose health care providers to potentially lethal substances or incapacitate a pilot or driver, leading to a crash. Decontamination is discussed in greater depth later in this chapter. Crew members should consult a Poison Center at the nationwide number, 1-800-222-1222, Chemtrec at 1-800-262-8200, or a reliable reference source before approaching or transporting a patient exposed to suspected hazardous materials.

Airway

Patients with actual or impending airway compromise require definitive airway management. Endotracheal intubation provides optimal protection for an obtunded or otherwise at-risk patient. If endotracheal intubation is unsuccessful, several additional airway devices will provide varying degrees of protection until endotracheal intubation is performed. Hypoxia and aspiration should be avoided during airway adjunct placement. Devices such as the gum elastic bougie and techniques such as external laryngeal manipulation enhance the likelihood of successful endotracheal intubation. Continuous monitoring of oxygen saturation (SpO_2) and $ETCO_2$ will alert CCTPs to a displaced, clogged, or disconnected ET tube. Laryngeal mask airways and multilumen airways such as the Combitube can be used with continuous $ETCO_2$ monitors and ventilators. Intubated and nonintubated patients require the same degree of observation for airway compromise during critical care transport. Patients exposed to cholinergic or other secretion-producing substances are likely to require frequent ET suctioning.

Breathing

Spontaneously breathing patients may not be oxygenating and ventilating adequately to meet physiologic needs following a poisoning or overdose. Apneic patients require total extrinsic ventilatory support. In either case, oxygenation and ventilation need to be supported externally with positive-pressure ventilation and/or supplemental oxygen.

It is important to provide the correct oxygen concentration and ventilator settings for each patient and clinical situation. In carbon monoxide poisoning situations, for example, patients should receive 100% oxygen, despite normal oxygen saturation readings. The carbon monoxide bound to hemoglobin is misinterpreted as oxygen by conventional pulse oximetry devices, causing falsely elevated readings.

Conventional ventilator settings are often inadequate for a patient's needs following a poisoning or an overdose. Catastrophic consequences may occur if a patient who is hyperventilating to compensate for severe metabolic acidosis is chemically paralyzed and conventional ventilator settings are used. Respiratory compensation no longer protects the body from the devastating effects of the metabolic acidosis, leading to widespread organ, tissue, and cell dysfunction.

Alterations in pH also occur if a patient has underlying metabolic alkalosis and receives aggressive mechanical ventilation, further disrupting the essential pH balance. Any deviations from normal pH

impair the performance of body systems. Pulmonary edema, hypoxia, cardiovascular dysfunction, and hypermetabolic states each require special consideration when initiating and adjusting positive pressure ventilation. Hypermetabolic states require greater ventilation for removal of carbon dioxide than would otherwise be required. Cardiovascular dysfunction, persistent hypoxia, pulmonary edema, and acute respiratory distress syndrome each may benefit from precise adjustment of positive end-expiratory pressure. Chapter 6 provides more discussion on ventilator settings.

Oxygen saturation, blood gas analysis, and $ETCO_2$ provide valuable information when assessing the adequacy of oxygenation and ventilation. Changes in lung sounds, patient appearance, and cardiovascular function (as shown by ECG and assessment of perfusion) indicate improving or worsening respiratory functioning during transport. **Pulse CO-oximetry** is a technology available for noninvasive screening and monitoring for methemoglobinemia and carbon monoxide exposure **Figure 20-2**.

Circulation

Circulatory support for critically ill patients includes managing cardiac function, fluid balance, and vascular tone. Toxic exposures may adversely affect all of these components simultaneously. Impaired circulation may be caused by an airway or breathing problem (resulting in hypoxia and/or ischemia). Impaired airway or breathing may be resolved by a cardiovascular intervention (such as for pulmonary edema).

Figure 20-2 The Masimo Rad-57® Pulse CO-Oximeter™ allows clinicians to noninvasively measure oxygen saturation (SpO_2), carboxyhemoglobin (SpCO) and methemoglobin (SpMet) levels in the blood, as well as total hemoglobin (SpHb) and PVI for fluid responsiveness using a finger sensor.

Cardiac function is managed by medications that increase or decrease the heart rate or contractility. The therapy for an individual patient may be guided entirely by the patient's response or tailored specifically to the toxic substance involved. CCTPs should consult a reliable toxicology reference, a physician, or a Poison Center for precise guidance.

Fluid balance is closely related to vascular tone, kidney function, and electrolyte concentrations. Toxic exposures cause alterations in any of these, requiring corrective action. CCTPs should anticipate fluid volume replacement or diuresis of fluid-overloaded patients when managing circulatory function. In addition, CCTPs may need to remove excess electrolytes, replace them, or change their distribution.

Vascular tone is often managed with IV vasodilator or vasoconstrictor medications. Beta-blockers, calcium channel blockers, catecholamines, and nitrates are used along with specific agents required for peculiar toxic substances to optimize vascular tone. CCTPs should consult a toxicology reference, a physician, or a Poison Center for precise guidance.

Additional General Management

Select patients may require only minimal intervention following a suspected poisoning or drug overdose. A toddler who only possibly ingested a limited quantity of a minimally toxic substance is often managed with only observation for a prescribed period of time. Other patients may become critically ill immediately following a toxic exposure, requiring immediate invasive therapy and exhaustive critical care resources. CCTPs must evaluate the specifics of each situation and weigh the indications, risks, and benefits of each intervention considered for a given patient.

CCTPs must provide continuous assessment and monitoring of any patient being transported. Monitoring must be within the skills of the transport team and adequate for the clinical status of the patient. It is far better to perform excess monitoring (for example, ECG monitoring after exposure to a minimally toxic substance) than inadequate monitoring (for example, no pulse oximetry or capnography on an intubated patient).

Vascular access sufficient for administering emergency medications and fluids should be established in any patient exposed to a potentially harmful substance. Intubated patients require a nasogastric or orogastric tube for stomach decompression and protection from aspiration **Figure 20-3**. Patients who are unresponsive and patients requiring urine output monitoring should have an indwelling urinary catheter inserted and attached to a urine collection device.

Every patient must receive adequate sedation and analgesia while chemically paralyzed and should be prophylactically medicated against seizures. Vital signs (especially following a poisoning or overdose) are *not* a reliable method of measuring patient sedation or comfort. CCTPs frequently encounter unwarranted administration of antihypertensive medications to patients who are chemically paralyzed but lacking adequate analgesia or sedation: Discomfort is a common cause of unexplained hypertension and tachycardia in an intubated, chemically paralyzed patient. Analgesia and sedation should be administered frequently enough to avoid subtherapeutic levels. In addition, patients with increased drug tolerance or hypermetabolic states require significantly more sedation and analgesia while chemically paralyzed. Modern noninvasive commercial devices (such as the bispectral index monitor) that permit monitoring of sedation in intubated and/or chemically paralyzed patients are available.

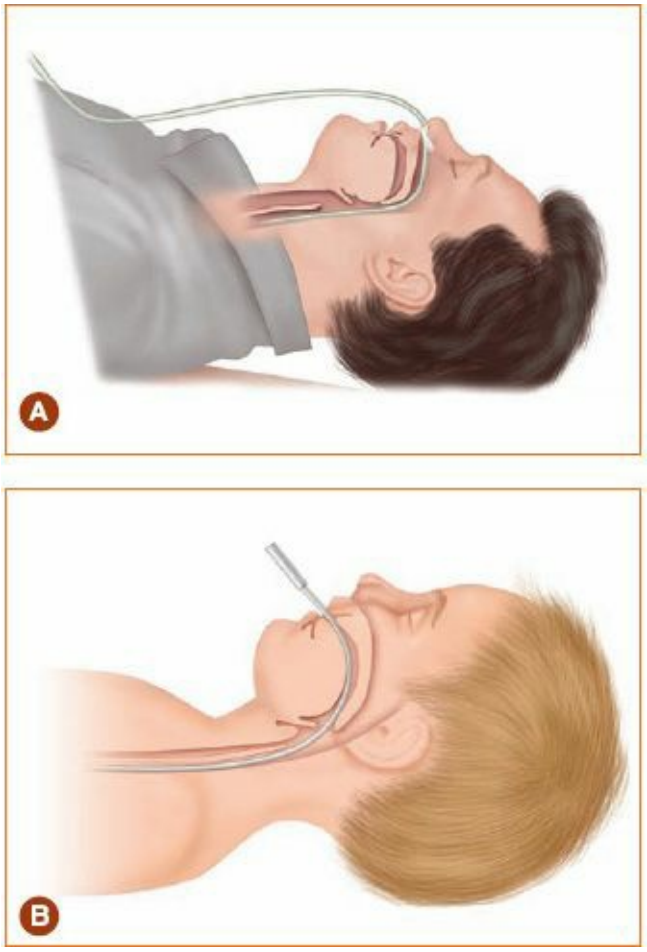


Figure 20-3 A nasogastric **(A)** or an orogastric **(B)** tube is required for stomach decompression and protection from aspiration in intubated patients.

Every patient should experience treatment that provides maximum comfort, privacy, and dignity, within the context of the clinical situation. Patients should be protected from unintentional temperature alterations. Environmental exposure (for example, to hot, cold, moisture, or noise) during transport, while loading or unloading patients, and in health care facilities can adversely impact patient care. Patients experience the same stressors of flight as do air-medical crew members but are frequently more susceptible and lack sufficient reserves to avoid detrimental consequences. Patients in ground transport units traveling through significant altitude changes will experience physiologic stressors similar to those in flight.

Nonintubated patients require **anxiolysis** and analgesia following many toxicologic exposures. Psychiatric and other at-risk patients may require sedative medications to facilitate care and promote comfort. Family members and friends should be assessed for dysfunctional coping during any crisis situation.

Decontamination

Patient decontamination following exposure to a toxic substance is directed at minimizing the quantity of that substance absorbed by the patient and the extent of any local damage. Decontamination may be as simple as providing the patient with fresh air for several minutes or as complicated as whole-bowel irrigation (WBI), taking many hours to perform. Prompt, effective decontamination has the potential to prevent the fatal consequences of an otherwise lethal overdose.

Skin Decontamination

Caustic agents and acetylcholinesterase inhibitors (such as pesticides and chemical weapons) can cause systemic toxic effects from dermal absorption. It is essential that health care providers use the correct PPE during skin decontamination. Numerous other chemicals cause severe local reactions or burns when placed in contact with skin. Skin decontamination, like all other methods of decontamination, is most effective if initiated immediately following exposure and may have no benefit if significantly delayed. Certain agents, such as sodium hydroxide and potassium hydroxide, react violently with water and require a specialized approach to skin and eye decontamination.

Any potentially contaminated clothing must be removed. Dry powders must be gently brushed away **Figure 20-4** and then the affected area should be flushed with copious amounts of water or saline. During irrigation, particular attention should be given to the patient's ears and groin, the area between the fingers and toes, and any skin folds. Hydrocarbon-based products often require a mild soap or shampoo for more effective removal.

No attempt should be made to neutralize chemicals on the skin; frequently, the reaction generates heat and causes additional injury. Only four chemicals are recommended for topical treatment beyond irrigation: hydrofluoric acid treated with calcium, oxalic acid treated with calcium, phenol treated with mineral oil or isopropyl alcohol, and white phosphorus treated with 1% copper sulfate. A reliable reference should be consulted before topical treatment is given.

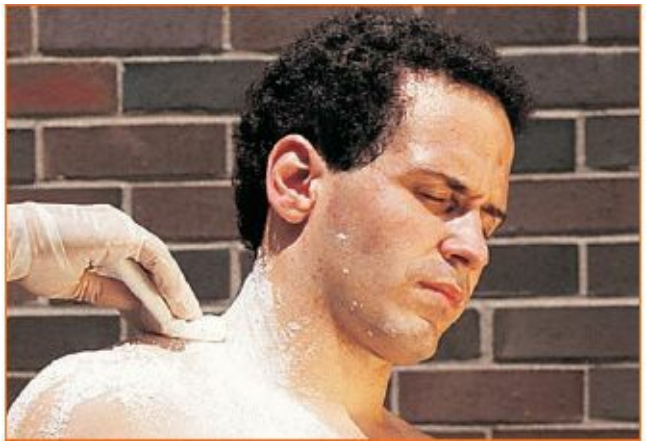


Figure 20-4 To decontaminate skin, gently brush away any dry powders before flushing the area with water or saline. Always ensure protection against hypothermia after decontamination because patients get cold quickly.

Figure 20-5 An irrigation adjunct, like the Morgan Lens, can help flush toxins away from the eye.

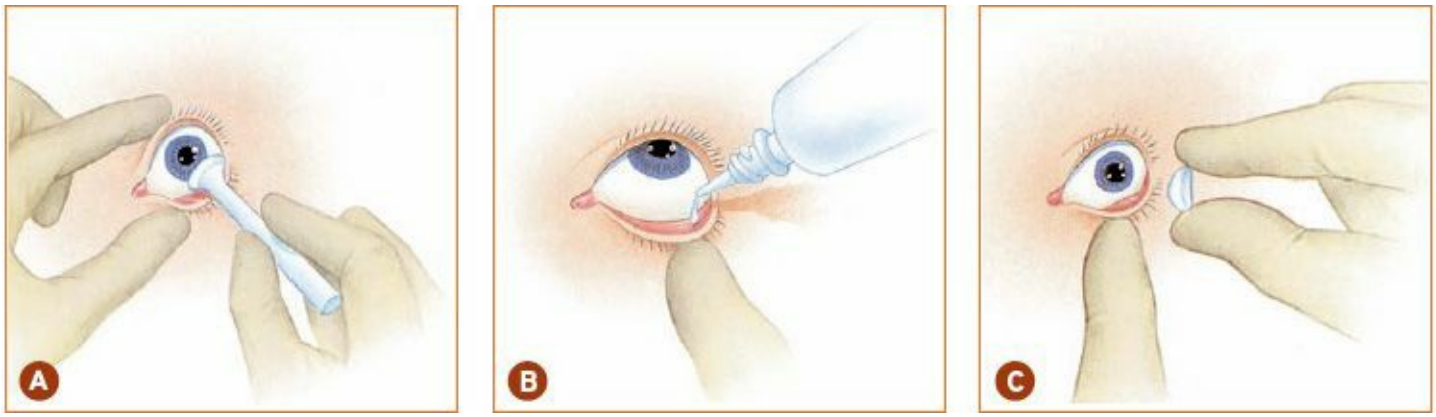


Figure 20-6 Remove contact lenses still in place before irrigation. **A.** To remove hard contact lenses, use a specialized suction cup moistened with sterile saline solution. **B.** To remove soft contact lenses, instill 1 or 2 drops of saline or irrigating solution. **C.** Pinch off the lens with your gloved thumb and index finger.

Ocular Decontamination

Many agents that cause toxicity from skin exposure will also cause damage when in contact with the eyes. The cornea is extremely sensitive to hydrocarbons and corrosive agents.

Each affected eye should be flushed with copious amounts of water, saline, or other approved solution. Local ocular anesthetic drops along with a commercially available irrigation adjunct (Morgan Lens) may be used **Figure 20-5**. Before irrigation, contact lenses still in place need to be removed **Figure 20-6**.

Each eye needs to be irrigated with at least 1 L of water, until the ocular pH is normal following exposure to an acid or base. (Ocular pH is tested by touching the pH paper to the moist surface of the conjunctival cul-de-sac of the affected eye. The pH paper is then compared with the shades on the package.) No attempt should be made to neutralize chemicals in the eye. The neutralization reaction often produces heat, capable of causing devastating injury. Once irrigation is complete, a Poison Center should be consulted for definitive treatment or a thorough ophthalmology exam should be arranged for any significant exposures.

Gastric Decontamination

Patients who have ingested toxic chemicals or dangerous amounts of otherwise therapeutic medications may benefit from gastrointestinal (GI) decontamination. Gastrointestinal decontamination is accomplished through gastric lavage and emptying, activated charcoal administration, and WBI. In each case, the goal is the prevention of systemic absorption of a toxic chemical or medication.

Orogastric Lavage

Pills, pill fragments, and liquid toxins can be removed from the stomach by gastric lavage. This procedure was once widely accepted, but it is now limited to a very small number of clinical situations. In addition, it is not performed in the transport setting. Patients who go to a health care facility within 1 hour of taking a life-threatening ingestion are candidates for gastric lavage. Indications include the following:

- Highly toxic ingestion, likely to remain unabsorbed in the stomach
- Substances poorly absorbed by activated charcoal
- No effective antidote or treatment therapy currently available

Despite the potential to prevent harmful stomach contents from being absorbed, gastric lavage has

several disadvantages. It is a time-consuming, invasive procedure that will delay administration of activated charcoal or oral antidote medications. The amount of actual pill (or fragment) that is removed is limited. This procedure requires cooperation from awake, alert patients. Resistance is common, and sedative medications place patients at increased risk of aspiration. In addition, gastric lavage may propel toxic substances further into the digestive tract and limit the effectiveness of other treatments. The role of gastric lavage following a caustic ingestion is controversial. Contraindications include the following:

- Minimally toxic ingestion
- Significant time since ingestion and absorption has likely occurred
- Highly effective antidote available
- Inability of patient to protect airway during the procedure (ET intubation may be required before gastric lavage)

In terms of complications, aspiration is the main risk during gastric lavage. An injury to the airway, stomach, and esophagus is also possible.

Highly toxic liquids can be evacuated from the stomach using a common orogastric or nasogastric tube much more quickly than by the conventional gastric lavage procedure. For pills or pill fragments, a large (36F to 40F in adults) orogastric tube is inserted. Patients are placed in the left lateral position. After placement confirmation, saline or water is alternately instilled and removed until the effluent is clear (no pills, fragments, or stomach contents visible), or, in adults, until 2 L has been instilled and removed. Patients, especially children, need to be monitored for electrolyte abnormality and hypothermia. At the completion of the procedure, the orogastric tube provides easy access for the administration of activated charcoal. A closed system **Figure 20-7** prevents cross contamination by containing the gastric contents. Some sets include a port for charcoal administration to maintain the closed system.

Emesis induced by syrup of ipecac is no longer a routine treatment of toxic ingestions. Health care providers may encounter patients who received ipecac administered by misinformed members of the public or in one of the rare situations in which ipecac may be beneficial. Ipecac-induced emesis theoretically removes a portion (approximately 28%) of ingested toxins before systemic absorption.

Figure 20-7 A closed system for gastric lavage. The tubes shown range from sizes 32F to 36F and the solution bag holds 3.5 L.

Ipecac is generally limited to home use when patients would have a long delay before access to activated charcoal treatment. Similarly, patients who ingest chemicals or medications not well absorbed by activated charcoal might be candidates for ipecac, although WBI is preferred. Pills with a larger diameter than an appropriately sized orogastric tube are another possible indication for ipecac-induced emesis for gastric emptying.

Ipecac has many contraindications and side effects, almost completely eliminating its usefulness in toxic ingestions. Any patient who is sedated, obtunded, or has a history of seizure activity is not a candidate for ipecac. Ingestion of any medication with the potential for CNS depression or seizures, any hydrocarbon, or any corrosive agent is an absolute contraindication to ipecac. Aspiration is a potentially lethal consequence of inappropriate administration of ipecac. Side effects of ipecac include sedation, vomiting-related trauma (including Mallory-Weiss tear), and delay in the administration of activated charcoal or oral antidote.

Activated Charcoal

Ingestions of numerous medications and chemicals are amenable to absorption with activated charcoal in the digestive tract. **Activated charcoal** is a carbon-based liquid with an incredible absorptive ability **Figure 20-8**. It is typically administered orally or via a nasogastric or orogastric tube to decrease the available quantity of a toxic substance. Activated charcoal also has the novel ability to remove certain toxic substances from circulating blood by "gut dialysis." Orally administered activated charcoal can reduce the available quantity of a purely IV overdose of certain select medications (such as theophylline, carbamazepine, salicylates, digoxin, and some TCAs) or decrease serum drug levels long after systemic absorption has occurred. This ability is also limited to certain select chemicals.

Figure 20-8 Activated charcoal.

Activated charcoal is administered as a single dose for routine ingestions and in repeated doses for select, severe exposures. Adults usually receive 1 g/kg, and children receive 0.5 to 1 g/kg. Activated charcoal is most effective immediately following oral ingestions (within 2 hours) and may have no benefit once systemic absorption of a toxin has taken place. Activated charcoal is indicated in the following situations:

- Ingestion of a harmful amount of a substance known to be adsorbed by activated charcoal within a time frame in which **adsorption** by charcoal is likely to take place (varies dramatically by

substance and situation)
- Unknown substances ingested or presumption of toxic ingestions in high-risk patients

Toxicology reference sources are vague regarding the outer time limits for single-dose activated charcoal administration. Several factors extend the usefulness of activated charcoal beyond the 2-hour window of optimal efficacy:

- Evidence of continued medication absorption demonstrated by increasing serum levels or unexplained continued deterioration in clinical status
- Ingestion of enteric-coated or sustained-release preparations
- Ingested drug packets with likelihood of rupture
- Exposure to substances removed through gut dialysis
- Other conditions that delay toxin absorption (such as food in the stomach and delayed GI motility)

Massive overdoses may require several doses of activated charcoal to adsorb a sufficient amount of toxin in the intestines (see the later section "Multiple-Dose Activated Charcoal").

Sedated patients with an unprotected airway are not candidates for activated charcoal. Unresponsive patients needing activated charcoal require definitive airway protection with an ET tube. Aspiration is often a lethal complication. Vomiting and constipation are frequent side effects of administration. Activated charcoal is also contraindicated following caustic ingestions, because it obscures the views in diagnostic procedures, and hydrocarbon ingestions because of the high aspiration potential. Numerous substances are not well adsorbed by activated charcoal. Lithium, iron, certain heavy metals, hydrocarbons, and alcohols will not be absorbed well. There may still be some benefit to using activated charcoal when these substances are taken simultaneously with toxins that can be absorbed. A careful risk-benefit analysis is essential. Activated charcoal is often combined with a saline or magnesium-containing cathartic agent to decrease the likelihood of constipation and decrease the absorption of toxins unaffected by activated charcoal. A Poison Center or a reliable toxicology reference should be consulted to determine the appropriateness of using activated charcoal in unusual poisoning situations.

Whole-Bowel Irrigation

One additional option exists for treating ingestions of toxic substances that are not amenable to treatment with activated charcoal. **Whole-bowel irrigation (WBI)** involves the patient drinking (or receiving via orogastric or nasogastric tube) large quantities of a nonabsorbable, electrolyte-balanced liquid that propels stomach and intestinal contents through the digestive system. WBI is indicated for significant ingestions of potentially toxic substances not well adsorbed by activated charcoal (for example, iron, lithium, potassium, and heavy metals). It can be given with activated charcoal for large quantities of sustained-release or enteric-coated medications (such as calcium channel blockers and theophylline) and is also indicated when the patient has swallowed capsules containing illicit drugs (as done by body stuffers and body packers) or foreign bodies (such as batteries). This procedure is performed in the intensive care unit.

Body packers (people who swallow carefully packaged capsules for smuggling purposes) and **body stuffers** (people who hastily swallow illicit substances to avoid impending arrest) pose a challenge to health care providers. Body stuffers are at increased risk because of the often unreliable materials and marginal technique used during unplanned packaging. Patients may appear completely asymptomatic, but rapid, unexpected, catastrophic clinical instability develops as the packaging fails and the chemicals are rapidly absorbed by the digestive tract.

Commercial products for WBI are widely available and are used for other purposes, such as preparation for intestinal surgery and diagnostic procedures. Adult patients receive 1.5 to 2 L/h for 4 to 6

hours or until the rectal effluent is clear. Clinicians may use radiography to assist in the diagnosis or monitoring of radiopaque materials.

Enhanced Elimination

Certain, select poisoning and overdose situations are appropriate for **enhanced elimination**. This process replaces or augments the body's normal method of eliminating, modifying, or breaking down toxic substances. If the patient is exposed to lethal quantities of a toxic substance, the ordinary route of elimination from the body is impaired by the overdose or other pathology, or the patient will likely not tolerate adverse effects of the poisoning because of a preexisting medical condition, enhanced elimination should be considered. Substances concentrated heavily in the blood or extracellular fluids and only minimally concentrated in body tissues are more likely to respond to enhanced elimination. Highly protein-bound medications are less likely to be successfully removed. Multiple-dose activated charcoal, urinary manipulation, and various modes of dialysis each enhance elimination of certain toxins. A toxicologist or nephrologist should be consulted when considering these interventions.

Multiple-Dose Activated Charcoal

The unique ability to extract certain toxins from the bloodstream permits activated charcoal to remove chemicals and medications already absorbed into the body. Multiple-dose activated charcoal can reduce the elimination half-life of various chemicals, theoretically improving clinical outcomes. Typically, a reduced dose of activated charcoal is administered every 1 to 6 hours for several doses, or for up to 12 hours total. A toxicologist should be consulted for specific dosing recommendations.

Urinary Manipulation

Alkalinization of urinary pH promotes the increased excretion of weak acids such as salicylates and phenobarbital. Sodium bicarbonate is added to IV fluids to maintain a urinary pH of 7 to 8.5, usually by adding 1 to 2 mEq/kg every 3 to 4 hours. During therapy, fluid volume status, serum pH, and electrolytes (especially potassium and sodium) must be carefully monitored. Hypokalemia is extremely common. Frequent point-of-care testing and/or potassium replenishment during transport, if available, should be considered.

Peritoneal Dialysis, Hemodialysis, Hemoperfusion, and Continuous Renal Replacement Therapy

These modes provide additional options for clinicians managing a severe poisoning or overdose. CCTPs will be called to transport patients who are receiving these treatments. Treatments generally cannot be continued during transport and will need to be discontinued or interrupted to safely transport the patient. Specialized training and equipment is required, and the description is beyond the scope of this text. Patients requiring these services may be transported to tertiary care centers from smaller community facilities and clinics.

Specific Toxicologic Emergencies

Pharmaceutical and Abuse Agents

Acetaminophen

Acetaminophen (Tylenol, Paracetamol, APAP) is the most commonly ingested drug resulting in overdose. It is used widely as an analgesic and antipyretic and often is found combined with narcotic analgesic and cold-symptom ingredients in prescription and over-the-counter preparations. Toxicity from acetaminophen is complicated by other agents when a combination of medications is ingested.

Acetaminophen toxicity is insidious. Toxic signs and symptoms may not manifest until well after it is

too late for any beneficial effects from the known antidote (acetylcysteine). Liver failure results from untreated acetaminophen toxicity and usually requires transplantation or causes death. Ethanol, isoniazid, phenytoin, barbiturates, and carbamazepine predispose patients to increased risk of **hepatotoxicity** following overdose.

The diagnosis of acetaminophen poisoning is based on patient history and laboratory analysis. Documentation of time, duration, and nature of exposure is essential, because signs and symptoms of acetaminophen toxicity evolve in four distinct phases. The acetaminophen (Rumack-Matthew) nomogram is useful only for single, acute ingestions and is not reliable for exposures that have occurred over several hours or days, when gastric motility is altered (such as by anticholinergics or opiates), or for extended-release acetaminophen preparations **Figure 20-9**. A serum acetaminophen level should be obtained at least 4 hours (but < 24 hours) following ingestion and the level and time plotted on the nomogram. The nomogram will indicate whether toxicity is anticipated and treatment is indicated. *Once toxicity is established, future acetaminophen levels will not assist clinical management*. The serum acetaminophen level will continue to decline regardless of toxicity. A metabolite actually causes hepatotoxicity, misleading providers into discontinuing antidote administration prematurely based on a decreasing acetaminophen level. A toxicologist should be consulted for situations not covered adequately by the nomogram or if treatment questions persist.

Figure 20-9 The acetaminophen nomogram. Reproduced from *Pediatrics*, Vol. 55, Pages 871-876. Copyright 1976 by the AAP.

Nausea and vomiting are quite common during acetaminophen toxicity. In addition, patients have anorexia, dehydration, diaphoresis, and elevated liver function test results. During subsequent days, abdominal pain (usually in the right upper quadrant), oliguria, hepatomegaly, coagulopathy, and eventual liver failure develop. A patient who presents with abdominal pain, nausea, and/or vomiting following a probable acetaminophen overdose should be assumed to have toxicity until proven otherwise.

Acetaminophen toxicity is treated with initial GI decontamination with activated charcoal. Acetylcysteine can be given orally or intravenously as the antidote for acetaminophen. Acetylcysteine can prevent serious hepatotoxicity from acetaminophen when initiated within 24 hours of ingestion and can

completely eliminate toxic effects when given within 8 hours.

Signs and Symptoms

Acetaminophen Toxicity

- Phase 1 (0 to 24 h):
  - Nausea and vomiting
  - Anorexia
  - Dehydration
  - Diaphoresis
  - Elevated liver function test results
- Phase 2 (24 to 72 h):
  - Abdominal pain (usually in the right upper quadrant)
  - Continued elevation of liver function test results
- Phase 3 (72 to 96 h):
  - Oliguria
  - Hepatomegaly
  - Coagulopathy
  - Liver failure
- Phase 4 (longer than 4 days):
  - Resolution of symptoms or multiple organ failure

Transport Management

Acetaminophen Toxicity

- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Administer acetylcysteine (Mucomyst or IV Acetadote).

Treatment with acetylcysteine should begin immediately for symptomatic patients presenting more than 8 hours after ingestion. Oral acetylcysteine is given with a 140-mg/kg loading dose, followed by 70 mg/kg every 4 hours for 17 additional doses. Patients often require aggressive treatment with antiemetics during oral acetylcysteine therapy. Refractory vomiting, from toxicity or related to oral acetylcysteine, often requires IV acetylcysteine. Patients with uncomplicated acetaminophen poisoning who require IV acetylcysteine generally receive 20 hours of treatment. A loading dose is administered over 15 to 60 minutes followed by an IV infusion. Fluid and electrolyte abnormalities are possible when pediatric patients receive intravenous acetylcysteine. In emergency situations, the oral preparation can be given intravenously through an inline micropore filter. A toxicologist or a Poison Center should be consulted for specific IV dosing recommendations.

Amphetamines

Amphetamines are available as therapeutic medications (such as methylphenidate [Ritalin, Concerta],

amphetamine-dextroamphetamine combination [Adderall], and dextroamphetamine [Dexedrine]); used as illicit stimulants for personal, academic, or professional enhancement; and abused in society for euphoric properties (for example, ecstasy and methamphetamines). These drugs produce many desirable and adverse effects through central and peripheral nervous system stimulation. An amphetamine dose-toxicity paradox exists. A tolerance quickly develops in frequent users, but toxicity may occur when there is only a slight increase in the dose beyond the "therapeutic" range.

Patients have classic sympathomimetic symptoms (also discussed earlier in the "Toxic Syndromes [Toxidromes] and Medication Reaction Syndromes" section). Amphetamine toxicity produces hypertension, tachycardia, hyperactivity (although amphetamines are often used in the treatment of hyperactivity disorders), restlessness, anxiety, diaphoresis, tremors, fasciculations, and rhabdomyolysis. Severe toxicity causes seizures, myocardial ischemia, and cerebral hemorrhage. Amphetamines may cause **methamphetamine washout syndrome** (similar to cocaine washout syndrome, sometimes referred to as a meth-crash) when used in larger quantities over a longer period of time. Amine stores within the body become depleted following prolonged amphetamine use, leading to excessive sleep, hunger, and depression. This syndrome is completely opposite of the typical effects of amphetamine use.

Urine drug screening may identify patients taking amphetamines. Serum drug levels are not clinically useful. The urine output and results of blood tests should be monitored for signs of dehydration and rhabdomyolysis.

Decontamination with activated charcoal or WBI should be considered when clinically appropriate. The treatment is supportive and based entirely on presenting signs and symptoms. Benzodiazepines are used initially for seizure control or sedation and can be supplemented with phenobarbital for seizures and haloperidol for sedation, if needed. Patients with significant hyperthermia should be cooled aggressively. Fluids should be replaced by the IV route as clinically indicated. Hypertension that does not respond to sedative medications is treated with vasodilators. Tachyarrhythmias can be treated with IV esmolol and propranolol.

Signs and Symptoms

Amphetamine Overdose

- Hypertension
- Tachycardia
- Hyperactivity, restlessness, and anxiety
- Diaphoresis
- Tremors
- Fasciculations
- Rhabdomyolysis
- Seizures
- Myocardial ischemia
- Cerebral hemorrhage

Transport Management

Amphetamine Overdose

- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Administer benzodiazepines for seizure control or sedation.
- Administer phenobarbital and haloperidol (Haldol), if needed.
- Provide aggressive cooling for significant hyperthermia.
- Replace IV fluids.
- Administer vasodilators for refractory hypertension.
- Administer IV esmolol and propranolol for tachyarrhythmias.

Benzodiazepines

Benzodiazepines (for example, diazepam [Valium], midazolam [Versed], and lorazepam [Ativan]) are a group of medications used therapeutically for anxiolysis, sedation, muscle relaxation, and seizure control. Toxicity occurs following therapeutic error, accidental or intentional ingestion, and illicit misuse and abuse in the community. Judicious administration by health care providers may prevent serious adverse effects when these medications are used therapeutically.

Deaths due to pure benzodiazepine overdoses are rare. Overdoses are often easy to manage conservatively, although sedation and respiratory depression may persist for several days with certain preparations. Benzodiazepine exposure becomes problematic when multiple agents with sedative properties are combined. When benzodiazepines are taken with ethanol, narcotics, and other sedative agents, profound CNS and respiratory depression occurs. Toxic effects also include hypothermia, ataxia, and slurred speech. Most urine drug screens test for benzodiazepines, although clonazepam (Klonopin) and alprazolam (Xanax) often are not detectable in the urine.

Activated charcoal adsorbs orally ingested benzodiazepines, but it is often contraindicated by an unintubated patient's decreased level of consciousness. Intubation should be considered whenever there is significant concern about a patient's ability to protect his or her airway, especially during transport. The majority of patients require only conservative management and careful observation.

Flumazenil (Romazicon) is a benzodiazepine antagonist, only appropriate for a small minority of overdose situations. Benzodiazepines, even in toxic doses, exert sedative and seizure-protective effects, which are desirable during clinical treatment. Reversal with flumazenil places patients at risk for increased intracranial pressure, sedative withdrawal, increased agitation, and potentially untreatable seizure activity. Flumazenil is contraindicated in poisoning or overdose by unknown substance(s); patients with a history of seizures; toxic exposures to chemicals known to cause seizures, increased intracranial pressure, agitation, or CNS stimulation; and patients taking benzodiazepine medications on a long-term basis. The vast array of situations contraindicating flumazenil severely limits its usefulness following benzodiazepine overdose. Flumazenil is useful when a therapeutic error during procedural sedation requires benzodiazepine reversal. The toxic effects of many benzodiazepines last much longer than reversal with flumazenil. Unrecognized resedation will occur in patients not monitored adequately. The adult dose is 0.2 mg IV, increased to 0.3 and 0.5 mg at 30-second intervals for a maximum dose of 3 mg. Children's doses are titrated beginning at 0.01 mg/kg up to a maximum of 1 mg total. Flumazenil still has safe use in situations in which CCTPs are confident of the substances being treated and the clinical status of the patient, such as overmedication during procedural sedation. These types of situations may not occur frequently enough in critical care transport to justify routine use of flumazenil by CCTPs.

Signs and Symptoms

Benzodiazepine Overdose

- CNS and respiratory depression
- Hypothermia
- Ataxia
- Slurred speech

Transport Management

Benzodiazepine Overdose

- Manage the ABCs.
- Perform GI decontamination with activated charcoal unless an unintubated patient has a decreased level of consciousness.
- Intubate the patient.

Beta-Adrenergic Blocking Agents

Beta-adrenergic blocking agents (beta-blockers) are used therapeutically for a wide variety of medical conditions. Toxicity occurs from an intentional or accidental overdose in the community or profound therapeutic error in a health care facility. Atenolol (Tenormin), propranolol (Inderal), and metoprolol (Lopressor) are common beta-blockers. These medications inhibit catecholamines centrally and peripherally. Beta-1 blockers decrease heart rate, myocardial contractility, myocardial conduction, and myocardial oxygen consumption. Beta-2 blockers relax blood vessel walls, constrict bronchi in the lungs, and block catecholamines in the GI and genitourinary tracts. Beta-1 and beta-2 blockers cause hypoglycemia through inhibition of **glycogenolysis** and **gluconeogenesis**. These medications are prescribed based on their beta-1– or beta-2–blocking actions. During overdoses, any beta selectivity is lost, resulting in beta-1– *and* beta-2–blocking effects.

Patient response is largely related to preexisting cardiovascular status. One normal dose for an adult may have catastrophic effects on a small child. An overdose is significantly worse if beta-blockers are combined with other cardiotoxic substances. In adults, two to three times the therapeutic dose has the potential to be fatal.

In addition to hypotension, bradycardia, and hypoglycemia, patients may exhibit bronchospasm, conduction disturbances, pulmonary edema, and, sometimes, seizures, mental status depression, and even tachycardia. Gastric lavage is indicated for recent (< 1 to 2 hours), significant ingestions (but is not performed in the transport setting). Activated charcoal is also administered immediately after lavage or as soon as possible after a toxic ingestion. Some, but not all, beta-blockers can be removed by dialysis for enhanced elimination.

Signs and Symptoms

Beta-Adrenergic Blocking Agent Overdose

- Hypotension
- Bradycardia
- Hypoglycemia

- Bronchospasm
- Conduction disturbances
- Pulmonary edema
- Possible seizures, mental status depression, and tachycardia

Transport Management

Beta-Adrenergic Blocking Agent Overdose

- Manage the ABCs.
- Administer activated charcoal.
- Provide enhanced elimination.
- Administer glucagon.
- Administer IV atropine or use cardiac pacing for bradycardia.
- Administer catecholamines, including dopamine and epinephrine, for refractory hypotension and bradycardia.
- Administer high-dose insulin (given with glucose).
- Administer milrinone when invasive monitoring technology is available.

Glucagon is the treatment of choice for a beta-blocker overdose. When given at increased doses, it exerts a positive inotropic effect. An initial dose of 5 to 10 mg IV is followed by an infusion at 1 to 5 mg/h in adults. This treatment requires a significant amount of glucagon, often more than is available to transport teams. Glucagon is normally reconstituted with phenol. With the quantity of glucagon required, phenol toxicity may occur. To avoid the risk of phenol toxicity, glucagon powder should be reconstituted with normal saline or 5% dextrose in water.

Bradycardia is initially treated with IV atropine (0.01 to 0.03 mg/kg) or cardiac pacing. Some sources recommend an isoproterenol infusion beginning at 4 µg/min and titrated to effect. Catecholamines, including dopamine and epinephrine, are given for refractory hypotension and bradycardia. In addition, high-dose insulin (given with glucose to prevent hypoglycemia) has been shown to improve survival in beta-blocker overdoses. **Phosphodiesterase** inhibitors (for example, milrinone) have an **inotropic** effect in beta-blocker overdoses but should be used only when invasive monitoring technology is available because they often result in hypotension. A toxicologist or a Poison Center should be consulted for guidance during complicated transports.

Calcium Antagonists (Calcium Channel Blockers)

Calcium channel blockers are used clinically to treat hypertension, migraines, cardiomyopathy, and other conditions. These medications include amlodipine (Norvasc), diltiazem (Cardizem), and verapamil (Calan). They act by decreasing intracellular calcium, resulting in vasodilation, decreased myocardial contractility, slowed conduction, and decreased heart rate. Management of calcium channel blocker overdoses is complicated by the widespread availability of sustained-release preparations.

Signs and Symptoms

Calcium Antagonist Overdose

- Hypotension
- Bradycardia
- Nausea and vomiting
- Altered mental status
- Metabolic acidosis
- Hyperglycemia

Transport Management

Calcium Antagonist Overdose

- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Administer IV calcium chloride or calcium gluconate.
- Administer glucagon, high-dose insulin, and epinephrine for hypotension and bradycardia.
- Perform cardiac pacing for bradycardia.
- Administer milrinone.

Hypotension and bradycardia are the most common signs of overdose with calcium channel blockers. Other effects include nausea, vomiting, altered mental status, metabolic acidosis, and hyperglycemia. These clinical features may evolve as additional medication is absorbed from sustained-release preparations. One tablet is often enough to cause serious toxicity or death in a small child. Any medication taken beyond the therapeutic dose should be considered potentially toxic.

Gastrointestinal decontamination is accomplished with activated charcoal, often given in repeated doses. Gastric lavage should be considered for recent, severe ingestions. The use of WBI is appropriate for enteric-coated or sustained-release preparations but is not done by CCTPs.

IV calcium is the antidote to an overdose with a calcium channel blocker. *If the overdose includes any cardiac glycoside medications (for example, digitalis), IV calcium should be avoided owing to the risk of lethal arrhythmias*. Adult patients are given calcium chloride 10% (10 mL) or calcium gluconate 10% (20 mL) every 5 to 10 minutes as needed. In severe cases, patients have received more than 15 g of calcium safely. Glucagon, high-dose insulin, and epinephrine are useful in the management of hypotension and bradycardias associated with an overdose with calcium channel blockers. In addition, cardiac pacing may be required for severe, symptomatic bradycardia. Select phosphodiesterase inhibitors (such as milrinone) improve contractility but often result in systemic vasodilation, limiting their usefulness. Atropine is not generally helpful for overdoses with calcium channel blockers.

Cardiac Glycosides

Cardiac glycosides are used therapeutically as digoxin and digitoxin for heart rate control and increased contractility in heart failure and supraventricular arrhythmias. Cardiac glycosides are also found naturally in plants such as foxglove, oleander, and lily of the valley. Peak effects from oral ingestion occur in 6 to 12 hours. Potential ingestions require 12 to 24 hours of observation before discharge of the patient from the health care facility.

Symptoms of cardiac glycoside toxicity are often vague, frequently resulting in misdiagnosis. Patients have nausea, vomiting, and abdominal pain, possibly accompanied by lethargy, confusion, weakness, and/or yellow-green visual disturbance. Almost any ECG change (with the exception of supraventricular

tachycardia) may be present. Bradycardia and conduction disturbances are common. Hyperkalemia is common following cardiac glycoside toxicity. Hypokalemia predisposes to toxicity in patients receiving digoxin or digitoxin. Serum drug levels are extremely valuable in the diagnosis and management of cardiac glycoside poisoning. The therapeutic level of digoxin is 0.5 to 2 ng/mL; and digitoxin, 10 to 30 ng/mL. These levels become falsely elevated during digoxin immune Fab (ovine) (Digibind) treatments, discussed subsequently.

Cardiac glycosides are adsorbed in the intestines by activated charcoal. Severe exposures will benefit from multidose charcoal (discussed in the earlier section "Multiple-Dose Activated Charcoal"). Gastric lavage is not usually indicated.

Symptomatic bradycardia is treated with atropine or cardiac pacing. Hyperkalemia must be corrected promptly, without the use of calcium. *IV calcium may cause lethal arrhythmias in patients with a cardiac glycoside overdose*. Increases in the level of intracellular calcium due to cardiac glycosides are responsible for the increased myocardial contractility (beneficial) and for the development of digitalis toxicity. Calcium can prolong the PR and QRS intervals, significantly worsening digitalis toxicity. Lidocaine and phenytoin assist in the management of ventricular arrhythmias.

Digoxin-specific antibodies (Digibind and Digifab) are the antidotes for cardiac glycoside overdoses and the preferred treatment for ventricular arrhythmias. Dosing is based on the serum drug level and the patient's weight. Severe hyperkalemia, arrhythmias, and renal disease are indications for antidote administration. A toxicologist or a Poison Center should be consulted for specific treatment and dosing recommendations.

Signs and Symptoms

Cardiac Glycoside Overdose

- Nausea and vomiting
- Abdominal pain
- Possible lethargy, confusion, and/or weakness
- Bradycardia and conduction disturbances
- Hyperkalemia

Transport Management

Cardiac Glycoside Overdose

- Manage the ABCs.
- Perform GI decontamination with activated charcoal (single dose or multidose).
- Administer atropine for bradycardia.
- Perform cardiac pacing for bradycardia.
- Administer lidocaine and phenytoin for ventricular arrhythmias.
- Administer digoxin-specific antibodies (Digibind or Digifab) for ventricular arrhythmias.

Cocaine

Cocaine shares many toxic properties with amphetamines and other sympathomimetics. This potent drug is used therapeutically for anesthesia and vasoconstriction of the mucous membranes, but toxic exposures

more often occur following illicit use and abuse. Cocaine is rapidly absorbed from all mucous membranes: the GI tract, lungs, rectum, and vagina. Depending on the route and method, the effects of cocaine peak in 1 to 30 minutes, and it has a half-life of up to 90 minutes. Cocaine handlers may hide cocaine internally for smuggling purposes (body packers) or rapidly conceal cocaine internally when arrest is believed to be imminent (body stuffers), posing a particular challenge to health care providers.

Diagnosis is made by obtaining a patient history and performing urine drug screens and a clinical examination. Chest pain is the most common symptom of cocaine intoxication. In addition to myocardial ischemia and infarction, chest pain occurs from cocaine-induced aortic dissection, pneumothorax, and pneumomediastinum. Young patients, without a cardiac history, who have an acute myocardial infarction or acute coronary syndrome should be assessed for the possibility of cocaine use.

Other symptoms of cocaine intoxication involve many body systems. Ventricular and supraventricular tachycardias are common. Hypoxia, acidosis, and sympathetic stimulation contribute to the development of tachycardias. Bradycardia also occurs from vagal stimulation in select situations. Most cocaine deaths are ultimately caused by arrhythmias. Additional vascular effects include hypertension and renal or intestinal vasospasm.

Cocaine has significant effects on the CNS. Patients may have cerebral hemorrhage or infarction, coma, anxiety, agitation, delirium, and psychosis. Seizures occur following exposure, but status epilepticus suggests continued absorption or concomitant toxicity. Heavy cocaine users also have **cocaine washout syndrome**, profound exhaustion with the ability to regain normal mental status and orientation when aroused. Other harmful effects from cocaine include hyperthermia, muscle rigidity, various movement disorders, rhabdomyolysis, acidosis, pulmonary edema, and exacerbation of asthma. Patients exhibit skin alterations such as tissue necrosis, ulcerations, and scratches, sometimes because of scratching imaginary insects. The aforementioned signs and symptoms are far from a complete list of the effects of cocaine intoxication.

Signs and Symptoms

Cocaine Toxicity

- Chest pain
- Tachycardias
- Cerebral hemorrhage or infarction
- Coma
- Anxiety, agitation, delirium, and psychosis
- Seizures
- Hyperthermia
- Muscle rigidity and movement disorders
- Rhabdomyolysis
- Acidosis
- Pulmonary edema
- Exacerbation of asthma
- Skin alterations (for example, tissue necrosis, ulcerations, and scratches)

Transport Management

Cocaine Toxicity

- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Administer benzodiazepines.
- Administer IV hydration.
- Monitor for rhabdomyolysis; alkalinize the urine with IV sodium bicarbonate if evidence of rhabdomyolysis is present.
- Provide active external cooling for hyperthermia.
- Administer vasodilators for hypertension.
- Administer thrombolytics for cocaine-induced myocardial infarction or transport patients to a health care facility capable of prompt percutaneous transluminal coronary angioplasty (PTCA).
- Administer benzodiazepines, phentolamine, and calcium channel blockers to mitigate sympathetic tone or vasospasm.
- Administer nitrates, oxygen, and aspirin for cocaine-induced myocardial infarction.
- Beta-blockers should not be administered to patients with recent cocaine ingestion.

Activated charcoal and WBI are useful for orally ingested cocaine-containing packages. The effects of orally ingested cocaine peak within 60 minutes of consumption. Activated charcoal may theoretically benefit if given immediately after ingestion, but any delay drastically reduces its efficacy. Cocaine-containing packages trapped in the digestive tract may require surgical removal if WBI is unsuccessful.

There is no antidote for cocaine. The treatment is supportive and based on clinical presentation. Benzodiazepines can be administered for agitation, anxiety, muscle rigidity, and seizure control and have a beneficial effect on hypertension. Treatment involves IV hydration adequate to maintain a urine output of at least 3 mL/kg/h, monitoring for rhabdomyolysis, alkalinizing the urine with IV sodium bicarbonate if evidence of rhabdomyolysis is present, and active external cooling for patients with hyperthermia. Various specialists (for example, in cardiology, neurosurgery, or vascular surgery) should be consulted for any cocaine-related end-organ injury.

Hypertension that does not respond to benzodiazepines should be treated with caution. Beta-blockers given for hypertension may cause an increase in blood pressure because of unopposed alpha-adrenergic stimulation. The use of a vasodilator medication should be considered instead of a beta-blocker, or other provision should be made for alpha-adrenergic control.

Cocaine-induced myocardial infarction should be treated with conventional strategies, including thrombolytics (or PTCA in the hospital in certain cases), with the exception that beta-blockers must be withheld to avoid unopposed alpha stimulation leading to severe hypertension. Benzodiazepines, phentolamine, and calcium channel blockers may additionally mitigate sympathetic tone or vasospasm in patients with cocaine intoxication. Care for cocaine-induced myocardial ischemia includes nitrates, oxygen, and aspirin.

Opioids and Opiates

Opioids are the class of chemicals that include naturally derived opiates (for example, heroin, morphine, and codeine) and newer artificial medications with the same properties (for example, fentanyl and meperidine). These chemicals are used heavily for analgesia in modern health care and also are used and abused throughout the world for their euphoric properties. Absorption occurs through oral, dermal, lung, IV, and mucosal routes, resulting in a vast range of times for onset, peak, and duration of effects. Toxicity is highly variable and is determined by the amount, route and rate of administration, and individual

tolerance.

Opioid substances may or may not be detected by routine urine drug screening tests, depending on the particular chemical involved. Prehospital and emergency department health care providers frequently encounter opioid-intoxicated patients. The diagnosis is routinely made by clinical presentation or when symptoms are reversed by an opioid antagonist such as naloxone (Narcan).

In opioid intoxication, patients have profound CNS and respiratory depression. The triad of CNS depression, respiratory depression, and pinpoint pupils offers a presumptive diagnosis. In addition, euphoria, noncardiogenic pulmonary edema, and dermatologic signs of needle marks or subcutaneous ulcerations are often present. Hypoxia and death occur following severe respiratory depression in untreated patients. Patients have more unusual symptoms when exposed to select opioids (for example, seizures with meperidine or propoxyphene; histamine release with morphine). Manifestations of hypoxic brain injury may further complicate the clinical diagnosis in opioid-intoxicated patients.

Signs and Symptoms

Opioid Toxicity

- Profound CNS and respiratory depression
- Pinpoint pupils
- Euphoria
- Noncardiogenic pulmonary edema
- Dermatologic signs of needle marks or subcutaneous ulcerations

Transport Management

Opioid Toxicity

- Manage the ABCs.
- Administer naloxone (Narcan) to reestablish a patent airway and spontaneous breathing.
- Perform GI decontamination with activated charcoal.

Airway and breathing are frequently compromised in severe opioid overdoses. Initial treatment involves establishing and maintaining a patent airway while supporting oxygenation and ventilation. Naloxone, when administered promptly, reverses the opioid effects and often reestablishes a patent airway and spontaneous breathing.

Naloxone is the most common antidote for acute opioid overdose. It can be administered intravenously, intramuscularly, subcutaneously, via ET tube, and intranasally. Dosing is titrated to reverse respiratory arrest or depression without precipitating withdrawal symptoms in long-term users. Because the reversal effects of naloxone do not last as long as the effects of many opioids, careful monitoring is needed following naloxone administration to avoid an unrecognized return of toxic effects. Naloxone can also be administered as an IV drip to maintain CNS and respiratory functioning in patients with long-acting opioid toxicity. Activated charcoal is indicated for recent oral ingestions of opioid substances, especially when they are combined with additional toxic chemicals. Following an oral opioid overdose, patients should be evaluated for the presence of acetaminophen and salicylates.

Salicylates

Poisoning from aspirin (acetylsalicylic acid) and other salicy-late-containing compounds continues to challenge health care providers. In addition to aspirin, salicylates are found in topical analgesics, liniments, and antidiarrheal agents. Toxic exposure occurs from accidental ingestion by children, therapeutic error, and as a suicidal gesture. Ingestion of less than 1 mL of a concentrated topical preparation can cause toxicity in small children.

The diagnosis of salicylate poisoning is made by obtaining a patient history and performing a clinical examination and laboratory testing. Serum salicylate levels can be obtained at most health care facilities; samples should be drawn at least 2 to 4 hours following suspected exposure (longer or with serial testing when delayed absorption is suspected). A urine salicy-late screening test allows providers to determine the presence or absence of salicylates immediately at the bedside.

Presumptive clinical diagnosis often occurs when patients report taking excessive doses of salicylate products and have tachypnea and tinnitus. Any new patient complaint of tinnitus, dizziness, or deafness should prompt an evaluation for possible salicylate poisoning. Salicylate toxicity can cause the spectrum of neurologic changes from mild confusion to coma. Nausea, vomiting, abdominal pain, GI bleeding, and hyperthermia may also be present. In severe cases, salicylates cause pulmonary edema, liver or renal failure, dehydration, electrolyte abnormalities, profound metabolic acidosis (although respiratory alkalosis is also frequently present), and coagulopathy.

Patients require careful attention to the ABCs. Initially, patients exhibit a CNS-driven tachypnea. As the poisoning evolves, the respiratory rate and depth increase to compensate for worsening metabolic acidosis. For patients who require intubation and mechanical ventilation, their rates, tidal volume, and minute volume should be matched carefully to avoid undermining their potentially lifesaving compensatory hyper-ventilation. Fluid volume and electrolyte status should be carefully monitored during treatment and transport. Acidosis and subsequent treatments will alter the elimination and distribution of critical electrolytes such as potassium. Potassium replenishment should be anticipated during patient treatment and transport, especially if urinary alkalinization is performed.

Signs and Symptoms

Salicylate Toxicity

- Tachypnea
- Tinnitus, dizziness, or deafness
- Hyperthermia
- Neurologic changes from mild confusion to coma
- Nausea, vomiting, abdominal pain, and GI bleeding
- Pulmonary edema
- Liver or renal failure

Transport Management

Salicylate Toxicity

- Manage the ABCs.
- Monitor fluid volume and electrolyte status.
- Provide potassium replenishment.

- Perform GI decontamination with ipecac (for children).
- Perform GI decontamination with multiple-dose activated charcoal.
- Perform urinary alkalinization for reduction of the salicylate level.

There is no antidote for salicylate poisoning. Gastrointestinal decontamination may include ipecac for small, accidental ingestions in young children or gastric lavage and WBI for massive ingestions in older children, adolescents, and adults. Depending on the quantity of aspirin or salicylate ingested, patients may require repeated doses of activated charcoal to adsorb the vast quantity of available toxin.

Salicylate toxicity is a candidate for several modes of enhanced elimination. Urinary alkalinization is extremely effective for reducing the salicylate level. This simple procedure can easily be initiated while other interventions are performed or during transport. Salicylate toxicity is also effectively treated in the hospital with hemodialysis and **hemoperfusion**, although hemoperfusion does not provide the same correction of electrolyte levels and acidosis as provided by hemodialysis. Both hemodialysis and hemoperfusion are performed in the intensive care unit; treatments in progress need to be discontinued prior to transport.

SSRIs (and Noncyclic Antidepressants)

This category includes medications such as fluoxetine (Prozac), paroxetine (Paxil), sertraline (Zoloft), and a host of others. These medications offer a less toxic alternative to TCAs for the treatment of depression and anxiety and for smoking cessation. In general, toxicity is limited, although severe exposures can cause seizures, serotonin syndrome (discussed earlier in the "Toxic Syndromes [Toxidromes] and Medication Reaction Syndromes" section), cardiovascular alterations, and death. No clear toxic dose is established. Neither urine nor serum drug screening is widely available to assist in diagnosis or management.

Signs and Symptoms

SSRI Toxicity

- Sedation, ataxia, dizziness, and coma
- Nausea, vomiting, and diarrhea
- Headache, restlessness, shivering, and diaphoresis
- Hypotension, QRS prolongation, and sinus tachycardia
- Muscle rigidity, tremors, and hyperreflexia

Transport Management

SSRI Toxicity

- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Provide volume resuscitation for hypotension.
- Administer IV benzodiazepines for seizures.

Sedation, ataxia, dizziness, and coma are CNS complications of toxicity due to select SSRIs. Nausea, vomiting, diarrhea, headache, restlessness, shivering, and diaphoresis may also occur. Hypotension, prolongation of the QRS interval, and sinus tachycardia are the usual cardiovascular manifestations of an SSRI overdose.

There is no antidote for SSRI poisoning or overdose. Ipecac is not recommended because of the potential for CNS sedation and seizure activity. Gastric lavage and WBI are not typically indicated unless SSRIs are taken with more toxic substances. Activated charcoal is effective for adsorption in the GI tract if given promptly following ingestion. The treatment for serotonin syndrome is discussed in greater depth in the "Medication Reaction Syndromes" section. In SSRI overdose situations with serotonin syndrome, the treatment is supportive based on symptoms. Hypotension is treated initially with volume resuscitation. Seizures are initially treated with IV benzodiazepines. The ABCs need to be carefully monitored in severe overdoses.

Toxic Alcohols (Ethylene Glycol and Methanol)

The toxic chemicals ethylene glycol and methanol are commonly available in the community and present a significant challenge to health care providers following accidental or intentional ingestion. Each can cause profound, lethal metabolic acidosis following ingestion of very small quantities. Ethylene glycol is the primary component in vehicle antifreeze. Methanol is the primary component in windshield washer fluid and other solvents. Accidental exposure to these chemicals occurs when they are mislabeled or placed in a beverage container. Children may be drawn to the sweet taste of ethylene glycol. People with alcoholism may consume either chemical while searching for an ethanol substitute.

Diagnosis and treatment can be guided by serum methanol or ethylene glycol levels, but these tests are usually only available in larger health care facilities. Even in larger tertiary centers, results for quantitative serum levels may take many hours or several days. The long delay undermines the clinical usefulness of these tests for diagnosis and management.

Clinicians have two additional options to assist with diagnosis. Ethylene glycol often has fluorescein added, which may or may not be visible when urine is viewed under a Wood's (UV) lamp. This method is not entirely reliable but may offer some assistance. Certain types of urinary drainage bag tubing will create the appearance of a false-positive result under a UV light. Hippurate and oxalate crystals in the urine are also highly suggestive of ethylene glycol poisoning.

Each of these chemicals (methanol and ethylene glycol) causes an elevated serum osmolar gap, a finding that is very useful for timely diagnosis in outlying facilities. By comparing the calculated serum osmolality with the measured serum osmolality, clinicians can determine the osmolar gap. When hospital laboratories measure serum osmolality, it is essential that the "freezing point method" be used. Other analysis methods that permit evaporation of alcohols in the specimen will give inaccurate results. This gap is adjusted for the presence of serum ethanol and unreliable when the patient has alcoholic ketoacidosis. This is not an exact test; at least 12 other chemicals or conditions will cause an elevated osmolar gap. When no other useful diagnostic testing is available, this test can assist with diagnosis and guide initial treatment.

Calculated osmolality:

$$2\,(\text{Serum Sodium}) + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8} = 290\ \text{mOsm/L (Normal)}$$

For a rough estimation of the serum level (mg/dL), the osmolar gap is multiplied by the conversion factor listed in **Table 20-2**. (BUN indicates blood urea nitrogen.)

Toxic signs and symptoms develop following the consumption of 1 to 1.5 mL/kg of ethylene glycol. Patients initially show ataxia, slurred speech, and lethargy, characteristic of ethanol intoxication 3 to 4

hours after ingestion, possibly accompanied by nausea and vomiting. During this time, laboratory analysis reveals an elevated serum osmolar gap. As the toxicity progresses (4 to 12 hours after ingestion), profound anion-gap metabolic acidosis with a drop in the serum bicarbonate level develops (see Chapter 16 for a further discussion of acid-base balance). Seizures, cerebral edema, and coma are common. Respiratory manifestations include tachypnea, hyperventilation, and pulmonary edema. Severe exposures lead to renal failure, cardiac arrhythmias, and conduction disturbances.

TABLE 20-2 Osmolar Gap Conversion Factors

Substance	Conversion Factor
Ethanol	4.6
Ethylene glycol	6.2
Methanol	3.2
Isopropyl alcohol	6.0

Adapted from: Olson KR, ed. *Poisoning and Drug Overdose*, 5th ed. New York, NY: McGraw-Hill; 2007:32.

Patients with methanol poisoning have signs and symptoms somewhat different from those of ethylene glycol exposure. Toxicity occurs when more than 100 mg/kg is ingested. Death can occur after ingestion of 20 to 150 g. During the first several hours, patients exhibit signs of classic ethanol intoxication, possibly accompanied by gastritis. An elevated serum osmolar gap is also present. Toxicity progresses into a severe anion-gap metabolic acidosis. Patients complain of a "snow-field," hazy visual disturbance, or blindness. Renal failure, seizures, coma, and death occur in severe untreated exposures.

Gastric emptying with gastric lavage (or gastric suctioning) is the only effective method of decontamination for ethylene glycol and methanol poisoning. Patients require aggressive treatment for the ABCs following severe exposures. Two antidotes are available for the treatment of methanol and ethylene glycol poisoning: ethanol and fomepizole (Antizol). Either one can be used.

Signs and Symptoms

Ethylene Glycol Toxicity

- Ataxia, slurred speech, and lethargy
- Nausea and vomiting
- Seizures, cerebral edema, and coma
- Hyperventilation
- Pulmonary edema
- Renal failure
- Cardiac arrhythmias and conduction disturbances

Signs and Symptoms

Methanol Toxicity

- Ataxia, slurred speech, and lethargy
- Possible gastritis
- "Snowfield," hazy visual disturbance, or blindness
- Renal failure
- Seizures
- Coma

Transport Management

Ethylene Glycol and Methanol Poisoning

- Manage the ABCs.
- Administer ethanol (oral or IV) or fomepizole (Antizol).

Ethanol, given orally or intravenously, promotes methanol and ethylene glycol excretion through an alternative metabolic pathway, drastically reducing the metabolic acidosis. Serum levels are titrated to approximately 100 mg/dL (which may be measured in serum or tested at the bedside using a breathalyzer device). Many hospital pharmacies carry IV ethanol preparations along with a suitable bottle of spirits from a liquor store. Patients who receive ethanol therapy require close monitoring. Ethanol may cause abnormal behavior, CNS depression, gastritis, pancreatitis, hyponatremia, and hypoglycemia.

Fomepizole is a newer commercial antidote for ethylene glycol and methanol poisoning, creating the same alternative metabolic excretion pathway as ethanol does. It is significantly more expensive than ethanol but does not require the same depth and frequency of monitoring as ethanol therapy. Fomepizole does not cause CNS sedation and does not require specific serum monitoring. In select cases, fomepizole may cause headache, nausea, vomiting, dizziness, fever, or rash.

Tricyclic Antidepressants

These highly toxic medications are frequently prescribed as last-resort antidepressants to patients likely to attempt self-harm. TCAs are also prescribed to patients for the treatment of chronic pain, neuropathy, and migraines. Toxicity usually occurs from accidental ingestions by young children and intentional overdoses in patients attempting self-harm. The toxic dose is approximately 10 to 20 mg/kg. Any TCA overdose should be considered potentially life-threatening.

Signs and Symptoms

Tricyclic Antidepressant Overdose

- Dry mouth and skin
- Dilated pupils
- Seizures (often intractable)
- Delirium, agitation, and hallucinations
- Rhabdomyolysis
- Muscle tremors
- Hyperthermia
- Sinus tachycardia

- Conduction disturbance
- Ventricular arrhythmias
- Hypotension

Transport Management

Tricyclic Antidepressant Overdose

- Manage the ABCs.
- Administer IV sodium bicarbonate for conduction disturbance, ventricular arrhythmia, or refractory hypotension.
- Administer IV barbiturates for seizures.
- Perform chemical paralysis (accompanied by electroencephalographic monitoring) for intractable seizures.

Any patient admitted to a health care facility for an intentional overdose should be screened for the presence of TCAs. Most urine drug screens test for TCAs, and the measurement of serum levels is often available to assist diagnosis and management. Some non-TCA medications may cause false-positive urine screen results (for example, cyclobenzaprine [Flexeril]). A 12-lead ECG should be performed on any patient with a suspected TCA overdose to observe for cardiotoxic evidence or effects.

Prolongation of the PR, QRS, or QT interval indicates serious TCA toxicity. Patients often exhibit sinus tachycardia with ventricular tachycardia or fibrillation, torsades de pointes, and asystole in severe exposures. Hypotension and cardiogenic shock are also possible.

Patients exhibit many anticholinergic signs and symptoms following TCA overdose (see the earlier "Toxic Syndromes [Toxidromes]" section discussion on anticholinergic syndrome). Effects include dry mouth and skin, dilated pupils, seizures (often intractable), delirium, agitation, hallucinations, rhabdomyolysis, muscle tremors, and hyperthermia.

Ipecac is completely contraindicated following TCA exposure. Gastric lavage is appropriate for recent severe ingestions. Activated charcoal is effective. Clinicians should strongly consider multidose activated charcoal for severe exposures.

Patients require aggressive supportive treatment following a TCA overdose. Seizures may require IV barbiturates if initial treatment with benzodiazepines is ineffective. Chemical paralysis should be considered for intractable seizures to avoid hyperthermia and muscle breakdown. Chemical paralysis will stop muscle activity associated with seizures but will not stop brain seizure activity. Electroencephalographic monitoring is essential.

Any conduction disturbance, ventricular arrhythmia, or refractory hypotension is treated with IV sodium bicarbonate. Initial boluses of 1 to 2 mEq/kg are followed by a maintenance infusion to maintain a serum pH of 7.50 to 7.55.

Chemical Agents

Acetylcholinesterase Inhibitor (Organophosphate and Carbamate) Toxicity

Patients may become exposed to acetylcholinesterase inhibitors during unintentional contact with certain pesticides or as victims of a chemical weapon release. **Acetylcholinesterase** breaks down **acetylcholine** at the neuromuscular junction. When acetylcholinesterase is inhibited by these chemicals, excess acetylcholine accumulates in muscarinic, nicotinic, and CNS receptors, causing continued activation of

these receptors. Acetylcholinesterase inhibitors are absorbed through inhalation, dermal contact, mucous membranes, and the digestive tract. Exposed patients pose a major threat to unprotected health care workers during treatment and transport.

Diagnosis is based on patient history, similar illness of multiple people at the same location, and the acetylcholinesterase inhibitor toxidrome. Laboratory measurement of red blood cell acetylcholinesterase has been developed. It is neither widely available nor very helpful in the acute diagnosis and management of exposed patients.

Patients may have two distinct patterns of toxicity, depending on which receptor(s) are involved. Nicotinic receptor toxicity produces tachycardia, hypertension, fasciculations, weakness, hyperglycemia, and dilated pupils. Muscarinic receptor toxicity produces the classic SLUDGE syndrome, plus bronchorrhea, sweating, constricted pupils, and bradycardia. Nicotinic and muscarinic toxicity both produce altered mental status, coma, and seizure activity in severe exposures.

Skin and eye decontamination should be initiated promptly by rescuers or health care providers using appropriate PPE. The potential for rescuer exposure is high. Any potentially contaminated clothing and jewelry should be removed and irrigation begun. Water or saline is used, combined with a mild soap or detergent if these chemicals are combined with hydrocarbons.

Massive recent oral ingestions of acetylcholinesterase inhibitors may be treated by gastric lavage. This procedure should be avoided in patients with an unsecured airway because of the high potential for seizures and aspiration. Activated charcoal is also appropriate when the airway is adequately protected.

Signs and Symptoms

Acetylcholinesterase Inhibitor Toxicity

- Nicotinic receptor toxicity
  - Tachycardia
  - Hypertension
  - Fasciculations
  - Weakness
  - Hyperglycemia
  - Dilated pupils
  - Severe exposures: altered mental status, coma, and seizure activity
- Muscarinic receptor toxicity
  - SLUDGE (Salivation, Lacrimation, Urination, Diarrhea, Gastroenteritis, Emesis)
  - DUMBELS (Diaphoresis/diarrhea, Urination, Miosis, Bradycardia/bronchospasm/bronchorrhea, Emesis, Lacrimation, and Salivation)
  - Bronchorrhea
  - Sweating
  - Constricted pupils
  - Bradycardia
  - Severe exposures: altered mental status, coma, and seizure activity

Transport Management

Acetylcholinesterase Inhibitor Toxicity

- Perform decontamination: irrigation with water or saline (combined with a mild soap or detergent if these chemicals are combined with hydrocarbons).
- Manage the ABCs.
- Perform GI decontamination with activated charcoal.
- Administer atropine.
- Administer pralidoxime (2-PAM, Protopam).
- Administer nondepolarizing muscle relaxants.
- Administer IV benzodiazepines or barbiturates for seizures.

Atropine and pralidoxime (2-PAM, Protopam) are the antidotes for acetylcholinesterase inhibitor toxicity. Atropine decreases airway secretions, reverses bradycardia, and lessens wheezing. The initial dose is 0.5 to 2 mg IV and is doubled every 5 minutes until the patient's condition improves. Severe exposures require vast quantities of atropine, potentially depleting hospital and transport team supplies. Tachycardia is not an indication to withhold additional atropine doses.

Pralidoxime can regenerate acetylcholinesterase activity following certain organophosphate exposures. It is not needed following carbamate exposures. A bolus of 1 to 2 g is administered over several minutes, followed by an IV infusion. The military and other high-risk areas may have supplies of these medications readily available to rapidly treat toxic exposures.

The use of succinylcholine for RSI should be avoided following exposure to acetylcholinesterase inhibitors. Succinylcholine activates the same neuromuscular junction and will cause an extended chemical paralysis. The use of nondepolarizing muscle relaxants is recommended. Otherwise, aggressive management of airway and breathing is used when clinically indicated. Seizure activity is controlled with IV benzodiazepines and barbiturates.

Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless, tasteless gas that causes serious toxicity through environmental exposures. This substance is created during the incomplete combustion of carbon-based materials. It has an affinity for hemoglobin, 200 to 270 times greater than that of oxygen. Human toxicity occurs in industrial settings, during fires, from indoor heating appliances and generators, from misdirected automobile exhaust, and following intentional exposures by people attempting self-harm.

CO binds to hemoglobin more tightly than does oxygen and prevents the cell from producing energy using oxygen. CO displaces oxygen, normally bound to hemoglobin, resulting in profound tissue and organ hypoxia. In addition, CO exerts direct adverse effects on internal cellular functioning. The cells are forced to conduct anaerobic metabolism to supply energy needs. Anaerobic metabolism results in the accumulation of lactic acid, which results in cell acidosis. The organs that are highly dependent on the supply of oxygen are the first to exhibit signs of CO poisoning. The brain and heart are the primary sites of CO toxicity owing to their large oxygen requirements. The heart may exhibit ischemia as chest pain, shortness of breath, arrhythmia, a drop in cardiac output, and, in severe cases, cardiac arrest. The brain may exhibit its dysfunction as headache, mild confusion, altered mental status, seizures, and coma.

CO poisoning should be suspected in any victim of a fire in a confined space and can be diagnosed by an elevated CO level in an arterial or a venous blood gas sample. Victims who have been removed from a CO-toxic environment for several hours may show low or normal CO levels, despite serious CO poisoning, owing to distribution of the CO in the tissues.

When the index of suspicion is high, clinical diagnosis is easily confirmed with a serum carboxyhemoglobin level. Levels do not directly correlate with severity of symptoms but will aid in

diagnosis when the history of exposure is unclear.

Controversies

Carbon monoxide is the leading cause of poisoning in every industrialized country, yet up to 50% of cases continue to be misdiagnosed. Long-term consequences of CO poisoning include a significantly increased incidence of cardiovascular events and deaths as well as neurologic changes ranging from decreased intelligence to seizures or Parkinson-like syndromes. Up to 10% of patients admitted to hospitals with suspected acute coronary syndromes, seizures, unstable angina pain, and headache are actually victims of CO poisoning. Because symptoms have little correlation to carboxyhemoglobin (blood CO levels), providers must maintain a high index of suspicion and use noninvasive screening technologies whenever available.

The oxygen saturation value is accurate only if it is directly measured on a blood-gas sample. Values calculated from arterial Pa_{O_2} or obtained from conventional bedside pulse oximetry devices are falsely elevated because of CO binding to hemoglobin. If any doubt exists regarding possible CO exposure, aggressive treatment should be started before laboratory results are obtained. CO-oximetry is an emerging technology that uses various wavelengths of light to measure carboxyhemoglobin and other values through a noninvasive skin sensor, similar to a pulse oximeter. This device provides rapid detection of CO and methemoglobinemia at the bedside and during transport.

Mild toxicity results in headache, nausea, vomiting, abdominal pain, and dizziness. Patients without a clear history of CO exposure are at risk of misdiagnosis as having a GI virus or colic. Moderate CO toxicity produces confusion, dyspnea, ataxia, tachycardia, and chest pain. Patients with severe CO toxicity have seizure activity, coma, cardiac arrhythmias, hypotension, and myocardial ischemia. Cherry red skin is an ominous indication of severe toxicity, but it may or may not be present. Hyperthermia, syncope, and incontinence are also possible.

Patients should be removed from any toxic environment. Fresh air needs to be provided until supplemental oxygen is available. CO has a half-life of 3 to 4 hours at room air. If 100% oxygen is administered, the CO half-life decreases to 30 to 90 minutes. In severe exposures, hyperbaric oxygen reduces the CO half-life to 15 to 23 minutes. Any symptomatic patient should receive 100% oxygen immediately if CO poisoning is suspected. Hyperbaric oxygen has the ability to improve patient status up to 3 weeks following exposure to CO.

Special Populations

Fetal hemoglobin interacts differently with CO than maternal hemoglobin, placing an unborn child at greater risk of CO poisoning. Although maternal blood levels of CO will begin to decline immediately after exposure to CO ceases, fetal blood levels of CO continue to rise for several hours. This interaction often necessitates referral of pregnant patients with CO poisoning to hyperbaric oxygen therapy (HBO) to avoid continued fetal CO poisoning.

Signs and Symptoms

Carbon Monoxide Toxicity

- Mild: headache, nausea, vomiting, abdominal pain, and dizziness
- Moderate: confusion, dyspnea, ataxia, tachycardia, and chest pain
- Severe: seizure activity, coma, cardiac arrhythmias, hypotension, myocardial ischemia, cherry red skin, hyperthermia, syncope, and incontinence

Transport Management

Carbon Monoxide Toxicity

- Perform CO oximetry.
- Remove the patient from the toxic environment.
- Provide fresh air.
- Give 100% oxygen immediately.
- Manage the ABCs.
- Perform ET intubation; fill the ET tube cuffs with water if hyperbaric treatment is anticipated.
- Administer IV benzodiazepines or barbiturates for seizures.
- Assess for other possible causes of altered mental status or cardiovascular dysfunction.

The goal of therapy is to displace the CO, under pressure, from the hemoglobin and the mitochondria. Patients with altered mental status, seizures, or serious burns in the setting of CO poisoning should have their airway actively managed by ET intubation. In preparation for hyperbaric therapy, patients who require ET intubation and have CO poisoning should have the ET tube cuffs filled with water.

The remaining treatment for CO exposure is supportive. Airway and ventilator support is needed for comatose patients. Seizures should be controlled with IV benzodiazepines or barbiturates. Other possible causes of altered mental status or cardiovascular dysfunction should be considered.

Caustics and Corrosives

Patients may be exposed to caustic agents through occupational and industrial exposure, household contact, and a vast array of other possible scenarios. Caustics include acids, alkalis, and a host of other chemicals, each causing mucosal, skin, or internal organ damage following exposure.

The duration of exposure, concentration, pH, and particular substance influence the degree of toxicity. Exposure can occur through inhalation, ingestion, or dermal or eye exposure. Systemic toxicity is possible following exposure through any of these routes. Acidic substances cause **coagulation necrosis**, which limits the depth and extent of injury. Alkali substances form **liquefactive necrosis**, allowing extensive tissue penetration. Hydrocarbons and hydrofluoric acid are discussed separately (see the sections “Hydrocarbons” and “Hydrofluoric Acid/Hydrogen Fluoride [HF]”).

Diagnosis is often aided by the patient’s reported history or description of the setting where exposure occurred or symptoms began. The Material Safety Data Sheets (MSDS) and other documents may indicate the particular chemical involved along with suggestions for treatment, decontamination, and PPE needed for rescuers **Figure 20-10**.

Eye and skin exposures require copious irrigation with saline or water. Any basic or acidic substances require a normal mucosal pH before irrigation is discontinued. A reliable toxicology reference should be consulted before beginning any topical treatments other than irrigation.

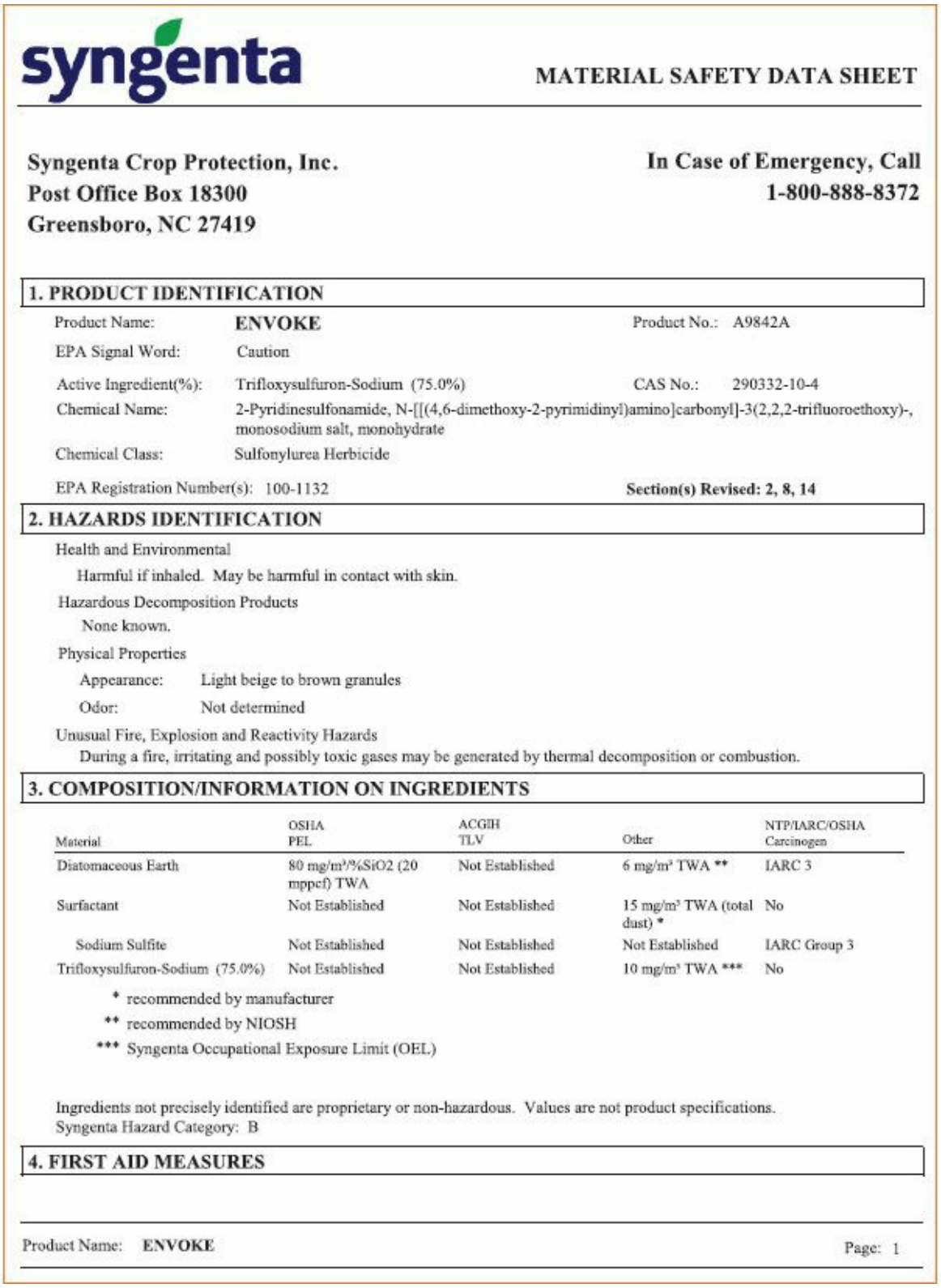
Patients with an inhalation exposure require immediate fresh air. Symptomatic patients may require supplemental oxygen, bronchodilator medications, humidified oxygen or air, or assisted ventilation in

severe exposures.

The ingestion of caustic agents has the potential to cause devastating injury. Close monitoring for airway compromise, GI bleeding and perforation, and free air in the thorax or abdomen is needed. Patients should immediately be given water or milk to drink, but inducing vomiting and attempting neutralization should not be attempted. A toxicologist or gastroenterologist should be consulted for specific treatment recommendations.

Chlorine, Ammonia, and Asphyxiate Gases

Various gases have the potential to displace oxygen in a patient's lungs; to damage or irritate the mouth, nose, and airway; or to otherwise disrupt effective respiration. These substances exist in gas form or become gases through chemical reactions or when a liquid or solid container is somehow compromised. Exposures occur during a hazardous materials release or when adequate safety precautions are not implemented during the use or storage of these chemicals. Toxicity is highly variable, depending on concentration, duration of exposure, preexisting health of exposed people, and a myriad of environmental factors.



syngenta

MATERIAL SAFETY DATA SHEET

Syngenta Crop Protection, Inc.
Post Office Box 18300
Greensboro, NC 27419

In Case of Emergency, Call
1-800-888-8372

1. PRODUCT IDENTIFICATION

Product Name: **ENVOKE** Product No.: A9842A

EPA Signal Word: Caution

Active Ingredient(%): Trifloxysulfuron-Sodium (75.0%) CAS No.: 290332-10-4

Chemical Name: 2-Pyridinesulfonamide, N-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3(2,2,2-trifluoroethoxy)-, monosodium salt, monohydrate

Chemical Class: Sulfonylurea Herbicide

EPA Registration Number(s): 100-1132 **Section(s) Revised: 2, 8, 14**

2. HAZARDS IDENTIFICATION

Health and Environmental

Harmful if inhaled. May be harmful in contact with skin.

Hazardous Decomposition Products

None known.

Physical Properties

Appearance: Light beige to brown granules

Odor: Not determined

Unusual Fire, Explosion and Reactivity Hazards

During a fire, irritating and possibly toxic gases may be generated by thermal decomposition or combustion.

3. COMPOSITION/INFORMATION ON INGREDIENTS

Material	OSHA PEL	ACGIH TLV	Other	NTP/IARC/OSHA Carcinogen
Diatomaceous Earth	80 mg/m³/%SiO2 (20 mppcf) TWA	Not Established	6 mg/m³ TWA **	IARC 3
Surfactant	Not Established	Not Established	15 mg/m³ TWA (total dust) *	No
Sodium Sulfite	Not Established	Not Established	Not Established	IARC Group 3
Trifloxysulfuron-Sodium (75.0%)	Not Established	Not Established	10 mg/m³ TWA ***	No

* recommended by manufacturer

** recommended by NIOSH

*** Syngenta Occupational Exposure Limit (OEL)

Ingredients not precisely identified are proprietary or non-hazardous. Values are not product specifications.
Syngenta Hazard Category: B

4. FIRST AID MEASURES

Product Name: **ENVOKE** Page: 1

Figure 20-10 The Material Safety Data Sheet (MSDS). The MSDS contains a great deal of critical safety information, including product identification, accidental release measures, exposure control/personal protection, and toxicologic information on multiple pages. Be certain to read the entire MSDS.

Transport Management

Caustic and Corrosive Exposure

- Perform decontamination: irrigate the eyes or skin with water or saline (combined with a mild soap

or detergent if these chemicals are combined with hydrocarbons).
- Check the mucosal pH; ensure the pH is normal before discontinuing irrigation.
- Manage the ABCs.
- For inhalation exposure
  - Provide fresh air.
  - Administer supplemental oxygen.
  - Administer bronchodilator medications.
  - Administer humidified oxygen or air.
  - Provide assisted ventilation in severe exposures.
- For ingestion
  - Monitor closely for airway compromise, GI bleeding and perforation, and free air in the thorax or abdomen.
  - Provide immediate water or milk to drink.
  - Do not induce vomiting or attempt neutralization.

Symptoms include lacrimation; coughing; drooling; airway discomfort, swelling, and irritation; and labored, rapid, or absent breathing. Lung auscultation may reveal wheezing, pulmonary edema, and absent or decreased breath sounds. In exposures to simple asphyxiate gases, patients may have only increased breathing, with or without hypoxia.

Immediate fresh air should be provided for patients with an inhalation exposure. Symptomatic patients may require supplemental oxygen, bronchodilator medications, humidified oxygen or air, or assisted ventilation in severe exposures. Pulmonary edema may require specific, aggressive management when present following a toxic inhalation. A toxicologist or a Poison Center should be consulted for customized treatment guidance.

Signs and Symptoms

Chlorine, Ammonia, and Asphyxiate Gases Toxicity

- Lacrimation, coughing, and drooling
- Airway discomfort, swelling, and irritation
- Labored, rapid, or absent breathing
- Wheezing
- Pulmonary edema

Transport Management

Chlorine, Ammonia, and Asphyxiate Gases Toxicity

- Manage the ABCs.
- Provide fresh air.
- Administer supplemental oxygen.
- Administer bronchodilator medications.
- Administer humidified oxygen or air.

- Provide assisted ventilation in severe exposures.
- Administer nebulized normal saline.
- Administer a half-strength preparation of sodium bicarbonate by nebulizer.
- Administer IV corticosteroids in refractory cases.

Patients should receive thorough decontamination before transport. Toxic fumes and residual chemicals pose a risk to unprotected transport team members. Toxic inhalations of chlorine and chloramine (the gas formed from mixing household bleach and ammonia) cause upper airway irritation, coughing, and burning, resulting from the formation of hydrochloric acid on the airway linings. Nebulized normal saline (5 mL) can be effective in alleviating symptoms when humidified air or oxygen is not available in the transport vehicle. In addition, severe symptoms often respond to neutralization of the hydrochloric acid using a half-strength preparation of sodium bicarbonate administered by nebulizer (2.5 mL of 3.75% or 4.2% sodium bicarbonate mixed with 2.5 mL of saline). Administration of IV corticosteroids may also be helpful in refractory cases.

Cyanide Poisoning

Cyanide is used heavily in the manufacturing industry, in laboratory settings, as a chemical weapon, and in homicides and suicides. Cyanide is present in certain fruit pits and seeds, and poisoning may occur from a metabolite during therapeutic administration of sodium nitroprusside (Nipride). The combustion of any carbon- and nitrogen-containing product has the ability to produce cyanide. Cyanide is produced when wool, plastic, and other combustible materials are burned; the combustion of plastics has the ability to produce lethal doses of cyanide. Toxicity may occur through any route: dermal, parenteral, inhalation, or ingestion. Any exposure to cyanide should be considered potentially lethal. Toxicity may be immediate or delayed, depending on the route and manner of exposure.

Cyanide poisons the cellular mitochondria by binding to the last step in energy production in which oxygen is used to produce adenosine triphosphate, or ATP, the energy currency of the cell. Cyanide inhibits the last step in energy production and effectively makes the cell unable to use the oxygen that is present. The clinical result is signs of hypoxia (dizziness, headache, air hunger, and acidosis), but without decreases in pulse oximetry or Po_2. Patients with low-level exposure may have nonspecific symptoms such as weakness and dizziness, whereas large-concentration exposures can result in seizures, coma, cardiovascular collapse, and death in 2 minutes or less.

Cyanide poisoning should be considered in any victim of a fire in a confined space, especially if the patient was in a room in which there was combustion of synthetic- and plastic-containing materials. Any patients potentially contaminated with cyanide require extensive decontamination by rescuers wearing chemical-protective suits with an environmentally isolating breathing apparatus (for example, self-contained breathing apparatus). Health care providers have been injured when patient emesis has released cyanide gas into the treatment room. The patient's skin and soaked clothing may additionally expose health care workers to cyanide during patient treatment if adequate precautions are not taken.

Patient or bystander reports and the location of exposure or manner of onset of symptoms may provide clues to assist in the diagnosis of cyanide poisoning. The classic "bitter almond" smell may or may not be present from hydrogen cyanide. Rescuers should avoid intentionally seeking smells and odors to identify toxic chemicals. Up to 40% of the population is unable to detect the odor of hydrogen cyanide.

Cyanide poisoning is confirmed with serum cyanide and thiocyanate levels. A whole-blood cyanide level greater than 2.5 μg/mL is associated with severe toxicity. A level greater than 3 μg/mL is usually lethal. Thiocyanate is the metabolized form of cyanide, excreted normally in the urine. Thiocyanate toxicity begins at 60 mg/L and becomes potentially lethal at levels above 200 mg/L. In most cases,

thiocyanate poisoning is less severe than cyanide poisoning. In addition, patients have an elevated serum lactate level (> 6 mmol/L or > 10 mmol/L after smoke inhalation); metabolic acidosis; and an elevated, mixed venous oxygen saturation due to decreased tissue oxygen extraction. Laboratory screening should also include a serum carboxyhemoglobin level if there is any potential for a combined exposure (such as during a fire).

Neurologic symptoms include seizures, coma, headache, confusion, anxiety, and weakness. Cardiovascular manifestations include complete cardiovascular collapse, hypertension, hypotension, ST-segment and T-wave ECG changes, asystole, and numerous other conduction blocks and arrhythmias. Patients have hyperventilation, respiratory depression, pulmonary edema, or complete respiratory failure.

Signs and Symptoms

Cyanide Poisoning

- Dizziness
- Weakness
- Headache
- Air hunger
- Acidosis
- No decrease in pulse oximetry or Po_2
- Hyperventilation
- Respiratory depression
- Pulmonary edema
- Complete respiratory failure
- Confusion
- Anxiety
- Seizures
- Complete cardiovascular collapse
- Hypertension or hypotension
- ST-segment and T-wave ECG changes
- Asystole or other conduction blocks and arrhythmias

Transport Management

Cyanide Poisoning

- Perform skin decontamination: copious irrigation with soap and/or shampoo and water.
- Manage the ABCs.
- Gastrointestinal decontamination with activated charcoal.
- Administer amyl nitrite, followed by IV sodium nitrite, then sodium thiosulfate, or administer hydroxycobalamin instead of the amyl nitrite/sodium nitrite/sodium thiosulfate combination.
- Monitor levels of serum electrolytes, blood gases, glucose, and lactate.

Skin decontamination requires copious irrigation by adequately protected rescuers or health care

providers using soap and/or shampoo and water. Activated charcoal adsorbs orally ingested cyanide preparations. Gastric lavage is also appropriate in select situations and may be used to remove old activated charcoal and replace it with fresh activated charcoal following the lavage procedure. Health care providers must use adequate PPE during any gastric decontamination procedures.

Several cyanide antidote kits are commercially available **Figure 20-11**. Industrial, laboratory, and manufacturing settings may have these on site, with or without trained personnel to assist. The treatment of cyanide poisoning is a two-step procedure: (1) removal of the cyanide from the mitochondrial apparatus, and (2) removal of the cyanide from the mitochondria to be excreted by the kidneys. The removal of cyanide from the mitochondria is accomplished by inducing methemoglobinemia. Methemoglobin is the result of oxidation of Fe^{2+} on hemoglobin to the Fe^{3+} state. Cyanide has a higher affinity for methemoglobin than it does for the mitochondrial apparatus and, therefore, leaves the mitochondria and binds to the methemoglobin. Methemoglobin is formed by the administration of nitrites. Amyl nitrite is a time-enhancing inhalation antidote for use until IV access is established. Amyl nitrite is supplied in two perles (small, round, glass ampules) that may be crushed and held in gauze over the mouth and nose of the victim (or bag-valve intake) for 30 seconds of each minute if no IV access is available. Or, it may be placed into a ventilator circuit until IV sodium nitrite is available. IV sodium nitrite is then administered.

Figure 20-11 Cyanide antidote kits are available commercially and may already be present at industrial, laboratory, and manufacturing settings.

Sodium nitrite is administered, 300 mg IV (supplied in 10-mL vials), for 2 to 4 minutes in adults. Children receive 6 mg/kg (or 0.12 to 0.33 mL/kg, with a maximum dose of 10 mL, for 5 to 10 minutes). The dosing may be adjusted based on a patient's hemoglobin level. Health care providers should monitor for nitrite-induced methemoglobinemia (for example, hypoxia, headache, dizziness, and classic "chocolate cyanosis") during therapy.

The second step in the treatment of cyanide poisoning is to remove the cyanide from the body by metabolizing the cyanide to thiocyanate so that it can be excreted by the kidneys. An enzyme called rhodanese normally completes the conversion of cyanide to thiocyanate in the body and requires sulfurs to complete this step. Sulfur is supplied by the administration of one 12.5-g ampule of sodium thiosulfate for 10 to 20 minutes. The pediatric dose is 412.5 mg/kg (or 1.65 mL/kg) for 10 to 20 minutes. The aforementioned medications come packaged together as part of the Lilly Cyanide Kit. More recently, many EMS systems have been replacing this kit with the Cyanokit, which contains 5 g of hydroxocobalamin, that, when administered, binds cyanide to become a form of vitamin B_{12}. Sodium thiosulfate may be given with hydroxocobalamin, simultaneously through a separate IV line or immediately following the hydroxocobalamin infusion for synergistic effect. The two medications should

not be given simultaneously through the same IV line. The hydroxocobalamin may cause hypertension (possibly beneficial) and a reddish discoloration to the skin and mucous membranes. This medication also interferes with CO-oximetry and certain blood chemistry testing. It is important to remember that good supportive care in the absence of antidote, although not optimal, may save many patients.

In addition to supportive, clinically based treatments, monitoring of serum electrolytes, blood gases, glucose, and lactate levels is necessary during treatment.

Hydrocarbons

Hydrocarbons are a class of chemicals widely used in all parts of modern society. This class is divided into aliphatic and aromatic hydrocarbons. The chemicals are used in solvents, fuels, paints, and oils and for many other purposes. Abuse of hydrocarbons is done by "huffing" various highly **volatile** (easily changed from liquid to gas form) hydrocarbons. The toxicity of these substances is highly variable, depending on the nature of the individual chemical and its concentration, as well as the timing, duration, and route of exposure.

Clues to the diagnosis of hydrocarbon exposure often come from information provided by a patient and/or bystander or from containers or markings present at the scene. Many hydrocarbon compounds have a distinctive odor that may remain on a patient or the clothing, assisting in diagnosis. These same odors will adversely affect CCTPs when patients are placed in confined, poorly ventilated transport vehicles. Thorough decontamination is essential before transport.

The ingestion of most hydrocarbons will cause nausea and vomiting with possible gastritis or gastroenteritis. Systemic toxicity can occur following ingestion of selected hydrocarbons.

Inhalation or huffing of hydrocarbons will cause hypoxia, direct brain injury, hypercarbia during rebreathing, and cardiotoxicity and will sensitize myocardial tissues to catecholamines. **Sudden sniffing death syndrome** occurs when people (usually adolescents) are startled while secretly huffing hydrocarbons. The surge of catecholamines on a sensitized myocardium causes ventricular fibrillation or ventricular tachycardia. Vasopressin should be used instead of epinephrine during resuscitation.

Transport Management

Hydrocarbon Toxicity

- Manage the ABCs.
- Administer methylene blue.
- Administer chelating agents.
- Administer acetylcysteine.
- Administer lidocaine or other antiarrhythmic agents for ventricular arrhythmias.

Dermal or ocular exposure to hydrocarbons will cause local irritation, burns, or corneal injury. Patients may experience profound chemical burns when hydrocarbon-saturated clothing or footwear is not promptly removed and skin decontaminated. A mild soap or shampoo should be used when irrigating hydrocarbon-contaminated skin. Systemic toxicity is possible following dermal absorption of certain hydrocarbons.

Toxicity from hydrocarbons is extremely limited in highly viscous (thick) preparations such as motor oil and petroleum jelly. These chemicals do not pose much of an aspiration risk and are not generally believed to cause systemic toxicity. Patients receive observation and general supportive treatments.

Hydrocarbons such as gasoline, kerosene, and petroleum ether are of low viscosity (flow easily) and

pose primarily a risk of chemical pneumonitis following aspiration. Care is supportive, and gastric emptying is contraindicated owing to the potential for aspiration. There is not much risk of systemic toxicity otherwise.

The remaining groups of hydrocarbons have a high potential for aspiration chemical pneumonitis, in addition to the possibility of systemic toxicity. Systemic effects include all of the aforementioned CNS and cardiac manifestations. Patients may also have hypotension, ataxia, headache, altered mental status, seizures, and any variant of respiratory distress.

There is no general antidote for hydrocarbon poisoning, but methylene blue is used when a particular hydrocarbon causes methemoglobinemia. **Chelating agents** and acetylcysteine are also used for treating specific hydrocarbon poisonings. Careful monitoring of the respiratory and cardiovascular status is needed following exposure. Most poisonings require only supportive treatments. Lidocaine or other antiarrhythmic agents should be used instead of catecholamines (epinephrine) for ventricular arrhythmias following hydrocarbon poisoning.

Hydrofluoric Acid/Hydrogen Fluoride

Hydrofluoric acid is HF that has been placed into an aqueous solution. This potent caustic substance can cause devastating local and systemic toxicity from exposure to an extremely small amount of concentrated liquid. The fluoride in HF leaches calcium and magnesium from body tissues, resulting in profound, often lethal hypocalcemia and hypomagnesemia. These electrolyte alterations also prompt a massive release of sequestered potassium into the systemic circulation. Rapid clinical deterioration may occur unexpectedly. Poisoning by ammonium bifluoride has a presentation and treatment approach similar to that of HF. Many interventions following HF poisoning are quite unusual and require individualized guidance. A toxicologist or a Poison Center should be consulted for specific treatment decisions about HF exposure.

Signs and Symptoms

Hydrogen Fluoride Toxicity

- Throat discomfort, bronchospasm, stridor, and local airway injury
- Wheezing, rhonchi, or rales
- Chemical pneumonitis or pulmonary edema
- Hypocalcemia (often severe)
- Hypomagnesemia

Transport Management

Hydrogen Fluoride Toxicity

- Perform eye and skin decontamination: copious irrigation.
- Manage the ABCs.
- For ingestion exposure
  - Provide immediate stomach evacuation with a nasogastric or orogastric tube.
  - Administer a calcium- or magnesium-containing substance such as milk, antacids, magnesium citrate, or magnesium hydroxide.
- For skin exposure
  - Administer calcium gluconate or calcium carbonate gel.

- Administer calcium gluconate or calcium chloride for impending or documented hypocalcemia or hyperkalemia.
- Monitor serum magnesium levels and replenish as necessary.

Hydrogen fluoride may cause throat discomfort, broncho-spasm, stridor, and local airway injury following inhalation. Lung auscultation reveals wheezes, rhonchi, or rales. Serious inhalation exposures can cause delayed chemical pneumonitis or pulmonary edema. Some sources suggest using a nebulized calcium gluconate solution for treatment.

Eye exposure requires thorough, copious irrigation. Early ophthalmologic consultation and evaluation is essential following ocular HF exposure. Dermal exposure also requires copious irrigation. Potentially contaminated clothing should be removed. Rescuers must use adequate PPE to protect against accidental contact with HF during decontamination.

Ingestions of large amounts or concentrated solutions of HF are often lethal. If available, immediate stomach evacuation with a nasogastric tube or orogastric tube should take place. Patients should also immediately ingest a calcium- or magnesium-containing substance such as milk, antacids, magnesium citrate, or magnesium hydroxide. Vomiting should not be induced. There is no benefit to using activated charcoal, and it may obscure endoscopy evaluation. The ingestion of HF may cause vomiting, abdominal pain, and gastritis, in addition to profound systemic toxicity.

Systemic effects from HF poisoning are primarily related to hypocalcemia and electrolyte imbalance. Arrhythmias, tetany, muscle spasm, vasospasm, and acidemia are common. Patients with hypocalcemia and hypomagnesemia also have prolongation of the QT interval.

Calcium is the antidote for HF poisoning. Skin exposure to HF is treated with calcium gluconate or calcium carbonate gel. Distal extremity involvement should prompt consultation with a hand surgeon or other appropriate specialist. Patients may be treated with local IV, intra-arterial, or subcutaneous calcium administration. *Calcium chloride should not be used for subcutaneous or intra-arterial administration.*

Systemic toxicity requires aggressive replenishment of calcium. Calcium gluconate or calcium chloride should be administered to any patients with impending or documented hypocalcemia or hyperkalemia and repeated as necessary; several grams of IV calcium may be required. Monitoring of serum magnesium levels is needed, with replenishment as indicated.

Highly Toxic Substances

A vast array of toxic medications and chemicals are discussed throughout this chapter. The topics in this chapter may appear random, but each substance has been included for one of three reasons:

1. The substance may be frequently encountered in the critical care transport environment.
2. Exposure to the substance may require a specialized or unusual approach to treatment.
3. It may be highly toxic in very small quantities, requiring greater attention when exposure is suspected.

CCTPs should be aware that a variety of substances can cause serious illness or death in very small quantities, especially in children. **Table 20-3** provides a list of these substances, which require special attention when even small quantities are taken by children.

Hazardous Materials (Hazmat) Response

Introduction

Hazardous materials incidents pose challenges to emergency responders and health care providers for a variety of reasons. Scenes may be in remote, almost inaccessible, locations or in dangerous proximity to major population centers, posing an immediate threat to the health and safety of a community **Figure 20-12**. In many cases, numerous chemicals are released simultaneously. Identification, isolation, and mitigation of a hazardous materials release is often hampered by inadequately prepared or a limited number of available emergency responders. Weather, terrain, and other environmental factors complicate conventional disaster response plans. CCTPs should expect involvement in these incidents as emergency responders, through inadvertent contact, or while performing evacuation and interfacility patient transports.

TABLE 20-3 Substances Highly Toxic to Children (1-Pill Killers)

Calcium channel blockers
Beta-blockers
Tricyclic antidepressants (TCAs)
Afrin (oxymetazoline)
Visine (tetrahydrozoline)
Benzocaine
Camphor
Clonidine
Iron preparations
Lindane (for scabies and lice)
Oil of wintergreen and other topical salicylates
Opiates
Sulfonylureas (for diabetes)

Sources: Nechas E. One-pill killers. Aetna InteliHealth Drug Resource Center. Available at: http://intelihealth.com/IH/ihtIH/WSIHW000/8124/25875/312529.html. Updated October 28, 2003. Accessed January 15, 2009.

Vorman R. Pediatric toxicology, part 2: what EMS providers need to know about "one-pill killers." *EMS Magazine*. 2008;37(5):88–92.

Vorman R. Pediatric toxicology, part 3: what EMS providers need to know about "one-pill killers." *EMS Magazine*. 2008;37(6):61–68.

Figure 20-12 Hazardous materials scenes pose challenges to providers because of the substances involved and the potential effect on the community.

Role at Hazmat Scenes

Unless CCTPs receive specific training in hazmat response, their role on the scene may be quite limited. Hazmat scenes often involve mechanical extrication, fire suppression, chemical spill containment, population evacuation, crowd and traffic control, the use of complicated PPE, comprehensive primary decontamination, and so forth, which CCTPs may be ill prepared to perform without specialized training and equipment. These skills are well beyond the scope of this text.

CCTPs without specialized training or equipment should remain well outside the "Hot" and "Warm" zones, discussed later, where provider exposure to toxic substances is possible. The area and shape of these zones will vary dramatically based on the chemicals involved, wind direction, topography, and other factors. CCTPs can quickly change roles from rescuer to victim as the wind changes if they are not appropriately positioned.

CCTPs should be prepared to evaluate decontaminated patients delivered by hazmat team personnel. It is imperative that CCTPs collaborate with hazmat team personnel to determine which substances are potentially involved and what PPE is appropriate for health care providers during treatment and transport. Poison Centers and Chemtrec will provide invaluable assistance if reliable, on-site reference materials are not available.

CCTPs may be called on to assist members of the hazmat team or other emergency responders directly. These personnel often become ill or injured during the course of scene operations. Comprehensive PPE can easily cause heat-related illnesses and dehydration, especially when used during unfavorable environmental conditions (for example, on a hot day a long walking distance from the staging area to the working area). Fires, falls, traffic incidents, and equipment-related activities all create the potential for trauma to emergency responders. Inadequate PPE for hazmat personnel and incorrect decisions related to the staging area or evacuation place anybody on the scene at increased risk of toxic chemical exposures.

The main objective of CCTPs at hazmat scenes should be to assess and ensure personnel safety during patient care and transportation. Patient assessment and treatment must occur only after personnel safety has been ensured. Rescuers may needlessly place themselves at risk while attempting to resuscitate a patient who has no chance of being revived. As stated earlier in the chapter, inadequate decontamination on the scene exposes CCTPs to toxic chemicals and compromises the safety of the transport vehicle.

Decontamination

The need, manner, and end point of decontamination are largely determined by the substance involved and

the degree of the patient's exposure to the substance. Many substances are colorless, odorless, and tasteless. Hazardous materials exposures with delayed toxic effects may continue to progress without the awareness of uninformed emergency responders.

A hazmat scene is configured with the chemical source in the center of three concentric circles **Figure 20-13**. The scene may be configured as any other shape if wind, topography, or other factors influence scene access, chemical distribution, and so forth. The inside circle surrounding the chemical source is the **hot zone**, where PPE is essential. The **warm zone** surrounds the hot zone and is where decontamination occurs; PPE is used (often the same PPE as in the hot zone), and extraneous personnel are not present. The **cold zone** surrounds the warm zone. No environmental hazards should be present in the cold zone. Rescuers providing direct patient treatment in the cold zone must still use PPE, but other emergency responders are not at risk. Furtado et al offer the following recommendations for rescuer decontamination at hazmat incidents. Contaminated patients should undergo a similar procedure.

1. Contaminated tools, trash, and clothing are dropped in the hot zone.
2. Primary rescuer garment wash and rinse in the decontamination (warm) zone.
3. Primary garment removal in the decontamination (warm) zone.
4. Secondary garment wash and rinse in the decontamination (warm) zone.
5. Facepiece removal and drop in the decontamination (warm) zone.
6. Boot drop in the decontamination (warm) zone.
7. Inner glove removal in the decontamination (warm) zone; all clothing and gloves should be turned inside out during removal, without shaking.
8. Final shower and clothing change in the cold zone.

This procedure may be modified based on the particular chemical and the other aforementioned factors. Steps should be taken to maintain patient privacy and avoid unintentional hypothermia during decontamination procedures. Specially trained hazmat personnel in appropriate PPE may initiate lifesaving measures before or during patient decontamination. Unless a CCTP has specialized training and adequate PPE, hazmat team personnel should provide all decontamination in the hot and warm zones.

Transport Considerations

Once adequate patient decontamination has been performed and other safety concerns have been addressed, patients should be transported to the closest appropriate health care facility. A small community hospital may be the facility closest to a hazmat incident. These facilities are often unable to provide adequate treatment for a critically ill patient requiring complex interventions. Small community health care facilities may not have sufficient quantities of antidotes or many of the infrequently used antidotes. CCTPs should consider bypassing these facilities in favor of larger tertiary centers when the risks of a longer transport time are outweighed by the benefits of access to greater health care resources.

CCTPs should carefully monitor for the following situations during patient transport:

- Deterioration in the patient's clinical condition as the toxic exposure evolves
- Signs of inadequate decontamination
- Any interventions appropriate for the patient's clinical situation, within the ability of the transport team, that have not been already performed

Any indications of inadequate primary decontamination warrant prompt correction. Unusual smells or fumes in the transport vehicle, unexplained symptoms experienced by transport personnel, and new knowledge about the suspected toxic chemical may require immediate aircraft landing, vehicle

evacuation, and/or additional provider PPE.

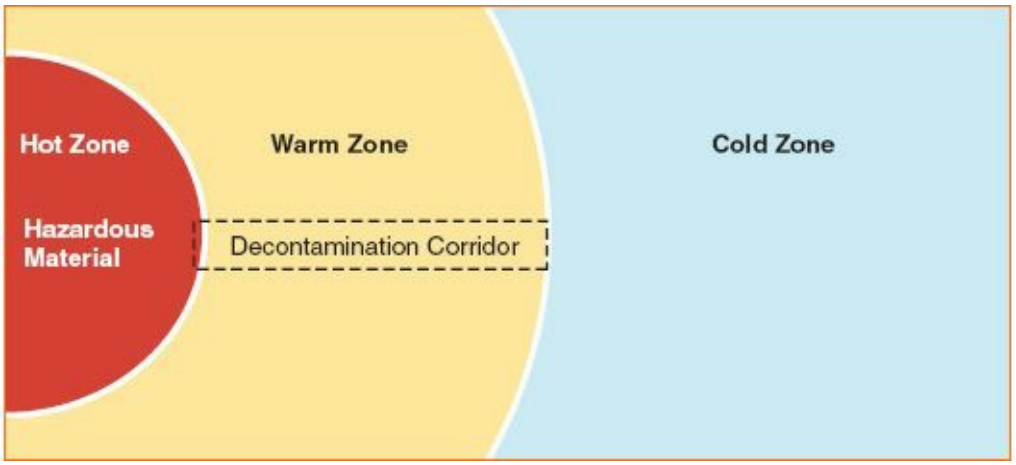


Figure 20-13 Configuration of a hazmat scene.

Patient transport further requires an adequate report to the receiving facility, including the aforementioned elements, and thorough, accurate documentation of the patient condition, interventions performed, and status or events during transport. Crews should weigh the benefits of providing a hazmat container to the receiving facility staff (for substance identification purposes) against any additional risks that transporting this container may pose. A common gasoline container provides minimal benefit with a high risk, whereas a well-sealed container of an unusual substance that is not highly toxic may greatly improve subsequent patient care while posing negligible increased risk. Every situation requires special consideration.

Radiation Emergencies

The threat of radiation exposure or nuclear incident is ever present in society. Ionizing radiation is capable of causing injury from the release of heat generated from a controlled or uncontrolled reaction. It is important to evaluate the nature of a particular radiation event to determine if there is any risk to emergency responders and health care providers. Radiation release may occur in health care, industrial, or laboratory settings; during transportation and disposal of radioactive materials; and following a military or terrorist event. Approach to treatment of a potentially exposed patient hinges on the distinction between **irradiation** and **contamination**. Health care providers and emergency responders are not placed at any risk by irradiated patients. In contrast, patients with radioactive contamination spread this material and expose others to radiation. Continued irradiation occurs until contaminating materials are contained or otherwise removed. The use of a radiation detection device (discussed later) helps identify which patients are contaminated with radioactive particles.

Further radiation exposure occurs during **incorporation**. Devastating individual patient toxicity develops when radioactive materials enter the body, causing ongoing internal exposure.

Figure 20-14 A radiation detection device.

Clues to the diagnosis of radiation exposure may be available by a number of possible methods. A history of radiation exposure through patient report of an isolated event or widespread knowledge of a large-scale event will greatly assist in diagnosis. The military, health care facilities, emergency responders, laboratories, and industrial settings have various radiation detection devices available **Figure 20-14**. Radioactive particles may be present in a patient's mucous membranes or body fluids and tissues. Radiation poisoning (**acute radiation syndrome**) may also be diagnosed clinically in potentially exposed people.

Emergency responders should develop a formal radiation response plan. Providers working in proximity to a radiation release need appropriate protective clothing for the situation and must use respirators to avoid inhaling contaminated material. There is little to no risk to the health care provider when caring for a patient who has been removed from the source of contamination. The patient should be decontaminated before the initiation of care, unless immediate life threats exist. Once decontaminated, no risk exists to the health care provider from radiation.

Patient decontamination involves removing contaminated clothing and jewelry and providing copious irrigation and thorough washing of the skin with soap and water. Providers should also be checked and decontaminated if necessary. Patients who are exposed to only electromagnetic radiation do not require decontamination. Following decontamination, patients should be reassessed for persistent radiation by using a hand-held or similar device. Persistent radiation requires repeated decontamination.

Patients may die within several hours of a massive radiation exposure. Early symptoms include nausea, vomiting, and diarrhea; abdominal pain; fever; mental status changes; coma; and shock. Following the first 1 to 2 days, patients may have a brief recovery period. Subsequent toxic effects include multisystem organ dysfunction, bone marrow depression, sepsis, hair loss, dermatologic injury, and eventual death.

Initial treatment is focused on removal of any accessible radioactive substances in the body. Chelating agents may bind and remove certain radioactive substances. Forced diuresis may also be helpful. Additional treatments are based on presenting symptoms and directed toward minimizing the potential for catastrophic infection.

Radiation burns appear the same as other burns, and their management varies only by the need to decontaminate the wound of radioactive particles. In addition, for massive doses of radiation, the systemic effects of radiation exposure should be considered and treated. Wounds should be dressed in clean, dry dressings.

Flight Considerations

The importance of safety during air-medical transport cannot be overstated when transporting patients following a poisoning, overdose, or toxic exposure. Affected patients pose a serious risk to the lives and health of the transport team.

As stated repeatedly throughout this chapter, patients should not be placed into a transport vehicle, especially a helicopter or airplane, until an effective initial decontamination has been completed. Unlike ground transport vehicles that can stop almost anywhere, helicopters and airplanes require safe areas to land that are often unavailable when conditions in the transport vehicle become unsafe owing to the presence of toxic substances from a contaminated patient. In many cases, effective decontamination requires the removal of all contaminated clothing and adequate cleansing and irrigation for a particular substance before air transport can begin.

Air-medical crews are also at risk from patients (or family members) with bizarre, aggressive, combative, or violent behavior. It is essential to screen patients (and family members) before transport and to use appropriate sedation and restraints. At least one fatal air medical crash has been attributed to a patient interfering with aircraft operation. The risk from patients or family members interfering with safe aircraft or transport vehicle operation should not be underestimated.

During air-medical transport, access to patients is often limited. In the confined space of an aircraft, with environmental variables and loud noises, it is often impossible to perform an adequate patient assessment and many therapeutic interventions. Noise and vibration undermine patient monitoring. Low light conditions in many aircraft make recognizing skin color changes, respiratory alterations, and even subtle emesis difficult to detect. Air-medical crew must be especially prepared to anticipate and manage changes during the flight.

Summary

The management of patients after poisoning or overdose is often a challenging, complicated endeavor. CCTPs must ensure transport team safety while optimizing the chances for patient recovery following a toxic exposure. This chapter serves only as a general overview of various toxicologic emergency situations. During actual patient care, early consultation with a toxicologist, a Poison Center, or a reliable toxicology reference will provide invaluable assistance. CCTPs must never lose focus on safety and the ABCs when treating or transporting patients experiencing a toxicologic emergency.

Case Study

YOU ARE A MEMBER OF A HELICOPTER FLIGHT CREW and you have been called to a rural facility to transport an 18-year-old male who reportedly took his mother's amitriptyline (a TCA) after his girlfriend broke up with him.

On arrival you receive a report from the attending physician. He states that the patient took an unknown amount of the TCA, amitriptyline, 1 hour before his arrival at the emergency department. The patient has been in the hospital for approximately 45 minutes. The patient was administered 60 g of activated charcoal when he arrived. The physician reports that all the patient's laboratory values were normal and he recently received the patient's arterial blood gas measurements, which were as follows: pH, 7.37; Pa_{CO_2}, 43 mm Hg; Pa_{O_2}, 80 mm Hg; and oxygen saturation, 96%. On an ECG, the only other finding was sinus tachycardia at a rate of 118 beats/min with a prolonged QRS interval at 120 ms. The patient has a 20-gauge antecubital IV line in place.

When you obtain the patient's history from his mother, she tells you that she takes 75 mg of amitriptyline at bedtime for depression. She said she receives 2 week's worth of medication at a time,

and she just filled her prescription. She found her son asleep on his bed with an empty pill bottle beside him. She was able to wake him up and then called 9-1-1. The mother states that her son told her that his girlfriend just broke up with him and that he just wanted to "end it all."

On your arrival at the patient's bedside you find an 18-year-old male who appears lethargic and currently lying on his side vomiting black charcoal. His vital signs are as follows: blood pressure, 92/60 mm Hg; temperature, 97°F (36.1°C); pulse rate, 118 beats/min; respiratory rate, 24 breaths/min; and oxygen saturation, 92%. His Glasgow Coma Scale score is 11. He opens his eyes to pain and is able to localize pain. His conversation is confused. The patient has scattered rhonchi throughout consistent with probable aspiration. You are unable to auscultate bowel sounds, and he has diminished reflexes.

1. What are your priorities with this patient prior to transport?
2. What other priorities would be important for the treatment of this patient?

Analysis

One of your most important priorities prior to transport is airway management. Depending on your transport vehicle, some providers might elect to "load and go" and secure the airway en route; however, your patient is already at high risk for aspiration and it would probably be more effective to manage the airway in the more controlled environment of the emergency department prior to transport. This patient needs rapid sequence intubation with both sedative and short-term paralytic medication. Nasogastric intubation would then be accomplished after proper sedation/paralysis was achieved. Cricoid pressure is also important during intubation to prevent any possible further aspiration. Long-term paralysis with a nondepolarizing paralytic should be avoided (unless your service has continuous electroencephalographic monitoring). Patients who have overdosed with TCAs are at high risk for seizures. Patients whose QRS interval is greater than 100 ms have up to a 34% chance of having seizures and up to a 14% chance of having a life-threatening arrhythmia. Once the patient has been paralyzed with a long-term paralytic you would be unable to tell if the patient was seizing.

Sedation is required to enable the patient to tolerate the ET tube. An anxiolytic agent such as lorazepam or midazolam should be used. These agents also protect against seizures. The typical sedative dose for midazolam is 0.025 to 0.05 mg/kg IV. If you are administering midazolam for seizures, the usual loading dose is much higher, 0.1 mg/kg IV or greater. Propofol, a short-acting sedative, can be used if your service carries it and/or the sending agent stocks the medication. The ET tube should be secured prior to transport, and a nasogastric tube should be placed. You would also want to instill 60 g of charcoal into the nasogastric tube and then clamp it prior to transport because the patient has vomited the first dose.

Lastly, you need to secure one or two additional large-bore IV tubes. Because of the anti-alpha-adrenergic effect of TCAs, this patient is at high risk for further hypotension. He will probably require aggressive fluid resuscitation to stabilize his blood pressure while en route. If the patient remains hypotensive despite fluids, vasopressors with an alpha-agonist effect (norepinephrine) would be administered to maintain blood pressure. The additional IVs should be started in flight.

Serum alkalinization with sodium bicarbonate is important in treating patients with a tricyclic overdose. Most Poison Centers recommend serum alkalinization if the QRS duration is 100 ms or greater, if the pH is less than 7.1, or if the patient is hypotensive or having arrhythmias. Alkalinization protects patients from the cardiotoxic effects of TCAs. Correction with sodium bicarbonate promotes protein binding of TCAs and improves myocardial contractility. The goal of therapy is a pH range between 7.5 and 7.55.

Sodium bicarbonate is initially given as a bolus of 1 to 2 mEq/kg IV. You then can begin an IV drip with 150 mEq of sodium bicarbonate per liter of 5% dextrose in water and titrate to keep your pH within

the goal (1 to 3 mL/kg/h). Frequent pH determinations are needed. This is difficult during transport, unless you have point-of-care testing in the helicopter. If the sending institution has started this therapy, it is usually maintained during the flight and it is not titrated until the pH is checked at the receiving facility. If the sending institution has not initiated this, typically the bolus dose is given and the drip would be started at the receiving institution.

Prep Kit

Ready for Review

- Poisons, chemicals, medications, and other toxic substances are found in dangerous quantities in homes, schools, agriculture, industry, and commercial establishments; on all modes of transportation; and naturally in the environment.
- If rescuers or health care providers do not use the correct PPE before initial patient contact, it may be too late for the equipment to provide adequate protection.
- CCTPs may encounter hazardous situations during anticipated patient transports between health care facilities, while responding to an emergency in the community, or by happening on a vehicular crash.
- Patients may sometimes exhibit violent, bizarre, or aggressive behavior, such that chemical or physical restraints may be necessary before effective assessment or treatment can begin.
- All patients should be evaluated for the presence of weapons before transport.
- Once the safety concerns of health care providers and emergency responders have been adequately addressed, the CCTPs should evaluate and manage any problems with the patient's ABCs.
- As with all patients, maintaining or establishing a patent airway is the top priority in the management of patients with a toxic exposure.
- A previously patent airway may become compromised during patient transport owing to alteration in the patient's mental status or seizure activity, evolving structural abnormalities from trauma or increasing edema, or obstructions from emesis, secretions, or a foreign body that becomes displaced during the transport.
- Patients who received activated charcoal or syrup of ipecac have an unusual risk of subsequent airway compromise.
- Breathing is second only to airway in importance during patient assessment. Without effective oxygenation and ventilation, tissue hypoxia, acidosis, and cell death will occur.
- Patients characterized by physiologic stress, acidosis, hypermetabolic states (such as in pediatrics), and exposure to many toxic substances require greater oxygenation and ventilation than would usually be required.
- Cyanosis, fatigue, and dyspnea indicate that a patient's respiratory status may be compromised.
- Lung auscultation revealing wheezing, pulmonary edema, and decreased, absent, or coarse breath sounds indicates unstable respiratory status.
- Evaluation of a patient's circulation requires assessment of end-organ tissue perfusion and overall cardiovascular functioning.
- During the evolution of a toxic exposure, a patient's circulatory status may decline because of any of the following:

- Direct exposure to cardiotoxic or vasoactive substances
- Fluid volume loss or redistribution
- Electrolyte disturbance
- Airway or respiratory compromise with secondary cardiac dysfunction
- Manifestations of preexisting cardiovascular disease
- Altered blood transport ability

- The patient, family, and bystander interviews may provide valuable clues to the nature and severity of a toxic exposure, including information about prodromal symptoms and preexisting medical problems.
- Transport teams must balance the usefulness of a complete head-to-toe physical examination with concerns for patient thermoregulation, privacy, patient access in confined transport vehicles, and the clinical needs of the patient.
- Hemodynamic and respiratory alterations (for example, tachycardia, bradycardia, hypertension or hypotension, and hyperthermia or hypothermia) may occur as a result of direct action on organs and tissues or through effects on the central nervous system.
- Advances in point-of-care testing technology allow CCTPs to measure blood chemistry values, arterial or venous blood gases, and other assorted values during transport and obtain results almost immediately.
- Exposure to certain classes of chemicals and medications produces a readily identifiable pattern of clinical signs and symptoms (toxidrome), which aids in diagnosis when the exact nature of the exposure is unclear.
- Some patients may experience life-threatening reactions to medications taken at therapeutic doses.
- Anticholinergic syndrome occurs following excessive exposure to medications such as antihistamines, atropine, and benztropine, resulting in muscarinic receptor blockade at the neuromuscular junction.
- Cholinergic syndrome (cholinesterase inhibitor toxicity) occurs following exposure to organophosphate and carbamate insecticides or to certain chemical nerve agents. Cholinesterase inhibitors may affect nicotinic receptors, muscarinic receptors, or both and alter the function of the neurotransmitter acetylcholine.
- Opioid syndrome commonly develops following illicit use or abuse of opioids in the community or as an adverse consequence of a therapeutic error or accidental ingestion of opioids (such as morphine, heroin, and fentanyl).
- The administration of an opioid antagonist as a treatment for opioid syndrome may precipitate severe, violent withdrawal symptoms. Many opioids also exert effects much longer than the duration of opioid antagonists, allowing for the return of life-threatening symptoms.
- Sympathomimetic syndrome involves overstimulation of the adrenergic nervous system, resulting in tachycardia, hypertension, agitation, seizures, hyperthermia, dilated pupils, and diaphoresis.
- Malignant hyperthermia occurs following the administration of succinylcholine or certain inhaled anesthetic agents to genetically susceptible patients and is characterized by muscle spasms, profound muscle rigidity, acidosis, hyperthermia, tachycardia, tachypnea, myoglobinuria, rhabdomyolysis, and hyperkalemia.
- Neuroleptic malignant syndrome is a potentially fatal reaction to antipsychotic and antischizophrenic medications in which hyperthermia, profound muscle rigidity, metabolic acidosis, confusion, and, in severe cases, renal failure, respiratory failure, arrhythmias, and cardiovascular collapse develop.
- Serotonin syndrome is an unusual response to serotonin-altering medications causing

hyperserotoninergic symptoms (for example, irritability, muscle rigidity, hyperthermia, diaphoresis, headaches, seizures, coma, tachycardia, and hallucinations).

- Interventions to maintain and enhance the ABCs should not be delayed in favor of interventions specific to toxicologic emergencies (decontamination, enhanced elimination, and antidote administration), which will not become effective rapidly enough to correct immediately life-threatening problems with the ABCs.
- Primary decontamination usually occurs at the scene or outside a health care facility or transport vehicle and is intended to protect rescuers and health care providers from exposure to a toxic substance during patient care and transport.
- Secondary decontamination, which is directed at minimizing patient absorption or injury from a toxic substance, poses much less risk to health care providers than does primary decontamination.
- Decontamination of a hazardous chemical should occur before an unprotected health care provider makes any contact with the material, whether the material is found at the site, on the patient, or on other providers.
- Potentially suicidal, combative, aggressive, violent, intoxicated, and confused patients should be restrained—using physical or chemical restraints—before transport.
- Endotracheal intubation provides optimal protection for an obtunded or otherwise at-risk patient.
- Conventional ventilator settings are often inadequate for a patient's needs following a poisoning or overdose.
- Oxygen saturation, blood gas analysis, and $ETCO_2$ provide valuable information about the adequacy of oxygenation and ventilation; ongoing monitoring of respiration focuses on changes in lung sounds, patient appearance, and cardiovascular function (as shown by an ECG and assessment of perfusion).
- Toxic exposures may cause alterations in vascular tone, kidney function, and electrolyte concentration, thereby disrupting fluid balance.
- Vascular access sufficient for administering emergency medications and fluids should be established in any patient exposed to a potentially harmful substance.
- Every patient must receive adequate sedation and analgesia while chemically paralyzed and should be prophylactically medicated against seizures.
- Patients with increased drug tolerance or a hypermetabolic state require significantly more sedation and analgesia while chemically paralyzed.
- Nonintubated patients require anxiolysis and analgesia following many toxicologic exposures.
- Psychiatric and other at-risk patients may require sedative medications to facilitate patient care and promote patient comfort.
- Patient decontamination following exposure to a toxic substance is directed at minimizing the quantity of the substance absorbed by the patient and the extent of any local damage. Decontamination may be as simple as providing the patient with fresh air or as complicated as whole-bowel irrigation.
- Caustic agents and acetylcholinesterase inhibitors can cause systemic toxic effects from dermal absorption; other chemicals cause severe local reactions or burns when placed in contact with skin.
- Flushing with water or saline (if the chemical is not water-reactive) is the recommended approach for removal of chemical agents from the patient's skin or eyes.
- Methods for gastrointestinal decontamination include activated charcoal administration, gastric lavage and emptying, and whole-bowel irrigation; however, gastric lavage and whole-bowel irrigation are not

done in the transport environment.

- Enhanced elimination may be considered if the patient is exposed to lethal quantities of a toxic substance, the ordinary route of elimination from the body is impaired by the overdose or other pathology, or the patient will likely not tolerate adverse effects of the poisoning because of a preexisting medical condition.
- Acetaminophen toxicity, which can result in liver failure in severe cases, is treated with initial gastrointestinal decontamination with activated charcoal, followed by oral or IV acetylcysteine (Mucomyst).
- In the field, treatment of an amphetamine overdose—which presents with classic sympathomimetic symptoms—is largely supportive.
- The majority of patients who experience benzodiazepine overdose require only conservative management and careful observation.
- Glucagon is the treatment of choice for a beta-blocker overdose.
- One tablet of a calcium channel blocker is often enough to cause serious toxicity or death in a small child. IV calcium is the antidote to an overdose of a calcium channel blocker.
- Symptoms of cardiac glycoside toxicity are often vague, frequently causing misdiagnosis.
- There is no antidote for cocaine; the treatment for overdose of this illicit drug is supportive and based on the clinical presentation.
- Patients who experience an opioid overdose have profound CNS and respiratory depression. Naloxone —which may be administered intravenously, intramuscularly, subcutaneously, via ET tube, or intranasally—will reverse opioid effects and often reestablish a patent airway and spontaneous breathing.
- Any new patient complaint of tinnitus, dizziness, or deafness should prompt an evaluation of possible salicylate poisoning. There is no antidote for salicylate poisoning, but this condition is a candidate for enhanced elimination.
- In selective serotonin reuptake inhibitor (SSRI) overdose situations with serotonin syndrome, the treatment is supportive and based on presenting symptoms.
- Ethylene glycol and methanol can cause profound, lethal metabolic acidosis following ingestion of even very small quantities of these chemicals. Two antidotes are available for the treatment of methanol and ethylene glycol poisoning: ethanol and fomepizole.
- Any patient admitted to a health care facility for an intentional overdose should be screened for the presence of tricyclic antidepressants (TCAs) and given aggressive supportive treatment following a TCA overdose.
- The potential for rescuer exposure is high when the incident involves acetylcholinesterase inhibitors, such as during unintentional contact with certain pesticides or as part of a chemical weapon release. Skin and eye decontamination should be initiated promptly by rescuers or health care providers using the appropriate PPE.
- The brain and heart are the primary sites of carbon monoxide (CO) toxicity owing to their large oxygen requirements. If any doubt exists regarding possible CO exposure, aggressive treatment should be started before laboratory results are obtained.
- The goal of therapy in case of CO toxicity is to displace the CO, under pressure, from the hemoglobin and the mitochondria. Patients with altered mental status, seizures, or serious burns in the setting of CO poisoning should have their airway actively managed by endotracheal intubation.

- Exposure to caustic agents can occur through inhalation, ingestion, dermal exposure, or eye exposure; ingestion of caustic agents has the potential to cause devastating injury.
- Chlorine, ammonia, and asphyxiate gases have the potential to displace oxygen in a patient's lungs; damage or irritate the mouth, nose, and airway; or otherwise disrupt effective respiration. Symptomatic patients may require supplemental oxygen, bronchodilator medications, humidified oxygen or air, or assisted ventilation in severe exposures.
- Cyanide poisoning is confirmed with serum cyanide and thiocyanate levels. Patients have a multitude of neurologic, cardiovascular, and respiratory symptoms. Any exposure to cyanide should be considered potentially lethal. Treatment includes administration of amyl nitrite, administration of IV sodium nitrite, and then administration of sodium thiosulfate. These antidotes are commercially available.
- Abuse of hydrocarbons occurs from "huffing" various highly volatile hydrocarbons. Sudden sniffing death syndrome occurs when people (usually adolescents) are startled while secretly huffing hydrocarbons.
- Hydrocarbons such as gasoline, kerosene, and petroleum ether pose primarily a risk of chemical pneumonitis following aspiration; the care of patients who have toxic exposures to these substances is supportive.
- Hydrofluoric acid is a potent caustic substance that can cause devastating local and systemic toxicity from exposure to an extremely small amount of concentrated liquid. Calcium is the antidote for hydrogen fluoride poisoning.
- A variety of substances can cause serious illness or death in very small quantities, especially in children. The top three include calcium channel blockers, beta-blockers, and TCAs.
- CCTPs without specialized training or equipment should remain well outside the "hot" and "warm" zones where provider exposure to toxic substances is possible.
- Poison Centers and Chemtrec may provide invaluable assistance to health care providers at incidents involving hazardous materials.
- The main objective of CCTPs at hazmat scenes is to assess and ensure personnel safety during patient care and transportation; only then can patient assessment and treatment occur.
- A hazmat scene is configured with the chemical source in the center of three concentric circles: the hot (contaminated) zone, where PPE is essential; the warm zone, where PPE is used and extraneous personnel are not present; and the cold zone, where no environmental hazards should be present.
- Once adequate patient decontamination has been performed and other safety concerns have been addressed, patients involved in hazmat incidents should be transported to the closest appropriate health care facility.
- CCTPs should carefully monitor for the following situations during patient transport:
  - Deterioration of the patient's clinical condition as the toxic exposure evolves
  - Signs of inadequate decontamination
  - Any interventions appropriate for the patient's clinical situation, within the ability of the transport team, that have not been already performed
- Radiation release may occur in health care, industrial, or laboratory settings; during transportation and disposal of radioactive materials; and following a military or terrorist event. Patients may die within several hours of a massive radiation exposure. The use of a radiation detection device will help identify which patients are contaminated with radioactive particles.
- Radiation burns appear the same as other burns; their management varies only by the need to

decontaminate the wound of any radioactive particles. In the flight environment, effective initial decontamination before take-off is extremely important. Other flight considerations for critical toxicologic emergencies include thoroughly screening patients and family members to prevent violence and behavioral problems during flight and anticipating changes in patient condition that may be difficult to detect during flight.

Vital Vocabulary

acetylcholine A chemical neurotransmitter in the parasympathetic nervous system.

acetylcholinesterase An enzyme that breaks down acetylcholine at the neuromuscular junction.

activated charcoal A carbon-based liquid with an incredible absorptive ability that is typically administered orally or via nasogastric or orogastric tube to decrease the available quantity of a toxic substance.

acute radiation syndrome Radiation poisoning.

adsorbtion The process of attracting molecules of a substance to the surface of that substance.

anticholinergic syndrome A syndrome that occurs following excessive exposure to medications such as antihistamines, atropine, and benztropine (Cogentin), or other substances such as Jimson weed, resulting in muscarinic receptor blockade at the neuromuscular junction; characterized by tachycardia, hyperthermia, dilated pupils, warm (or hot) dry skin, ileus, delirium, seizures, psychosis, and urinary retention.

anxiolysis The reduction of anxiety by the administration of an antianxiety agent.

body packers People who swallow carefully packaged capsules for smuggling purposes.

body stuffers People who hastily swallow illicit substances to avoid impending arrest.

chelating agents Chemical substances that bind to heavy metals to remove them from the body.

cholinergic syndrome Cholinesterase inhibitor toxicity.

cholinesterase An enzyme that hydrolyzes acetylcholine to acetic acid and choline.

coagulation necrosis The death of tissue resulting from a blockage of blood flow to that tissue, in which the blockage is caused by clots preventing blood from reaching the tissue.

cocaine washout syndrome Profound exhaustion with the ability to regain normal mental status and orientation when aroused; common with heavy cocaine users.

cold zone The area surrounding the warm zone at a hazardous materials scene. No environmental hazards should be present in this zone.

contamination A state in which a certain substance, such as blood, infectious material, or a toxic substance, is present on an item or surface; in the case of radiation, it is important to distinguish between this and irradiation.

DUMBELS Mnemonic for symptoms of anticholinesterase inhibitor toxicity: Diaphoresis/diarrhea, Urination, Miosis, Bradycardia/bronchospasm/bronchorrhea, Emesis, Lacrimation, and Salivation.

enhanced elimination A process that replaces or augments the body's normal method of eliminating, modifying, or breaking down toxic substances.

gluconeogenesis Glucose formation.

glycogenolysis The breakdown of glycogen to glucose.

hemoperfusion A treatment in which a patient's blood is filtered outside the body through a substance that removes toxic substances from it; a method of removing toxic substances from the blood.

hepatotoxicity Capable of damaging the liver.

hot zone The area immediately surrounding the chemical source at a hazmat scene.

incorporation In the radiologic context, a process in which radioactive materials enter the body during radiation exposure, causing ongoing internal exposure and creating devastating individual patient toxicity.

inotropic Affecting the contractility of muscle tissue, especially cardiac muscle.

irradiation Exposure to radiation; it is important to distinguish between this and contamination because health care providers and emergency responders are not placed at any risk by patients who have been exposed but are not contaminated.

liquefactive necrosis Tissue death caused by bacterial or fungal infections in which cellular destruction has occurred, leaving a lesion filled only with pus and the liquid remains of the tissue.

malignant hyperthermia A condition that may occur after administration of certain inhaled anesthetics and is characterized by muscle spasms, rigidity, acidosis, hyperthermia, tachycardia, tachypnea, myoglobinuria, rhabdomyolysis, and hyperkalemia.

methamphetamine washout syndrome Excessive sleep, hunger, and depression as a result of excessive amphetamine use.

neuroleptic malignant syndrome (NMS) A potentially fatal reaction to antipsychotic and antischizophrenic medications, characterized by patient hyperthermia, profound muscle rigidity, metabolic acidosis, and confusion.

opioids The class of chemicals that includes naturally derived opiates and newer artificial medications with the same properties.

opioid syndrome Toxicity that develops following illicit use and abuse of opioids.

phosphodiesterase An enzyme that helps break phosphodiester bonds, creating smaller nucleotides.

point-of-care testing Laboratory testing that is performed at the point of care, for example, the bedside, so that results can be quickly obtained and considered while decisions are being made about patient care.

primary decontamination A form of decontamination that usually occurs at a scene or outside a health care facility or transport vehicle to protect rescuers and health care providers from exposure to a toxic substance during patient care and transport.

prodromal Referring to the early signs and symptoms that occur before a disease or condition, such as a toxic exposure, fully appears, for example, dizziness before fainting.

pulse CO-oximetry A noninvasive screening and monitoring method for methemoglobinemia and carbon monoxide exposure.

rhabdomyolysis The destruction of muscle tissue.

secondary decontamination A form of decontamination that is directed at minimizing patient absorption or injury from a toxic substance; occurs after initial decontamination has occurred outside a health care facility or transport vehicle.

serotonin syndrome An unusual response to serotonin-altering medications causing hyperserotoninergic symptoms.

SLUDGEM Acronym for Salivation, Lacrimation, Urination, Diarrhea, Gastroenteritis, Emesis, and Miosis, which are symptoms of anticholinesterase inhibitor toxicity.

sudden sniffing death syndrome Occurs when a surge of catecholamines on a sensitized myocardium causes ventricular fibrillation or ventricular tachycardia; can occur when a person is startled while huffing hydrocarbons.

sympathomimetic syndrome A syndrome that involves over-stimulation of the adrenergic nervous system, resulting in tachycardia, hypertension, agitation, seizures, hyperthermia, dilated pupils, and diaphoresis.

toxidrome A group of symptoms, or syndrome, associated with toxicity of a given substance.

volatile Easily changed from liquid to gas form.

warm zone Area surrounding the hot zone at a hazmat incident.

whole-bowel irrigation (WBI) A gastric decontamination method that involves the patient consuming large quantities of a nonabsorbable, electrolyte-balanced liquid that propels stomach and intestinal contents through the digestive system.

Bibliography

Albertson TE. Amphetamines. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:73–76.

Albertson TE. Opiates and opioids. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:288–292.

Anderson IB. Ethylene glycol and other alcohols. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:193–197.

Azer M, Bailey H. Toxicity, salicylate. Updated November 8, 2007. Available at: http://www.eMedicine.com/MED/topic2057.htm. Accessed May 1, 2008.

Badawy MK, Maffei FA. Toxicity, selective serotonin reuptake inhibitor. May 18, 2006; updated November 12, 2008. Available at: http://www.eMedicine.com/PED/topic2786.htm. Accessed May 26, 2008.

Bartkus A. Toxicologic emergency transport. In: Clark DY, Stocking J, Johnson J, eds. *Flight and Ground Transport Nursing Core Curriculum.* Denver, CO: Air and Surface Transport Nurses Association; 2005:593–633.

Basic Disaster Life Support (BDLS). 2nd ed. Augusta, GA: National Disaster Life Support Education Consortium; 2001.

Benowitz NL. Beta-adrenergic blockers. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:131–133.

Blanc PD. Cyanide. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:176–179.

Brubacher JR. Beta-adrenergic agents. In: Goldfrank LR, Howland MA, Flomenbaum NE, Hoffman RS, Lewin NA, Nelson LS, eds. *Goldfrank's Toxicologic Emergencies,* 7th ed. New York, NY: McGraw-Hill; 2002:741–757.

Bruns JJ Jr. Toxicity, anticholinergic. Updated March 26, 2008. Available at: http://www.eMedicine.com/emerg/topic36.htm. Accessed May 26, 2008.

Defendi GL, Tucker JR. Toxicity, acetaminophen. May 15, 2007; Updated December 9, 2008. Available at: http://www.eMedicine.com/ped/topic7.htm. Accessed May 1, 2008.

Farrell S. Toxicity, acetaminophen. Updated October 3, 2007. Available at: http://emedicine.medscape.com/article/820200-overview. Accessed March 2, 2009.

Frommer DA, Kulig KW, Marx JA, Rumack B. Tricyclic antidepressant overdose: a review. *JAMA.* 1987;257(4):521–526.

Furtado MC, Walter FG, Klein R. Personal protective equipment and decontamination. In: Walter FG, Klein R, Thomas RG, eds. *Advanced Hazmat Life Support Provider Manual,* 3rd ed. Tucson, AZ: University of Arizona; 2003.

Goldfarb DS. Principles and techniques applied to enhance the elimination of toxic compounds. In: Goldfrank LR, Howland MA, Flomenbaum NE, Hoffman RS, Lewin NA, Nelson LS, eds. *Goldfrank's Toxicologic Emergencies,* 7th ed. New York, NY: McGraw-Hill; 2002:58–68.

Goto CS. Hydrocarbons. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:219–222.

Hannum J, Ly BT. Hydrogen fluoride and hydrofluoric acid. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:222–224.

Hoffman RS, Nelson LS, Howland MA, Lewin NA, Flomenbaum NE, Goldfrank LR, eds. *Goldfrank's Manual of Toxicologic Emergencies.* New York, NY: McGraw-Hill; 2007.

Horowitz BZ. Toxicity, calcium channel blocker. Updated December 4, 2008. Available at: http//:www.eMedicine.com/emerg/TOPIC75.htm. Accessed January 22, 2009.

Howland MA. Antidotes in depth. In: Goldfrank LR, Howland MA, Flomenbaum NE, Hoffman RS, Lewin NA, Nelson LS, eds. *Goldfrank's Toxicologic Emergencies,* 7th ed. New York, NY: McGraw-Hill; 2002:502–506.

Kerr GW, McGuffie AC, Wilkie S. Tricyclic antidepressant overdose: a review. *Emerg Med J.* 2001;18(4):236-241.

Koschel MJ. Management of the cyanide-poisoned patient. *J Emerg Nurs.* 2006;32(4 suppl):S19–S26.

Levine MD, Johnson JJ, Barron TP. Toxicity, hydrocarbons. September 6, 2006. Available at: http://www.eMedicine.com/EMERG/topic873.htm. Accessed June 3, 2008.

Ma OJ, Cline DM, Tintinalli JE, Kelen G, Stapaczynski S. *Emergency Medicine Manual,* 6th ed. New York, NY: McGraw-Hill; 2004.

Mack E. Focus on diagnosis: CO-oximetry. *Pediatr Rev.* 2007;28:73–74.

Malignant Hyperthermia Association of the United States. Emergency therapy for malignant hyperthermia. May 2008. Available at: http://medical.mhaus.org/PubData/PDFs/treatmentposter.pdf. Accessed May 26, 2008.

Mancini MC, Deshpande GG. Neuroleptic malignant syndrome. August 14, 2006; updated December 9, 2008. Available at: http://www.eMedicine.com/PED/topic1581.htm. Accessed May 26, 2008.

Masimo Corporation. Noninvasive measurement of methemoglobin, carboxyhemoglobin and oxyhemoglobin in the blood. Available at: http://www.masimo.com/rad-57/. Accessed July 2, 2008.

Merck Manuals Online Medical Library. *Acetaminophen poisoning.* Updated November 2005. Available at: http://www.merck.com/mmpe/sec21/ch326/ch326c.html#BGBHJFCE. Accessed March 2, 2009.

Merck Manuals Online Medical Library. *General principles: poisoning.* Updated November 2005. Available at: http://www.merck.com/mmpe/sec21/ch326/ch326b.html. Accessed May 26, 2008.

Mullen WH. Caustic and corrosive agents. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:147–159.

Mullen WH. Flumazenil. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:452–453.

Murphy NG, Benowitz NL. Calcium antagonists. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:143–146.

Newton EH, Shih RD, Hoffman RS. Cyclic antidepressant overdose: a review of current management strategies. *Am J Emerg Med.* 1994;12(3):376–379.

Olson KR. Comprehensive evaluation and treatment. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:5–30.

Roldan CJ, Habal R. Toxicity, cocaine. May 30, 2008. Available at: http://www.eMedicine.com/med/topic400.htm. Accessed June 1, 2008.

RxList. Nitropress. Available at: http://www.rxlist.com/cgi/generic/nitroprusside_ad.htm. Accessed June 3, 2008.

Sharma A, Tefera L. Toxicity, beta-blocker. January 8, 2007; updated August 20, 2008. Available at: http://www.eMedicine.com/emerg/topic59.htm. Accessed June 1, 2008.

Shochat G, Lucchesi M. Toxicity, carbon monoxide. August 8, 2008. Available at: http://eMedicine.medscape.com/article/819987-overview. Accessed February 6, 2009.

Smilkstein MJ. Techniques used to prevent gastrointestinal absorption of toxic compounds. In: Goldfrank LR, Howland MA, Flomenbaum NE, Hoffman RS, Lewin NA, Nelson LS, eds. *Goldfrank's Toxicologic Emergencies,* 7th ed. New York, NY: McGraw-Hill; 2002:44–57.

Tanen DA. Organophosphorus and carbamate insecticides. In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:292–296.

Torpy JM, Lynn C, Glass RM. JAMA Patient Page: malignant hyperthermia. *JAMA.* 2005;293(23):2958.

Wythe ET. Radiation (ionizing). In: Olson KR, ed. *Poisoning and Drug Overdose,* 5th ed. New York, NY: Lange Medical Books/McGraw-Hill; 2007:328–333.

Obstetric and Gynecologic Emergencies

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Objectives

1. Discuss the anatomy of the female reproductive system (p 842).
2. Recognize the physiologic changes during pregnancy (p 844).
3. Describe the changes that occur in the cardiovascular, respiratory, gastrointestinal, renal, and endocrine systems (p 844–846).
4. Discuss dermatologic changes that occur during pregnancy (p 846).
5. Discuss special areas of concern when performing a critical care transport of a pregnant patient (p 846).
6. Describe the management of the pregnant patient who is in cardiac arrest (p 847).
7. Discuss potential maternal cardiovascular complications exacerbated or induced by pregnancy (p 847).
8. Discuss fetal oxygenation and heart rate, including conditions associated with fetal distress during labor (p 847).
9. Discuss how to assess a fetus during a critical care transport (p 847).
10. Describe several methods of fetal monitoring during critical care transport of a pregnant patient, including electronic fetal monitoring (p 848).
11. Explain how to use a Doppler device (p 847–848).
12. Define the complications of pregnancy, including spontaneous abortion and causes of bleeding (p 849–851).
13. Recognize and treat abruptio placenta, placenta previa, and uterine rupture (p 851–853).
14. Discuss medical conditions that can exist during pregnancy, including pregnancy-induced hypertension, preeclampsia, eclampsia, and HELLP syndrome, and how to manage them during critical care transport (p 853–861).
15. Discuss medications used in treating pregnancy-induced hypertension, including labetalol and hydralizine (p 853).
16. Understand the concerns regarding preterm labor and premature delivery (p 855–858).
17. Discuss the use of tocolytic agents to interrupt labor (p 857–858).
18. Recognize fetal malpresentations of delivery, including frank breech, complete breech, incomplete breech, footling breech, and umbilical cord prolapse (p 858–860).
19. Discuss how to manage fetal malpresentations during a critical care transport, including frank breech, complete breech, incomplete breech, footling breech, and umbilical cord prolapse (p 859–

860).

20. Describe shoulder dystocia and how to manage this complication during a critical care transport (p 861).
21. Discuss multiple-birth deliveries and how to manage these during a critical care transport (p 862).
22. Briefly discuss postpartum care of the mother and infant during a critical care transport (p 862).
23. Describe potential postpartum complications and their management during critical care transport, including amniotic fluid embolism and postpartum hemorrhage (p 862–864).
24. Recognize and discuss how to manage gynecologic issues and emergencies during critical care transport, including ectopic pregnancy, pelvic inflammatory disease, toxic shock syndrome, ovarian cysts, pathologic cysts, ovarian torsion, and gynecologic trauma including vulvular hematoma and sexual assault (p 864–867).
25. Discuss flight considerations that pertain to a critical care transport of a pregnant patient or a patient with a gynecologic emergency (p 867).

Introduction

CCTPs face many challenges when treating female patients, especially if the patients are pregnant. Patient management is difficult when there are competing concerns for the woman and the fetus, physiologic changes induced by pregnancy, and the presence of potential life-threatening pathologic conditions. This chapter provides an overview of the physiologic changes in pregnancy and describes specific emergency conditions associated with pregnancy and postpartum care. Knowledge of the anatomic and physiologic changes that occur during pregnancy is essential to provide effective care to obstetric patients.

Controversies

A common concern when faced with a critically ill or injured pregnant woman is whether the mother or fetus takes priority. In all cases, the priority is resuscitation and stabilization of the mother. The key to a viable fetus, even when a decision is made to expedite delivery, is restoration of adequate perfusion in the mother.

Anatomy and Physiology of the Female Reproductive System

The female reproductive system includes a 28-day cycle that is regulated by a series of hormonal secretions. These hormones prepare the uterus for implantation of a fertilized egg and the start of pregnancy. In the absence of pregnancy, the uterine wall sheds its inner lining, known as the **endometrium**. The mucosal tissues and blood are discharged through the vagina, which is a process known as menstruation. Menstruation lasts approximately 3 to 5 days.

Figure 21-1 depicts the menstrual cycle. At the beginning of the menstrual cycle, there is a rise in the levels of the follicle-stimulating hormone (FSH), which triggers the start of the preovulatory phase, or follicular phase. During the next 7 to 8 days, the uterine wall begins to swell, or proliferate, and the process of creating an ovum begins with follicle stimulation in the ovaries. FSH stimulates the creation of an oocyte in the ovary. As the oocyte begins to mature, estrogen is secreted by the ovary, causing the endometrium to swell with blood. As the follicle matures, the levels of estrogen cause the anterior pituitary to secrete the luteinizing hormone (LH). Introduction of this hormone causes the release of the

oocyte, which is called an ovum now that it is mature, from the ovaries and the start of ovulation. For the next 24 to 48 hours, as the newly formed ovum travels down the fallopian tube to the uterus, the fertility period is at its peak. A hard shell known as the corpus luteum, which begins to secrete more estrogen, testosterone, and progesterone, is left behind in the ovary. As progesterone levels rise over the next 14 days, the endometrium continues to swell and provide a soft shelter for the egg. If there is no conception, the unfertilized ovum travels into the uterus. Progesterone levels drop off as the corpus luteum begins to dissolve in the ovary and menstruation begins, thus ending the cycle.

Conception and Gestation

At this point in the process, if fertilization has occurred and the egg, known as the zygote, implants in the endometrium, blastocytes inside the zygote secrete human chorionic gonadotropin (hCG), which extends the life of the corpus luteum, and progesterone continues to be secreted. The relatively inelastic placenta, which is attached to the wall of the uterus, supplies the fetus with oxygen and vital nutrients and removes waste products such as carbon dioxide. In addition, the placenta continues to secrete the progesterone needed to maintain a swollen endometrium and continue the pregnancy. The placenta and fetus are connected by the umbilical cord, which contains two arteries and one vein that carry the blood of the fetus to and from the placenta **Figure 21-2**.

The placenta is an ephemial, or transitory, organ that is formed early by the implantation of the blastocyst into the endometrium. The placenta grows with the fetus; it contains a thin membrane that separates maternal and fetal blood from coming into contact with one another. Nutrients, oxygen, and carbon dioxide diffuse across this membrane to nourish the placenta. It is important to understand that maternal blood is never shared with fetal blood; a fetal barrier prevents maternal blood from entering fetal circulation, although substances from the blood, such as nutrients, do transfer. CCTPs need to be aware of the medications that are allowed to be given to a pregnant patient, as well as those that should be avoided. The Food and Drug Administration classifies medications as follows: Class B medications are probably safe for the fetus; Class C medications should be used only if the benefit outweighs the risk; Class D medications have known risks to the fetus, but are used in some situations; and Class X medications are known teratogens and should not be used under the circumstance.

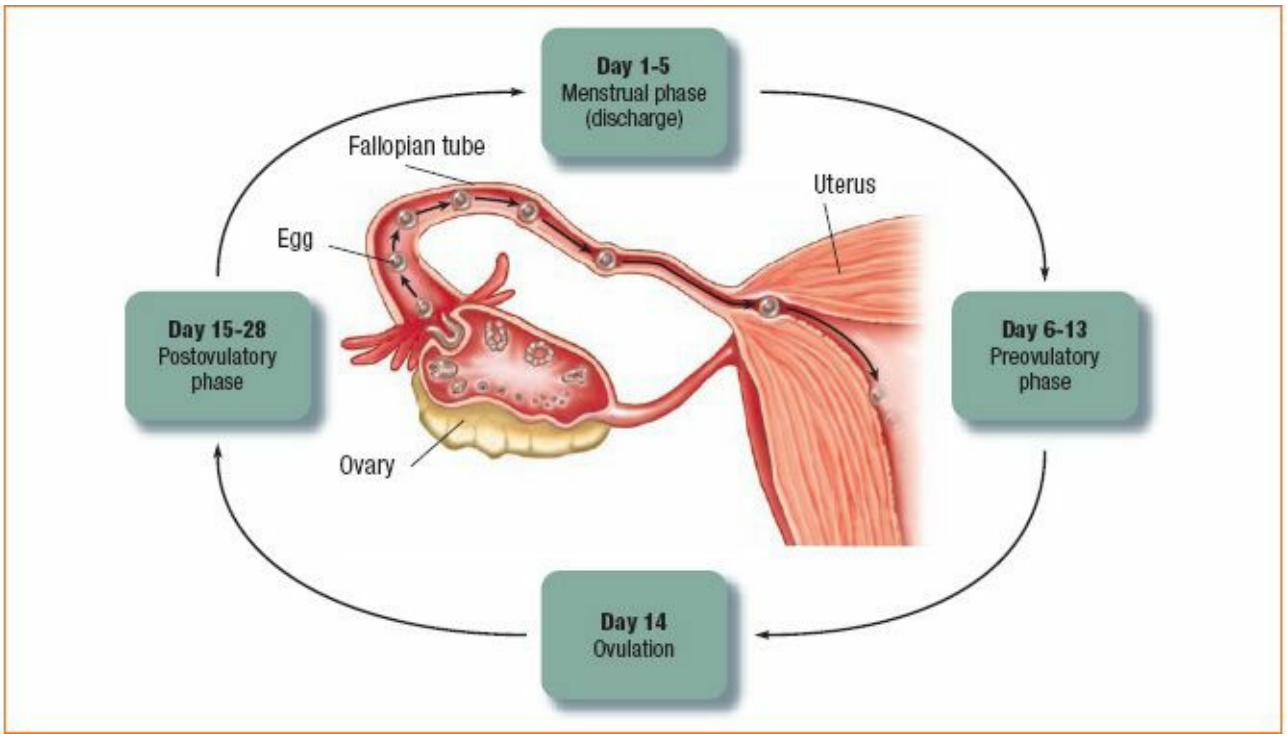


Figure 21-1 The menstrual cycle, based on an average 28-day cycle. The length of the cycle and number

of days in each phase vary from woman to woman, but generally fall within a range of 24 to 35 days.

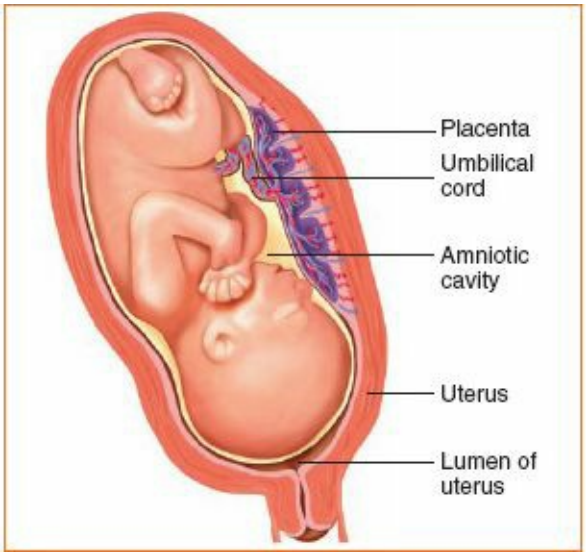


Figure 21-2 The umbilical cord and other structures of the uterus in a pregnant woman.

Early in pregnancy, the uterus is contained entirely within the protective ring of the pelvis. By the 12th week of **gestation**, the enlarged uterus begins to rise out of the pelvis and into the abdomen. By 20 weeks of gestation, the uterus is at the umbilicus, and by 34 to 36 weeks of gestation it reaches the costal margin. This expansion, from approximately 70 to 1,100 g at **term** (38 to 42 weeks' gestation), is due to marked stretching and hypertrophy of existing muscle cells, rather than generation of new cells. As the uterus enlarges, the walls become thin and more fragile. The bowel is pushed cephalad and is somewhat more protected from blunt injury to the abdomen. However, as the uterus rises out of the pelvis, it loses the bony protection provided by the pelvis and becomes more vulnerable to trauma. Also, as the fetus grows, the relative proportion of amniotic fluid to fetal size decreases, making the fetus more vulnerable to trauma. During the last 2 weeks of the pregnancy, the fetal head begins to drop lower into the pelvis in preparation for delivery. As a result, the fundal height, or distance from the pelvic ring to the top of the uterus, decreases by about 2 cm **Figure 21-3**. (To measure the fundal height, use a tape measure from pelvis to epigastrium. The MacDonald's Rule states that the length in centimeters is proportionate to the number of weeks of gestation. For example, a woman who is 15 weeks pregnant would have a fundal height of about 15 cm, give or take 1 to 3 cm. This method is valid from the 12th week until the 38th week of gestation.)

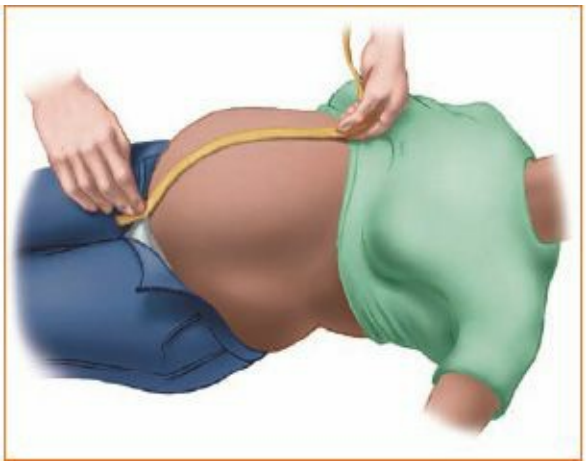


Figure 21-3 Measuring fundal height.

Physiologic Changes During Pregnancy

Pregnancy alters the normal physiology of females, affecting nearly every organ system. These changes may mask well-recognized signs or symptoms of disease, or normal changes may even masquerade as pathologic conditions. A functional knowledge of these alterations is essential to manage a pregnant patient effectively.

Cardiovascular System

Normal cardiac output at rest is approximately 5 L/min, but during pregnancy the cardiac output increases significantly. During the first 10 weeks of pregnancy, the cardiac output increases 20% to 30%, or 1 to 1.5 L/min, and by gestational term the cardiac output could be as high as 43% greater than the cardiac output in a patient who is not pregnant. This increase in the cardiac output is primarily due to an increase in plasma volume and a decrease in vascular resistance resulting from dilation of the uterine arteries. The combination of a greater plasma volume and dilated uterine arteries results in an increase in blood flow to the uterus. At term, the blood flow through the uterine arteries reaches 500 to 700 mL/min. The pregnant patient's pulse rate increases by 10 to 15 beats/min, reaching a maximum in the third trimester of pregnancy (the last 12 weeks [approximately the last third] of pregnancy). This increase in the baseline pulse rate must be considered when you are interpreting tachycardia in response to hypovolemia. The stress on the cardiovascular system is intensified by activation of the renin-angiotensin system during the third trimester of pregnancy, followed by an increase of insulin resistance resulting in high plasma glucose levels. Because these factors can result in gestational hypertension, risk factors that CCTPs must remain aware of include the following:

- Chronic hypertension
- Type 1 and type 2 diabetes
- History of smoking and cocaine use
- Family history of cardiovascular disease and hypertension

Also, the elevated position of the pregnant patient's diaphragm will cause a 2- to 3-cm heart displacement and axial rotation, which can result in a third heart sound and 12-lead abnormalities, such as right or left axis deviation.

Blood Volume and Composition

One of the most significant changes in pregnancy is the marked increase in total plasma volume. Circulating blood volume expands by an average of 40% to 45% as a result of an increase in both plasma volume and the number of erythrocytes. The level of increase depends on such factors as patient size, number of pregnancies **(gravida** [total number of pregnancies], and **parity** [number of pregnancies carried to term of more than 28 weeks' gestation and delivered]), and number of fetuses. The increase in plasma volume is greater than the increase in the level of erythrocytes, giving the appearance of anemia. The hematocrit value decreases, reaching its lowest point between the 32nd and 34th weeks of gestation. This point is referred to as the *physiologic anemia of pregnancy*. The average hemoglobin level in late pregnancy is between 10.5 and 11.0 g/dL, and the average hematocrit value is between 31% and 35%. The leukocyte count is generally elevated during pregnancy and may reach 25,000/µL during labor. However, beginning in the second trimester of pregnancy, leukocyte function is usually depressed and increased susceptibility to infection may occur. The platelet count during pregnancy is relatively stable and therefore **thrombocytopenia** is not usually seen.

Other changes in blood components during pregnancy include a decrease in the level of serum albumin and a lowered total serum protein. Many clotting factors are elevated during pregnancy.

Increased coagulation factors shorten the **prothrombin time (PT)** and **partial thromboplastin time (PTT)**. Fibrinogen levels increase dramatically during pregnancy, nearly doubling by delivery. A pregnant patient is more susceptible to lower extremity deep venous thrombus and pulmonary embolus.

Blood Pressure

At the onset of pregnancy, maternal blood volume increases as much as 30%. During mid-pregnancy, blood pressure often decreases as a result of progesterone, which relaxes the walls around the blood vessels. Lower blood pressures and other clinical signs such as increased jugular vein distention and bi-basilar crackles that can lead CCTPs to believe that the patient is in right-sided heart failure. Treatment should be focused on airway management and oxygen therapy with an understanding that the clinical signs and symptoms are more physiologic than pathologic.

As pregnancy progresses, blood pressure decreases, reaching its lowest point in the second trimester of pregnancy. A lower peripheral vascular resistance allows the diastolic blood pressure to decrease by 10 to 15 mm Hg, whereas the systolic blood pressure normally decreases by 5 to 10 mm Hg. Blood pressure returns to near pre-pregnancy levels at gestational term. During the third trimester of pregnancy, the body position of the pregnant patient may significantly affect her blood pressure and cardiac output. When she is lying supine, the uterus may compress the inferior vena cava, reducing venous return from the lower extremities, causing hypotension. This phenomenon is referred to as **supine hypotensive syndrome**. Displacement of the gravid uterus off of the vena cava by manual manipulation or by placement of the patient in the left lateral position relieves this obstruction and thus restores blood pressure.

For patients with potential spinal injuries, it is important to tilt the backboard to the left to relieve the pressure from the inferior vena cava. This position is accomplished by placing blankets or towels under the right side of the backboard **Figure 21-4**. Note that these materials are placed under the backboard, not directly under the patient.

Venous Pressure

The peripheral venous pressure in the lower extremities also rises progressively during the later stages of the pregnancy due to the compression of the inferior vena cava and pelvic veins by the uterus, which can lead to more bleeding than expected from minor wounds to the lower extremities. CCTPs must be cautious of excessive internal bleeding with minimal clinical signs and symptoms. Pregnant patients involved in blunt trauma must be considered to have internal bleeding even when they are hemodynamically stable.

Figure 21-4 Place pregnant patients with potential spinal injuries on a backboard with blankets and towels under the right side of the backboard to tilt it toward the left and relieve pressure from the inferior vena cava.

TABLE 21-1 Cardiovascular Changes in Normal Pregnancy

	Nonpregnant Patient	Pregnant Patient
Cardiac output	5,000 mL	6,000 mL (20%–40% increase)
Stroke volume	80 mL	110 mL (approximately 30% increase)
Pulse rate	80 beats/min	10%–20% increase
Red blood cell mass	5 million cu mm	33% increase
Systemic vascular resistance	1,500 dyne-sec/cm^{-5}	20% decrease
Mean arterial pressure	85 mm Hg	No change
Systolic blood pressure	120 mm Hg	120 mm Hg
Diastolic blood pressure	70 mm Hg	80 mm Hg

Adapted from: Sodré PM. Maternal physiology changes during pregnancy. *Obstet Gynecol*. Available at: http://www.medstudents.com.br/ginob/ginob5.htm. Accessed February 12, 2009.

Special Populations

The extreme physiologic and hemodynamic changes associated with pregnancy illustrated in this chapter highlight potential consequences resulting from preexisting conditions in the pregnant patient. It is imperative that the CCTP aggressively manage blood pressure, blood glucose, and conditions affecting perfusion in all pregnant patients with cardiac, renal, vascular, or neurologic conditions that may be exacerbated by the changes seen during pregnancy. Failure to do so can result in catastrophic consequences for the mother and infant.

These changes in the cardiovascular system can have a profound effect on any underlying medical condition. CCTPs may be called to transfer a pregnant patient with cardiovascular compromise.

Table 21-1 summarizes cardiovascular system changes during pregnancy.

Respiratory System

During pregnancy, minute ventilation increases due to an increase in tidal volume. The increase in tidal volume is thought to be secondary to increased levels of progesterone. This increased ventilation leads to a lower pCO_2 level of approximately 30 mm Hg (normal, 40 mm Hg). The respiratory rate increases slightly. The body tries to compensate for the increase in pH by creating a state of metabolic acidosis, which results only in a very small change. Arterial blood gas levels tend to be more alkalotic, with the average pH ranging from 7.40 to 7.47.

The increased oxygen demand of the growing fetus results in a significantly reduced oxygen reserve for the patient. Therefore, desaturation occurs more quickly in pregnant patients than in nonpregnant patients. Accessory muscle usage is also difficult in pregnant patients in respiratory distress because the diaphragm is displaced and the abdominal muscles are weakened, resulting in ineffective tripoding. CCTPs must be aware that pregnant patients in respiratory arrest become anoxic faster than nonpregnant patients. Early oxygenation and aggressive airway management with intubation should be primary considerations. Laryngoedema should be expected, with an average Mallampati score of 3 (discussed in

detail in Chapter 6). Consider use of alternate airways if unable to intubate; a bag mask alone will increase the already high risk of aspiration.

Gastrointestinal System

During pregnancy, gastric emptying is delayed and the gastroesophageal sphincter tone is reduced, making gastroesophageal reflux common. These factors combine to increase the likelihood of aspiration during pregnancy when an altered level of consciousness is present. The diaphragm rises an extra 4 cm during pregnancy as a result of the enlarging uterus and cephalad displacement of abdominal contents. The stress placed on the gastrointestinal system results in chronic nausea, vomiting, and, in severe cases, dehydration. Administration of IV fluids and oxygen and patient positioning (position of comfort or on the left side) can help lessen these effects during transport. If a pregnant patient requires placement of a chest tube, a more cephalad location for placement should be considered. The intestines are relocated to the upper part of the abdomen and therefore may be shielded by the uterus from blunt trauma, whereas the liver and spleen remain in their normal location under the diaphragm, but are pushed cephalad somewhat in late pregnancy. In addition, CCTPs must consider intubation early in the pregnant patient because of the higher probability of aspiration.

Renal and Endocrine Systems

During pregnancy, the renal system is stressed; there is a 30% increase in the glomerular filtration rate (GFR) along with **hydroureteronephrosis**. or ureter dilation and obstruction. These conditions lead to frequent urination with hematuria and flank pain. Relief can occur by placing the patient in the left lateral recumbent position. Renal calculi are no more common in pregnant patients than nonpregnant patients, and treatment should remain the same by observation and allowing the stone to pass. Urinary tract infections are also no more frequent in pregnant patients; however, early trimester infections that are left untreated can lead to pyelonephritis in the third trimester of pregnancy about 40% of the time. Patients with chronic renal failure (CRF) can undergo dialysis treatment without any risk to the fetus. Patients with acute renal failure will have issues with compensation of the respiratory alkalosis discussed earlier, so CCTPs must be aware of changes in pH levels and treat them accordingly.

At the beginning of conception, the endocrine system goes into overdrive. The pituitary and thyroid glands enlarge, resulting in the increased production of estrogen, progesterone, cortisol, and thyroxine. These hormones play an important role in fetal development and in preventing the fetus from aborting; however, in some cases, these elevated hormone levels alter the effectiveness of insulin and can result in a condition known as insulin resistance. After 20 weeks' gestation, insulin resistance can lead to elevated blood glucose levels and a condition known as gestational diabetes.

Thyroid conditions (hyperthyroidism or hypothyroidism) may affect fetal development. These conditions therefore should be monitored carefully during prenatal care. CCTPs should expect complications during delivery and a possible distressed neonate if a patient who has a thyroid disorder has had no prenatal care.

Dermatologic Changes

Hormonal changes have an impact on the largest organ of the body—the skin. In addition to acne, pregnant patients can experience linea negra, a darkened line that develops from the navel to the vagina, and chloasma, which causes darkened skin patches to develop on the cheeks and forehead. Finally, some late-term patients present with an itchy rash known as pruritic urticarial papules and plaques of pregnancy (PUPPP) that can be isolated to one region or systemic. Patients can be prescribed topical corticosteroid ointments to treat this condition, but CCTPs must be aware that this can be a life-threatening condition in

patients who have hypothyroidism. Patients will present in anaphylaxis and must be treated with aggressive airway management and beta-agonists.

Critical Care Transport of the Pregnant Patient

The role of the CCTP in any critical care transport is to safely and effectively manage, treat, and transport a patient to the hospital that can best meet the needs of the patient. The pregnant patient's dynamic physiologic changes and the developing fetus can pose a challenge for any hospital, especially those without obstetric services. In addition, pregnancy can lead to exacerbation of chronic diseases such as coronary heart disease, mitral valve prolapse, and aortic stenosis. Treatment needs to focus on maintaining the pregnant woman's hemodynamics. CCTPs need to remember that taking care of the pregnant woman means that the fetus is being taken care of as well. In critical cases, treatment of the pregnant patient is no different than that of the nonpregnant patient; for example, treatment of a pregnant patient with an existing cardiovascular condition does not differ from treatment of a nonpregnant patient with this condition.

The CCTP must perform a complete physical exam, including maternal and fetal hemodynamic monitoring and, if the patient is in labor, external vaginal evaluation for the presence of crowning. If delivery is imminent, the CCTP should assist in the delivery. If you arrive at the initial facility and find that delivery is imminent, delivery should be assisted at that facility; transport should not begin when delivery is imminent.

Transport Management

Cardiac Arrest in the Pregnant Patient

- Administer CPR.
- Provide early oxygenation.
- Place the pregnant woman in the Cardiff Wedge position.
- Defibrillate patients in ventricular fibrillation (but remove fetal monitors).
- Administer resuscitation medications.

Cardiac Arrest Management of the Pregnant Patient

CCTPs need to remember that the pregnant woman's health is the treatment priority. By appropriately treating the pregnant patient, the fetus also benefits. The standard rules of CPR apply, with chest compressions and limited interruptions for ventilation. Because pregnant patients become hypoxic faster, early oxygenation will improve overall outcomes. A late-term fetus can cause circulatory problems, so the following technique can be used. Tilt the patient 15° to 30° to the left or shift the fundus to the left to allow for effective venous return and maximal circulation. If a Cardiff wedge is available, this device is useful in achieving the technique. Defibrillation remains essential for patients in ventricular fibrillation and is safe for the fetus, but fetal monitors (discussed later in this chapter) must be disconnected prior to delivering a shock.

Again, pregnant patients diagnosed with myocardial infarction can be treated with the same medications and procedures as nonpregnant patients; however, delivery will be delayed to reduce stress on the healing myocardium.

Fetal Assessment and Monitoring

Several methods can be used to assess and monitor the fetus. Fetal circulation, oxygenation, and heart rate will be reviewed first.

Fetal Circulation, Oxygenation, and Heart Rate

Fetal circulation occurs through the umbilical vein and umbilical arteries. The umbilical vein carries oxygenated blood from the placenta to the fetus' right atrium via the ductus venosus and the inferior vena cava, and the umbilical arteries carry arteriovenous blood to the placenta. Because the fetus obtains its oxygen via the placenta, the fetal circulation bypasses the lungs until birth.

The oxygen content of the fetus is lower than that of a pregnant woman, even though the rate of oxygen consumption by the fetus is nearly twice that of a pregnant woman per unit weight. The developing fetus obtains its oxygen from the blood of a pregnant woman through the placenta and has only a small reserve of oxygen, usually only 1 to 2 minutes if the supply from the woman is cut off or diminished. During uterine contractions, blood flow through the uterine arteries to the placenta is momentarily interrupted. A healthy fetus with a normal placenta can withstand the stress of labor without having hypoxia develop because there is sufficient oxygen exchange during the interval between contractions.

TABLE 21-2 Conditions Associated With Fetal Distress During Labor

Source	Condition
Umbilical cord	Hematoma Knot in cord Nuchal cord Prolapsed cord Cord compression
Placenta	Infarction Abruption
Uterus	Tetanic contractions Hyperstimulation
Fetus	Anemia Infection
Maternal	Hypertension Hypotension Severe anemia Seizures

The fetal heart is sensitive to the changes in oxygen supply. Monitoring the fetal heart rate gives information concerning the oxygenation status of the fetus. If the heart of the fetus does not receive enough oxygen, the rate will decrease.

Hypoxia may be the result of many causes because fetal oxygenation can be impaired at different anatomic locations within the uteroplacental-fetal circulatory loop. For example, an infarction of the placenta (death of the placental tissue and formation of scar tissue in its place, usually only problematic in extreme circumstances) or a placental abruption (discussed later in this chapter) may lead to impaired

diffusion of oxygen from the maternal circulation to the fetal circulation. **Table 21-2** summarizes the conditions associated with fetal distress during labor.

Fetal Assessment

The true challenge that the CCTP has when transporting a critical pregnant patient is assessing the second patient, the fetus. All patient assessments start with the ABCs. Assessing the ABCs is extremely challenging in the fetus, but this does not mean that assessment and monitoring cannot be done effectively. Prior to transporting a pregnant patient, a fetal assessment needs to be performed.

A pregnant woman should observe a minimum of 10 kicks every 2 hours. As a first step, ask the woman about fetal movement and whether this has occurred. No movement indicates fetal distress and transport should be a priority. Next, auscultate the fundus to detect a heartbeat and any movement not initially felt **Figure 21-5**. It is not recommended to use a normal stethoscope, especially in early term patients; CCTPs should use a Doppler machine starting at 10 weeks' gestation or a fetal stethoscope (Fetoscope) **Figure 21-6** after 20 weeks to detect a heartbeat. The fetus' back is the optimal location to hear the heartbeat; feel for the most rigid part of the fundus to detect the proper location for auscultation. When a hand-held Doppler machine is used, apply conductive gel to the pregnant patient's abdomen at this location to aid in the ultrasound transducer and listen for the heartbeat. To confirm that you are listening to the fetal heart beat, take the pregnant patient's radial pulse and compare the rates; the fetal heart rate should be faster.

Figure 21-5 Checking for fetal responsiveness. After asking the woman about fetal movement, use a Doppler machine or fetal stethoscope, depending on the length of gestation, to feel for the most rigid part of the fundus (the fetus' back) and auscultate that part to detect the heartbeat and any movement not initially felt.

Figure 21-6 A fetal stethoscope (Fetoscope).

The normal heart rate of a fetus is between 120 and 160 beats/min **Table 21-3**. If a patient is in labor, monitor the fetal heart tones for at least 30 seconds every 15 minutes and record the results. The presence of fetal heart tones indicates life, and the absence of fetal heart tones can indicate death. In a noisy environment, however, auscultation of fetal heart tones can be challenging, and it is difficult to determine how the heart rate is changing over time. For this reason, continuous electronic fetal monitoring should be performed during high-risk transports. Continuous electronic fetal monitoring is discussed later in this chapter.

Electronic Fetal Monitoring

Electronic fetal monitoring can be done externally or internally, but during critical care transports it is usually only done externally. **External fetal monitoring** uses an ultrasonic transducer externally applied to the pregnant patient's abdomen **Figure 21-7**. The ultrasonic transducer monitors the fetal heart rate, and a strap around the patient's abdomen monitors uterine contractions. Both of these variables are simultaneously recorded onto a paper chart.

Electronic fetal monitoring is recommended for all high-risk interfacility transports. Monitoring enables continuous reporting of the fetal heart rate and uterine contractions by a 2-channel strip chart recorder that prints the results. The uterine contraction represents a stress on the fetus **Figure 21-8**. The alteration in fetal heart rate correlates with fetal oxygenation.

TABLE 21-3 Heart Rate Parameters

Rate	Beats/min
Normal	120–160
Abnormal • Tachycardia • Bradycardia	< 100 > 160 < 120

Figure 21-7 External fetal monitoring.

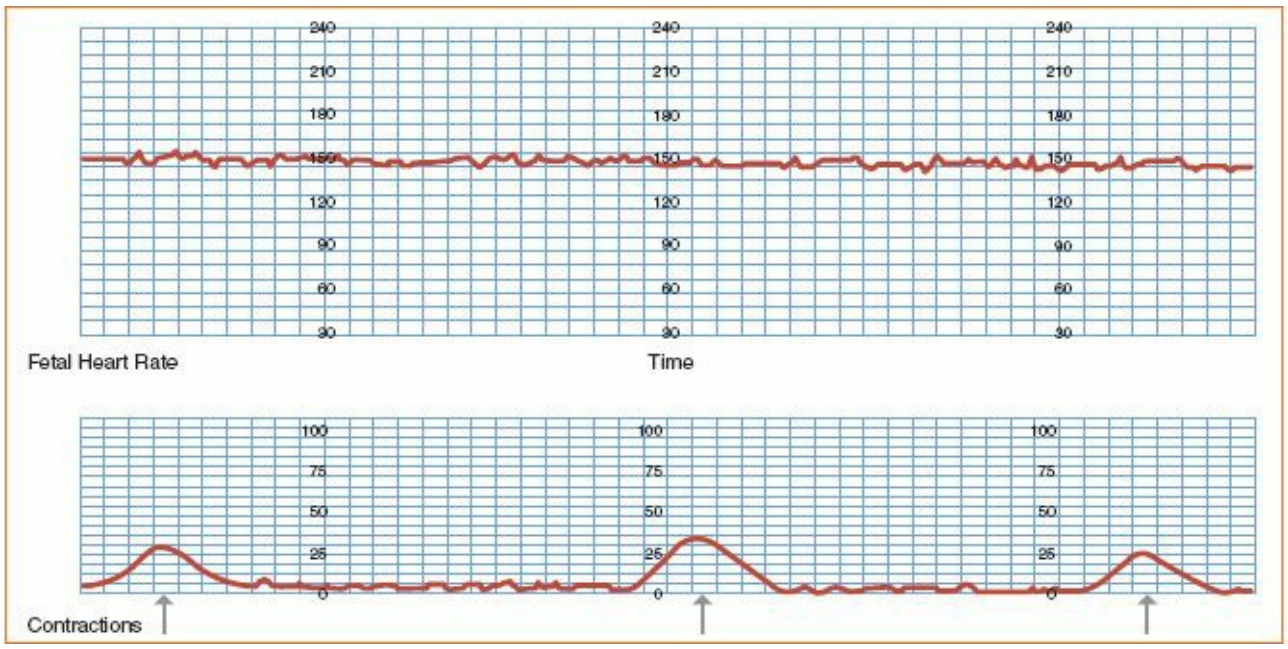


Figure 21-8 A fetal monitoring strip showing uterine contractions.

Equipment

The equipment needed in order to perform external fetal monitoring includes:

- Fetal monitor
- Conducting gel
- Tracing paper
- Ultrasonic Doppler
- External uterine activity Doppler

Steps

Skill Drill 21-1 shows the steps for performing fetal monitoring, which are discussed as follows:

1. Prepare all equipment prior to transport. Introduce yourself and explain the procedure to the patient **Step 1**. Be sure to clarify that this procedure is normal for all transports and that it is essential to monitor the baby's health during the trip.
2. Connect to the power source **Step 2**.

3. Connect the Doppler probe to the patient and adjust the volume **Step 3**.
4. Note that the fetal heart rate should be displayed in green.
5. Make sure that the *Auto Non-Stress Test* switch is in the off position **Step 4**.
6. Manually set the uterine Doppler to zero. Wait for the alerting sound.
7. Press the recording switch.
8. Set the fetal heart tone alarms.

Assessment of fetal monitoring strips rarely occurs during transport, though you may obtain a strip during transport if possible and needed.

Complications of Pregnancy

Spontaneous Abortion

Spontaneous abortion (miscarriage) is the loss of a fertilized developing ovum. Spontaneous abortion is common in early pregnancy with about 30% of all pregnancies ending prior to 20 weeks of gestation. Loss of a fetus after 20 weeks is called intrauterine fetal demise. About 80% of spontaneous abortions occur during the first trimester.

Spontaneous abortions almost always arise from some abnormality in the woman or the fertilized ovum and, in the majority of cases, fetal death occurs before any sign or symptom of the spontaneous abortion. Patients often present with vaginal bleeding or cramping. Assessment of the female patient with vaginal bleeding or abdominal pain focuses on identifying whether the patient is having a spontaneous abortion or whether the symptoms, such as shock, indicate an ectopic pregnancy. With a spontaneous abortion, blood loss is rarely large enough to cause hemodynamic instability. Hypotension should be addressed with volume replacement with isotonic fluids such as lactated Ringer's solution or normal saline. Treatment for women having a spontaneous abortion includes providing comfort and emotional support. Some patients who have a spontaneous abortion may not completely expel all of the products of conception and therefore may require a **dilation and curettage (D&C)**. In this surgical procedure, the physician manually dilates the cervix and scrapes the uterine lining to remove the remaining conception material. Patients having a spontaneous abortion need a D&C procedure to reduce the risk of infection caused by tissue left behind on the uterine wall. The patient should be transported to a facility where this procedure can be performed within 24 hours. CCTPs should maintain documentation of the patient's blood loss by recording the number of sanitary pads that are soaked during transport and relay this information to the receiving facility. Vaginal bleeding that soaks more than one pad every hour should be cause for concern. As with any invasive procedure, there is a risk for infection. CCTPs need to watch for the signs of postoperative infection, including fever, chills, tachypnea, and lethargy.

Signs and Symptoms

Spontaneous Abortion

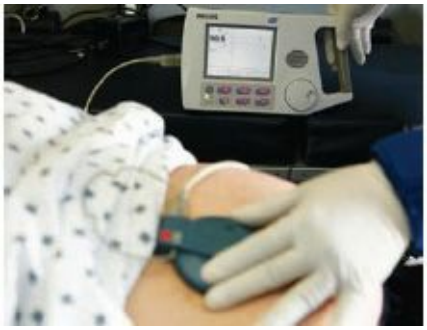
- Vaginal bleeding
- Cramping
- Abdominal pain
- Hypotension

Skill Drill 21-1

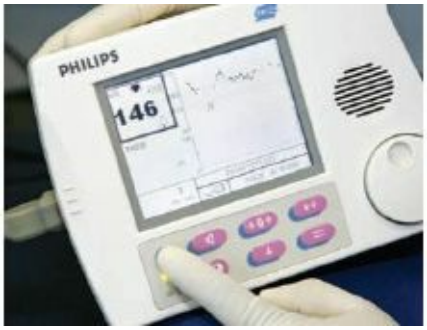
Fetal Monitoring

1 Prepare all equipment prior to transport. Explain the procedure to the patient.

2 Connect to the power source.



3 Connect the Doppler probe to the patient and adjust the volume. The fetal heart rate should be displayed in green.



4 Make sure that the *Auto Non-Stress Test* switch is in the off position. Manually set the uterine Doppler to zero. Wait for the alerting sound. Press the recording switch. Set the fetal heart tone alarms.

Differential Diagnosis

Spontaneous Abortion

- Abruptio placenta
- Ectopic pregnancy
- Placenta previa

Transport Management

Spontaneous Abortion

- Provide volume replacement with isotonic fluids.
- Provide comfort and emotional support.
- Place sanitary pads.

Bleeding

Vaginal bleeding in the third trimester of pregnancy is often associated with significant pathologic conditions that may endanger the life of the fetus and woman. In one third of cases of vaginal bleeding after 20 weeks of gestation, the fetus will die.

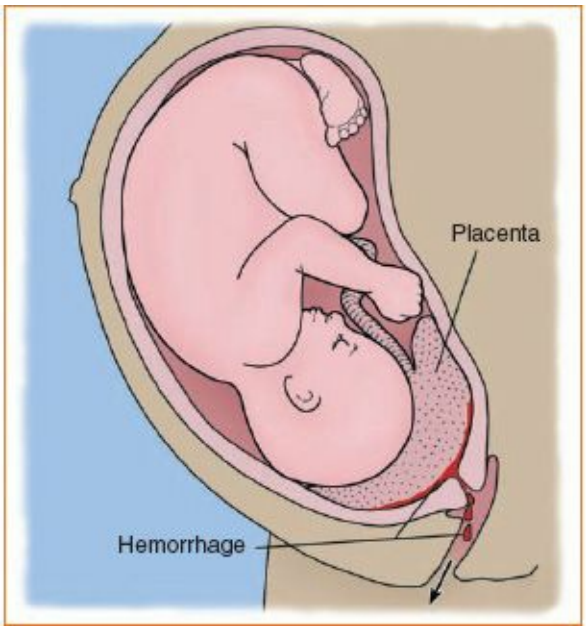


Figure 21-9 Placement of the placenta in abruptio placenta.

As the pregnant woman approaches term, there is an increased possibility of life-threatening hemorrhage associated with vaginal bleeding. Three major causes of life-threatening bleeding are abruptio placenta, placenta previa, and uterine rupture. Because of the high risk of maternal and fetal death associated with any of these clinical entities, any vaginal bleeding during the third trimester of pregnancy must be regarded as a dire medical emergency until proven otherwise. As a general rule, labor is not associated with significant vaginal bleeding. Therefore, you should suspect placenta previa or

abruptio placenta in any patient with third-trimester abdominal pain or vaginal bleeding.

Regardless of the cause of the vaginal bleeding, patient management is the same. The woman should be placed on her left side in a recumbent position to maximize blood return to the heart by preventing supine hypotensive syndrome. High-flow oxygen should be administered along with at least one large-bore IV Lactated Ringer's solution is the optimal treatment, but treatment with 0.9% normal saline as an alternate can be given in 250-mL boluses and titrated to the patient's hemodynamic status. Blood pressure should be maintained at no less than 90 mm Hg systolic. Rapid transport is required, with the receiving hospital notified of the nature of the problem as well as any changes in the condition of the patient en route. ***Under no circumstances should an internal vaginal exam be performed by the CCTP.***

Abruptio Placenta

Abruptio placenta is the premature separation of a normally implanted placenta from the uterine wall **Figure 21-9**. This condition accounts for 30% of vaginal bleeding in the second half of pregnancy. Abruptio placenta can be either spontaneous or a result of trauma.

Signs and Symptoms

Abruptio Placenta

- Vaginal bleeding
- Abdominal pain
- Back pain
- Uterine tenderness
- Signs of shock
- Lack of fetal heart sounds

Differential Diagnosis

Abruptio Placenta

- Ectopic pregnancy
- Placenta previa
- Preterm labor
- Spontaneous abortion

Transport Management

Abruptio Placenta

- Place patient on her left side, in recumbent position.
- Provide bleeding control.
- Provide oxygenation.
- Administer IV fluids.
- Provide rapid transport.

Spontaneous abruptio placenta is primarily associated with maternal hypertension but is also associated with increased maternal age, multiparity, smoking, cocaine use, and previous history of the condition. Bleeding from spontaneous abruptio placenta usually occurs late in the third trimester of pregnancy as the cervix begins to dilate in preparation for delivery.

Abruptio placenta associated with trauma is less common and is usually due to direct trauma to the abdomen. However, it is a complication in 1% to 5% of minor injuries that occur during pregnancy and up to 40% to 50% of major trauma injuries that occur during pregnancy. Because the placenta is less elastic than the uterus to which it is attached, when a pregnant woman is involved in trauma in which the uterus is stretched or deformed, the relatively inelastic placenta separates from the uterine wall, causing abruptio placenta.

Regardless of the cause, the presenting signs and symptoms of abruptio placenta include vaginal bleeding, abdominal pain, back pain, and uterine tenderness. Abruptio placenta can be concealed, however. The separation may occur in such a way that the bleeding is contained by the part of the placenta attached to the uterine wall. The amount of vaginal bleeding may be small or no blood is evident, and the diagnosis may be easily mistaken for premature labor. However, shock may ensue from the concealed blood loss.

On physical exam, the abdomen is usually tender and the uterus is rigid on palpation. When vaginal blood is present, it is usually dark red. Fetal heart sounds are often absent, because the fetus is either partly or completely cut off from its blood supply. The pregnant patient may exhibit signs of shock that may be out of proportion to the amount of vaginal bleeding.

Important factors in the management of the pregnant patient with suspected abruptio placenta are fetal status and the presence or absence of labor. Most women who experience abruptio placenta spontaneously go into labor. The same procedures for all vaginal bleeding in the third trimester of pregnancy should be followed, with a focus on bleeding control, oxygenation, and IV fluids to maintain hemodynamic stability. Again, those procedures are to place the patient on her left side in a recumbent position, administer high-flow oxygen, administer at least one large-bore IV of lactated Ringer's solution or normal saline, and provide rapid transport. Ultrasound should be used when a pregnant woman has experienced blunt force trauma in order to determine if the fetus has life-threatening injuries.

Placenta Previa

In placenta previa, the placenta is implanted low in the uterus and partially or completely covers the cervical canal **Figure 21-10**. Placenta previa accounts for 20% of bleeding episodes after 20 weeks of gestation. Bleeding usually occurs late in the third trimester of pregnancy as the cervix begins to dilate in preparation for delivery.

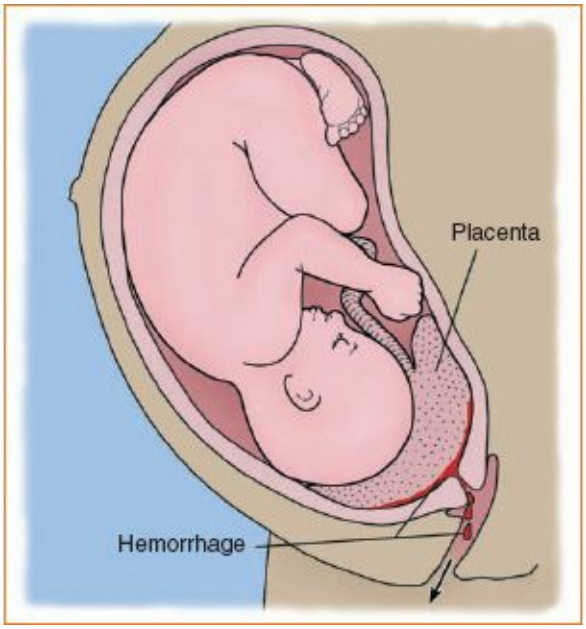


Figure 21-10 Placement of the placenta in placenta previa.

Signs and Symptoms

Placenta Previa

- Bright red vaginal bleeding
- Lack of abdominal pain
- Detectable fetal movement and heart sounds

Differential Diagnosis

Placenta Previa

- Abruptio placenta
- Ectopic pregnancy
- Preterm labor
- Spontaneous abortion

Transport Management

Placenta Previa

- Administer IV fluids.
- Administer oxygen.
- Allow minimal movement.
- Perform electronic fetal monitoring.

The usual presenting complaint of the woman with placenta previa is bright red vaginal bleeding. In

contrast to abruptio placenta, the bleeding in placenta previa is painless and the uterus remains soft. Fetal movement is detectable and fetal heart sounds remain audible because the blood supply to the fetus is not compromised.

Treatment of the patient with suspected placenta previa remains the same as with all patients with third-trimester bleeding. CCTPs need to make the mother the priority. Maintaining her hemodynamics with IV fluids and oxygenation will benefit the fetus as well. However, one must be cautious when examining the patient with placenta previa. Because the placenta is precariously placed over the cervical opening, minimal maneuvers to the cervix or uterus may induce heavy bleeding. Treatment should be accomplished with minimal movement; CCTPs should avoid palpating the fundus, which would cause fetal movement and possible placental tearing. Electronic fetal monitoring should be performed with minimal patient movement as well.

Uterine Rupture

Uterine rupture is a cause of catastrophic bleeding in the second half of pregnancy. Rupture associated with trauma is relatively uncommon. Maternal mortality of trauma-induced uterine rupture is about 10%. In contrast, the fetal mortality approaches 100%. Uterine rupture should be suspected in the pregnant trauma patient who has lost the palpable uterine contour, has easily palpated fetal parts, and has severe abdominal pain. As with any trauma in the pregnant patient, initial management is directed at the resuscitation and stabilization of the pregnant patient because fetal life depends on the patient's condition.

Signs and Symptoms

Uterine Rupture

- Loss of palpable uterine contour
- Easily palpated fetal parts
- Sharp, tearing abdominal pain
- Weakness
- Dizziness
- Thirst with signs of shock

Transport Management

Uterine Rupture

- Perform electronic fetal monitoring.
- Provide volume resuscitation.
- Administer oxygen.
- Provide rapid transport.

Uterine rupture can occur without trauma and is almost always associated with labor. A weakened portion of the uterine wall is at risk for rupture at any time during the gestational or delivery phase. Women who are most at risk have had many children or have had a previous cesarean section.

Some incomplete ruptures go unnoticed; however, complete ruptures are very dangerous to the health of the pregnant patient and fetus. The signs and symptoms of a complete rupture include sudden onset of

sharp, tearing abdominal pain. The diagnosis of uterine rupture should also be suspected in a pregnant patient in labor who reports weakness, dizziness, and thirst with signs of shock. Significant vaginal bleeding in this case may not be present. Fetal monitoring shows **long-term variability** as the fetal distress causes high fluctuations in heart rate. In some cases, the fetus and placenta exit the uterus into the abdominal cavity.

Treatment of spontaneous uterine rupture consists of volume resuscitation, oxygen, and 20 to 40 units of oxytocin in a 1,000-mL solution given IV. CCTPs need to prepare the patient for surgery because definitive care involves surgery and cesarean section. During transport, consider IV fluids and whole blood transfusions for hemodynamic stability along with oxygenation and advanced airway management.

Medical Conditions During Pregnancy

Pregnancy-Induced Hypertension

Pregnancy-induced hypertension (PIH), which occurs in about 8% of pregnancies, can have dangerous if not fatal consequences for the woman and fetus if left untreated. Risk factors include multiple gestations (twins, triplets), first-time pregnancies, and maternal chronic hypertension. In addition, women younger than 20 years or older than 40 years are at risk for PIH. PIH develops after 20 weeks of gestation, and there have been instances where PIH extends into the postpartum period, especially with patients who have a history of Guillain-Barré syndrome. CCTPs must take note that patients with a history of prior or chronic hypertension are at the greatest risk for PIH.

Although most PIH is managed effectively with diet and bed rest, uncontrolled hypertension can lead to eclampsia. Treatment must be focused on keeping the diastolic pressure below 90 mm Hg; effective medications are as follows:

- **Labetalol:** A beta-blocker that decreases systemic vascular resistance without affecting cardiac output. Administer a 20-mg IV push over 2 minutes. This administration may be repeated every 10 minutes with a 40- to 80-mg IV push until the maximum dose of 300 mg has been given.
- **Hydralazine:** A smooth-muscle relaxant that causes arteriolar relaxation, decreases vasospasms, and reduces blood pressure and cardiac output. Hydralazine is administered when the diastolic blood pressure is greater than 110 mm Hg. Administer 5 to 10 mg IV every 15 to 30 minutes up to 20 mg per hour until the diastolic blood pressure is 90 to 100 mm Hg.

Signs and Symptoms

Pregnancy-Induced Hypertension

- Blood pressure above 140/90 mm Hg
- **Proteinuria** (greater than 500 mg of protein in urine)
- Peripheral edema

Transport Management

Pregnancy-Induced Hypertension

- Administer medication such as labetalol and hydralazine to keep diastolic pressure below 90 mm Hg.

Preeclampsia and Eclampsia

Preeclampsia is the presence of the triad of hypertension, **pathologic edema**, and proteinuria (protein in the urine) due to pregnancy or recent pregnancy. Preeclampsia usually occurs after 20 weeks of gestation and may present within the first few weeks of the postpartum period. As discussed previously, blood pressure generally declines during pregnancy and therefore the definition of hypertension is different in pregnant patients and is defined as a blood pressure greater than 140/90 mm Hg or a systolic blood pressure 30 mm Hg above baseline.

The cause of preeclampsia is not known. Generally, the rise in blood pressure is the result of **vasospastic disease** of unknown cause. The proteinuria and subsequent edema is the result of vasospasm of the renal arteries, leading to decreased renal blood flow. In response to this decreased blood flow, the kidneys retain sodium and subsequently retain body water, which contributes to and aggravates the hypertensive state.

Signs and Symptoms

Preeclampsia

- Hypertension
- Edema or pathologic edema
- Proteinuria
- Headache
- Visual disturbances
- Abdominal pain
- Seizures
- Coma

Transitioning to Eclampsia

- Severe headache
- Scotomata (visual changes)
- Hyperreflexia (overactive reflexes)
- Epigastric pain
- Anxiety

Eclampsia

- Seizure

Differential Diagnosis

Preeclampsia

- Preexisting hypertension **Eclampsia**

Eclampsia

- Preexisting seizure disorder

Transport Management

Preeclampsia/Eclampsia

- Place the patient on her left side.
- Prevent/control seizures.
- Administer oxygen.
- Administer magnesium sulfate if seizures occur.

Presenting complaints of preeclampsia include headache, visual disturbances, edema, and/or abdominal pain. As preeclampsia progresses, evidence of end organ damage is seen. Hemodynamically, an increased D-dimer concentration (discussed in Chapter 8) leads to a condition known as hypercoagulative disorder, which causes increased clotting in the vasculature. Central nervous system (CNS) effects commonly include headache, visual changes, and, most importantly, seizures or coma.

Once seizures develop in the patient with signs and symptoms of preeclampsia, the condition is defined as **eclampsia**. Warning signs for the transition from preeclampsia to eclampsia are severe headache, scotomata (visual changes), **hyperreflexia** (overactive reflexes such as ankle clonus), epigastric pain, and anxiety. Preeclampsia may progress rapidly to eclampsia before, during, or after delivery, particularly during the first 48 hours postpartum, but occasionally the progression may be as long as 10 days after delivery. A change in diastolic blood pressure of 15 mm Hg above baseline, an increase in urine protein in a 24-hour period, or an increase of 100 mg/dL in random urine analyses 6 hours apart indicates the potential for eclampsia. The only true treatment for eclampsia is delivery. Maternal complications from eclampsia include permanent CNS damage, renal insufficiency, and death.

The management goals for the CCTP in preeclampsia and eclampsia include the prevention of seizures. If seizures do occur, rapid control of the seizures and the administration of magnesium sulfate are vital. Eclampsia should always be considered in a pregnant patient who is having a seizure or in a patient who has recently delivered an infant who is having a seizure. A pregnant patient with edema, hyperactive reflexes, and an elevated blood pressure requires no specific prehospital treatment other than prophylactic venous access. The patient should be lying on her left side. Oxygen may be administered.

Seizures may occur suddenly and should be anticipated. If seizures occur, routine seizure management and protection of the patient should be initiated. Treatment of seizures caused by eclampsia includes the administration of a 1- to 6-g IV bolus of magnesium sulfate, which is believed to act as a vasodilator and thereby reduces cerebral ischemia. Magnesium sulfate is a poor antihypertensive medication, but is an effective anticonvulsant that preserves uterine blood flow. If the patient already has an IV established, the dose for magnesium sulfate should be 2 g IV for 3 to 5 minutes. If no IV has been established, the loading dose is 4 g for 5 minutes IV If seizures continue, administer 2 g for 3 to 5 minutes.

Controversies

Practitioners may be reluctant to use large doses of benzodiazepines to terminate active seizures in a pregnant patient for fear of inducing respiratory depression in the infant if emergent delivery becomes necessary. The benefits of terminating seizure activity in the mother outweigh any concerns for the effects of benzodiazepines on the fetus. Should emergent delivery be performed, side effects observed in the infant are easily managed.

The administration of magnesium sulfate should be considered according to the transfer orders or upon consultation with medical direction. The administration of magnesium sulfate should always be accompanied by clinical observation for loss of reflexes, respiratory depression, and, if the patient is not currently experiencing a seizure, CNS depression. The antidote for magnesium sulfate toxicity is calcium gluconate. If magnesium sulfate is not available, standard seizure therapy may be followed, which includes the use of diazepam or other benzodiazepines. Even though benzodiazepines are effective anticonvulsants, they are not as effective as magnesium for the treatment of eclampsia. The administration of medications to lower blood pressure should be delayed until you reach the hospital because a careful titration of an antihypertensive medication is required to prevent a precipitous fall in blood pressure. Rapid lowering of blood pressure can result in uterine hypoperfusion and fetal morbidity or death.

HELLP Syndrome

A hemolytic disorder, **HELLP syndrome** occurs during the latter stages of pregnancy, usually after the 20th week of gestation. HELLP syndrome is named after its clinical findings, namely, hemolytic anemia, elevated liver enzyme levels, and a low platelet count.

The cause of HELLP syndrome is unknown, but a condition develops with the clotting cascade similar to end-stage shock patients with multiorgan dysfunction syndrome (discussed in Chapter 9).

Signs and Symptoms

HELLP Syndrome

- Hemolytic anemia
- Elevated liver enzyme levels
- Low platelet count
- Abdominal pain
- Blurred vision
- Headache
- Edema

Differential Diagnosis

HELLP Syndrome

- Cholecystitis
- Hepatitis
- Idiopathic thrombocytopenia

Transport Management

HELLP Syndrome

- Perform a blood transfusion.
- Administer magnesium sulfate (to prevent seizures).

As in preeclampsia, patients with HELLP syndrome will present with blurred vision, abdominal pain, headache, and edema. Treatment can include blood transfusions to combat the anemia and the administration of 1 to 6 g of magnesium sulfate to reduce the likelihood of having seizures. However, definitive treatment can only be accomplished with delivery of the fetus. CCTPs need to be aware that HELLP syndrome can occur postpartum 10% of the time.

Complications During Labor

Delivery includes a prodromal stage plus three distinct stages, the durations of which depend partly on whether or not it is the patient's first pregnancy (nullipara). **Table 21-4** provides a review of the stages of labor.

Transition of Fetal Circulation at Birth

Prior to birth, the fetus receives oxygenated blood from a pregnant woman via the umbilical vein, the ductus venosus, and the inferior vena cava of the fetus, which ultimately leads to the right atrium of the fetus. Prior to birth, the fetal lung is collapsed and filled with fluid, and most of the fetal blood flow is diverted away from the lungs. As the baby is delivered, fetal transition occurs—a process that enables the baby to breathe **Figure 21-11**. During fetal transition, the newborn's lungs must expand with air within seconds. As the baby's lungs become filled with air, the pulmonary vascular resistance decreases and systemic vascular resistance increases. Blood begins to flow to the lungs, picking up oxygen. The change in pulmonary pressure results in closing of the foramen ovale, the opening that allows blood to flow from the right atrium to the left atrium of the fetus, bypassing the lungs prior to birth.

Anything that delays this decline in pulmonary pressure during fetal transition can lead to delayed transition, hypoxia, brain damage, and, ultimately, death.

Preterm Labor

Preterm delivery represents 8% to 10% of all births and results in 60% of all perinatal morbidity and mortality. In critical care medicine, the emphasis is on interrupting preterm labor. **Preterm labor** is defined as the onset of labor before 36 weeks of gestation, as evidenced by rhythmic uterine contractions and cervical **dilation**. This presentation is cause for concern and often requires intervention such as specialized care at a facility capable of managing preterm labor and **premature** infants (infants born before 36 weeks of gestation or weighing less than 5 lb at birth).

Of all pregnant patients, 25% to 50% will experience contractions without being in preterm labor, known as Braxton-Hicks contractions. Treatment includes lowering anxiety levels in the pregnant patient along with bed rest and supportive care.

TABLE 21-4 Stages of Labor

Stage of Labor	Average Time (Nullipara)	Average Time (Multipara)	Frequency of Contractions	Characteristics
Prodromal stage	Varied	Varied	N/A	Bloody show (mucus mixed with blood) expelled from the vagina
First stage	8 to 12 h	6 to 8 h	5- to 15-min intervals	Labor pains and contraction of the uterus begin. Pain may radiate into the small of the back. Amniotic sac often ruptures toward the end of this stage, signified by a gush of fluid from the vagina. This stage lasts until the cervix is fully dilated.
Second stage	1 to 2 h	30 min	2 to 3 min apart	Begins as the baby's head enters the birth canal. Woman's pulse rate increases, sweat appears on her face. Cervix becomes fully dilated and effaced. Crowning occurs (presenting part of baby becomes visible). Delivery is imminent. This stage concludes when the baby is fully delivered.
Third stage	5 to 60 min	5 to 60 min	N/A	Placental stage Lasts from delivery of the baby until the placenta has been expelled and the uterus has contracted. Uterine contraction is necessary to squeeze shut all of the tiny blood vessels left exposed when the placenta separates from the uterine wall.

The use of critical care transport for a pregnant patient in preterm labor can be critical if the transferring facility is incapable of managing these patients. Knowledgeable CCTPs provide the critical link between the local hospital and the tertiary care facility. Careful patient monitoring, being prepared to treat changes in the patient's condition while in transit, and a smooth transfer of care to the receiving hospital will improve patient outcomes. Emphasis on continued bed rest and limiting physical activity are vital to extend gestation, and CCTPs need to be aware that during a stressful transport, sedatives may need to be administered. Discuss with the receiving physician the use of proper sedative agents to reduce anxiety and ease in the transfer. Also consider IV hydration, which increases intravascular volume and uterine blood flow, decreases pressure on the cervix, and inhibits release of pitocin.

Premature or preterm labor and fetal immaturity are the leading causes of neonatal mortality. Precipitous delivery may occur with premature labor. On presentation, the patient should be asked about the frequency and duration of contractions as well as the passage of blood-stained mucus and whether there has been rupture of membranes signaled by a gush of fluid or constant fluid leakage. This condition, known as premature rupture of membranes, accounts for up to 50% of all preterm deliveries. Knowledge of these signs and symptoms allows CCTPs to understand the stage of labor the patient is entering. Although all prehospital deliveries are considered emergencies, the way to reduce risk to the pregnant woman and fetus is to be prepared to deliver at any time. In preterm delivery between 26 and 34 weeks' gestation, the administration of glucocorticoid therapy such as betamethasone and dexamethasone results in stronger fetal lung tissue and increased surfactant fluid production, resulting in decreased cases of fetal respiratory distress syndrome. CCTPs can discuss beginning this course of treatment during transport to the receiving facility.

Signs and Symptoms

Preterm Labor

The following, occurring prior to 36 weeks' gestation:

- Rhythmic uterine contractions
- Cervical dilation
- Rupture of membranes
- Passage of blood-stained mucus

Differential Diagnosis

Preterm Labor

- Abruptio placenta
- Ectopic pregnancy
- Placenta previa
- Spontaneous abortion

Transport Management

Preterm Labor

- Provide or continue therapy to prolong gestation (fetal maturation therapy) or interrupt labor.
- Provide or continue bed rest.
- Consider hydrating the patient.
- Consider administration of tocolytic agents.

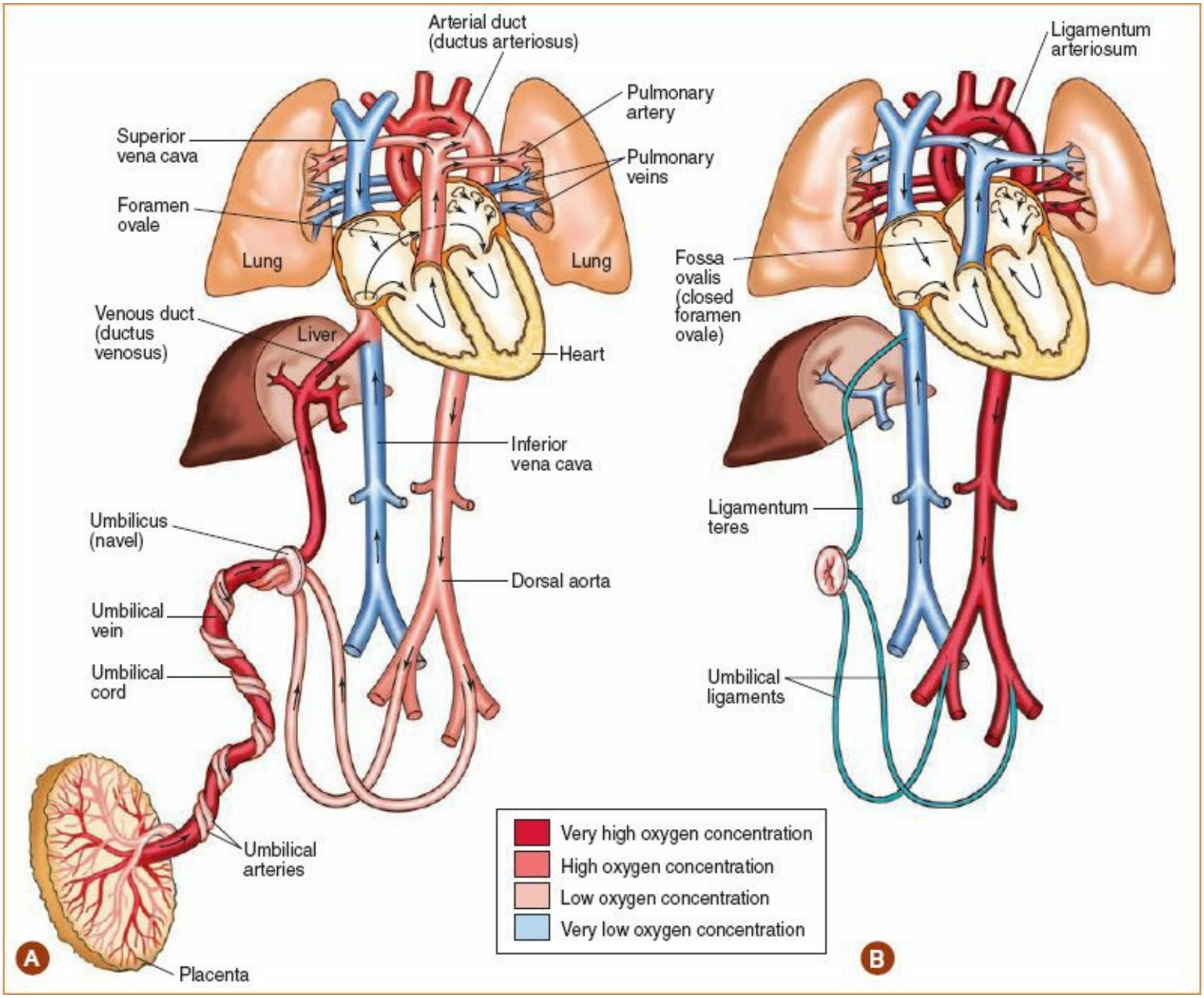


Figure 21-11 Fetal circulation. **A.** Oxygenated blood from the placenta reaches the fetus through the umbilical veins. Blood returns to the placenta via two umbilical arteries. **B.** Fetal circulation following transition.

Viability of the fetus is possible at 23 weeks of gestation, but fetal mortality and morbidity rates are extremely high. If a viable fetus is present and the woman is relatively healthy, the medical management is directed towards prolongation of gestation if possible. Treatments to prolong gestation include fetal maturation therapy, bed rest, hydration, and the administration of pharmacologic agents, termed **tocolytics**, to interrupt labor.

Inversely, prolonged pregnancy over 40 weeks can lead to instances of fetal hypoxia during labor and a higher percentage of meconium production. CCTPs need to be aware of these risks and have meconium suctioning equipment available prior to delivery **Figure 21-12**.

Tocolytic Agents

The ideal tocolytic agent effectively antagonizes myometrial (uterine) muscle contraction and has no systemic effects. Unfortunately, the complexity of the physiology behind smooth-muscle contraction makes this task difficult. The uterus is composed of smooth muscle, under the control of the auto-nomic nervous

system, and requires sufficient concentrations of calcium to contract. The beta-2 receptors located on the myometrial cell membranes control the intracellular calcium concentration. These receptors activate an enzyme called aden-ylcyclase, which is responsible for the conversion of adenosine triphosphate to cyclic adenosine monophosphate, which causes a decrease in the intracellular calcium concentration.

TABLE 21-5 Premature Labor Therapies

Class	Drug	Use	Bolus Dose
Tocolytic	Magnesium sulfate	Stop uterine contraction	1–6 g IV, then 2–4 g/h infusion
	Terbutaline	Stop uterine contraction	0.25–1 mg SC every 2–4 h

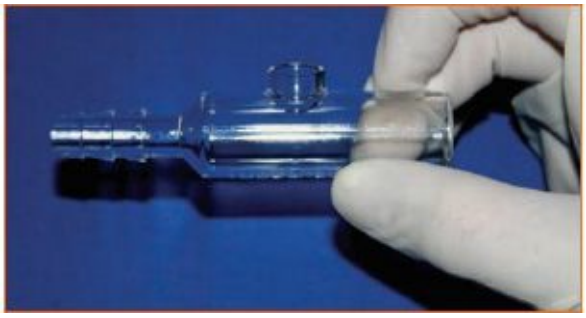


Figure 21-12 A meconium aspirator.

Tocolytics are only marginally effective in stopping preterm labor. The two most commonly administered tocolytic agents are terbutaline (up to 1 mg) and magnesium sulfate (1 to 6 g). Terbutaline is a beta-2 agonist that, when used in sufficient doses, can be successful in reducing uterine contractions. The doses used to reduce contractions, however, can result in side effects by stimulating beta-2 receptors elsewhere, such as in the myocardium. Stimulation of myocardial receptors can result in tachycardia or frank myocardial ischemia in patients with underlying coronary artery disease. Thus, the patient should have continuous cardiac and fetal monitoring during transport and be frequently assessed for signs and symptoms of myocardial ischemia, such as chest pain and pulmonary edema. Terbutaline should not be administered if the maternal pulse exceeds 140 beats/min.

The effects of beta-2 stimulation of the pancreas can result in hyperglycemia, which can be monitored in the pregnant patient using finger stick glucose checks. If the patient's baby is delivered during transport, the baby could have hypoglycemia as well. Fetal hypoglycemia results secondary to the need for increased insulin production by the fetus to compensate for high maternal glucose levels. Other less significant effects of terbutaline treatment include headache, tremor, vomiting, fever, and hallucinations.

Another common drug effectively used to arrest preterm labor is magnesium sulfate. This medication is usually administered as a 1- to 6-g bolus followed by a continuous infusion at 2 to 4 g/h. The mechanism by which magnesium inhibits uterine contraction is not completely clear, but it asserts its effects through the antagonism of calcium in muscle cells. The doses required to achieve this effect are close to toxic. Symptoms of magnesium toxicity are decreased patellar reflexes and, ultimately, respiratory depression and respiratory arrest. In a patient receiving magnesium, the patellar reflexes should be monitored frequently, urine output should be at least 25 mL/h, and there should be no signs of respiratory depression. If any signs or symptoms of toxicity are noted, the infusion should be stopped and medical control contacted for further instruction. Magnesium is cleared by the kidney, thus making the accurate measurement of urine output using a Foley catheter important. If the patient's urine output falls,

the serum magnesium level will rise. Therapeutic levels of magnesium are between 4 and 7 mEq/L. Patellar reflexes become diminished when magnesium levels are between 8 and 10 mEq/L, and respiratory depression and failure occur when levels rise above 12 mEq/L. If the patient exhibits a toxic reaction such as respiratory depression, the infusion should be stopped; 1 g of calcium gluconate should be given by *slow* IV push, and oxygen should be administered to maintain a saturation of greater than 90%. In the face of respiratory failure, the patient should be intubated.

Table 21-5 lists premature labor therapies.

Delivery

Vertex, or head-first presentation, is the ideal presentation for all deliveries; crowning is observed as the second stage of labor begins. The role of the CCTP is to ensure that the delivery proceeds without any complications. It is the responsibility of the CCTP's trained eye to note unexpected events when the baby is presenting.

Normal crowning and normal shoulder presentation are shown in **Figure 21-13** and **Figure 21-14**, respectively.

Malpresentations

Breech presentation is the most common malpresentation, occurring in 4% of all deliveries. With this presentation, there is an increased risk of asphyxia and fetal mortality, though one third of these are considered preventable. Breech presentations are more common in the premature infant and in those with a low birth weight, although two thirds of breech infants weigh more than 2,500 g.

There are four common variants of breech presentation:

1. **Frank breech**: both of the infant's hips are flexed and the feet are near the infant's head **Figure 21-15**.
2. **Complete breech**: the hips are flexed and the legs are flexed at the knees and oriented transversely to the birth canal; the buttocks present with the legs flexed and the feet along the buttocks **Figure 21-16**.
3. **Incomplete breech**: similar to complete breech except one foot extends into the birth canal **Figure 21-17**.
4. **Footling breech**: both feet extend into the birth canal

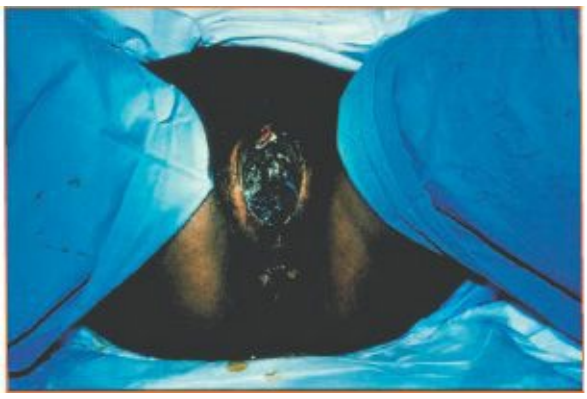


Figure 21-13 Normal crowning.

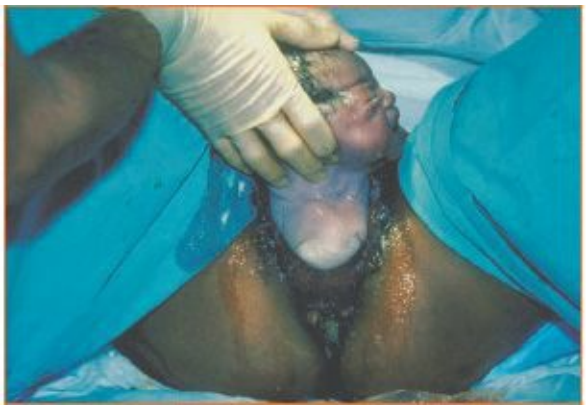


Figure 21-14 Normal shoulder presentation.

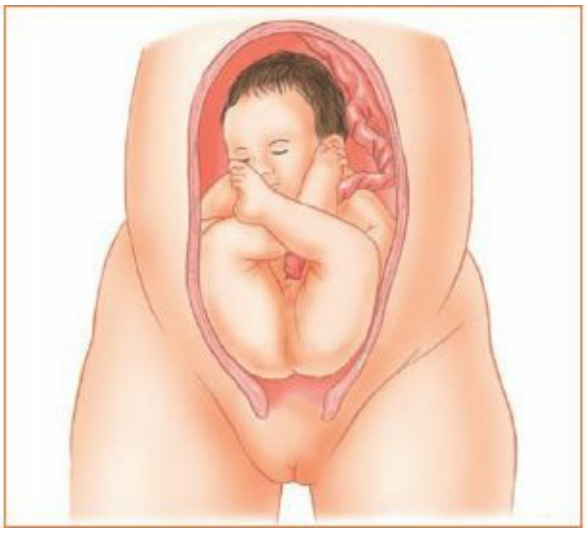


Figure 21-15 Frank breech presentation.

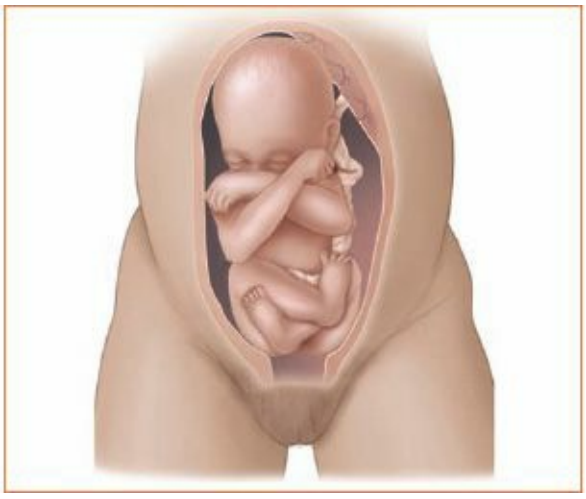


Figure 21-16 Complete breech presentation.

Figure 21-17 Incomplete breech presentation.

The footling breech is the only malpresentation in which field delivery should be considered. All other limb presentations cannot be manipulated in the field and must be transported to the nearest appropriate facility.

To deliver a footling breech baby, the patient is usually positioned supine with hips flexed, as in a normal delivery, or maybe on her hands and knees. As the baby (ie, the feet) emerges from the birth canal, the body of the infant should be supported and directed toward the mother's anterior.

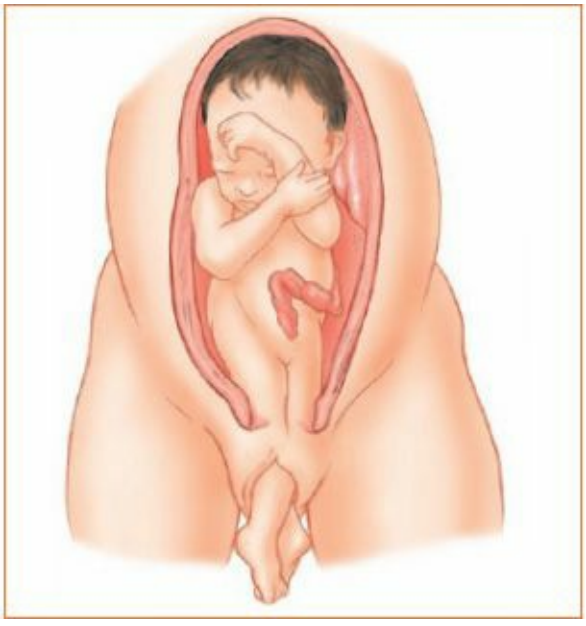


Figure 21-18 Footling breech presentation.

Special Populations

In breech delivery, the cervix is less than completely dilated, which will likely delay delivery of the head.

- Maintain the infant's airway with the index and middle finger on either side of the infant's mouth and nose, pushing the vaginal wall away.
- Suction as soon as the head delivers. If the head does not quickly deliver, continue transport.
- With a footling breech delivery, support the baby toward the mother's anterior as he or she emerges.

The head will likely be the most difficult to deliver because it is the largest body part. In a normal delivery, the head fully dilates the cervix and allows for easy passage of the remainder of the body. However, in the case of a breech delivery, the head delivers last and the cervix is less than completely dilated. In this case, the head is likely to be delayed in its delivery as the cervix dilates maximally. Once the umbilicus has cleared the birth canal and is visible, this means that the head has cleared the cervix and the infant can be held. With one hand supporting the infant, place the index and middle finger of the other hand in the birth canal against the birth canal wall on either side of the infant's mouth and nose, pushing the wall away from the airway. This maneuver should provide a route for the infant to breathe and should be maintained en route to the hospital or until delivery is complete. Disengage the legs from the birth canal if they have not yet been delivered. As the shoulders present, the arms are delivered by hooking the index finger over each of the baby's shoulders. As the shoulders clear, rotate the baby so that its chest is facing upward and apply gentle traction until the hairline is visible. During the entire process, if there is an extra provider available, he or she can assist the delivery by applying suprapubic pressure. Suctioning can be performed as soon as the head delivers, but if the head does not quickly deliver, transport should continue.

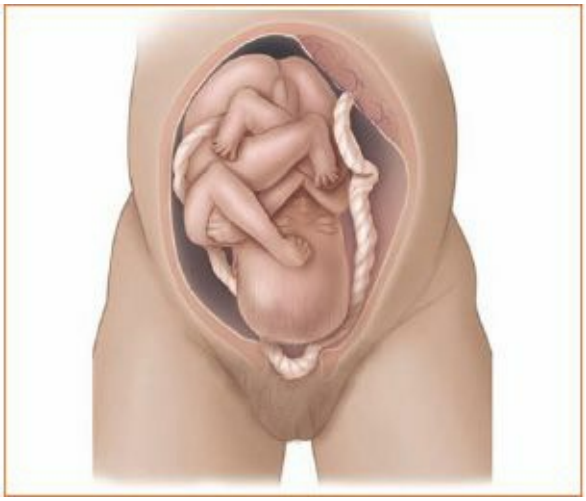


Figure 21-19 A prolapsed cord.

Umbilical Cord Prolapse

Occasionally the presenting part may be the umbilical cord **Figure 21-19**. This condition, in which the umbilical cord passes through the birth canal in advance of the infant, is known as a prolapsed cord and can have serious consequences for the infant.

The cord may be compressed between the bony pelvis of the mother and the head of the infant, thus decreasing blood flow to the infant. Low blood flow to the infant can cause fetal distress or death.

In the event of a prolapsed cord, the patient should be placed on 100% oxygen by nonrebreathing mask and placed in the knee-chest position on the stretcher. This involves the patient being prone on her elbows and knees with buttocks elevated **Figure 21-20**. If the patient cannot tolerate this position, an alternate is the Trendelenburg position. Once the patient is positioned correctly, encourage her not to push. Insert two gloved fingers into the vagina to gently elevate the presenting part to relieve pressure on the cord and restore umbilical pulse. *Do not push the cord back in*. To prevent umbilical cord drying that may result in atrophy of the vessels, gently cover any exposed cord with sterile saline gauze. Continue rapid transport and notify the receiving hospital as soon as possible.

Figure 21-20 The knee-chest position should be used in the event of a prolapsed cord.

Transport Management

Umbilical Cord Prolapse

- Administer oxygen.
- Position the patient in either the knee-chest position or the Trendelenburg position.
- Insert two gloved fingers to relieve cord compression.
- Encourage the patient not to push.
- Cover any exposed cord with a sterile saline gauze to prevent drying.
- Provide rapid transport.

Shoulder Dystocia

In about 1 of every 100 vertex deliveries, there exists a condition in which the infant is too large (macrosomia) to travel through the birth canal without additional clinical maneuvering. This is **shoulder dystocia**. CCTPs will know this condition exists after seeing a feature known as "turtle sign"; the fetal head will begin to protrude and then withdraw back inside as the shoulders, being too large, are prevented from exiting the uterus. Because the head is now stuck back inside and the face is pressing against the walls of the birth canal, there is a high risk for neonatal anoxia. CCTPs must now assist the mother with the next contraction; most contractions are no more than 30 seconds apart so there must be a rapid decision and treatment plan.

One common technique to assist the patient is known as the McRoberts maneuver. The patient is instructed to hyperflex her legs to her abdomen **Figure 21-21**; this will widen the pelvis and lower the lumbar spine, thus enlarging the birth canal. This procedure has been proven effective in 40% of deliveries. Pressing against the suprapubic region will increase the percentage of success. *Do not apply fundal pressure;* this can cause the stuck shoulder to rupture the uterus. *Do not pull on the baby's head;* pulling on the head can cause a brachial plexus injury, which can cause permanent disability or death of the infant.

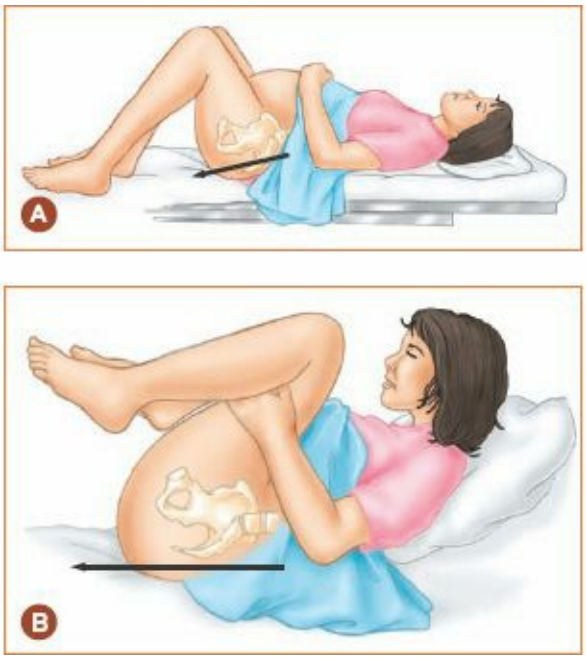


Figure 21-21 If the fetal head begins to protrude and then withdraws back inside (a sign of shoulder dystocia), the first step is to have the mother hyperflex her legs to her abdomen to help enlarge the birth canal.

If there is still no delivery, insert a gloved hand into the vagina and locate the humerus. Gently rotate the infant's arm towards its sternum; this action will displace the shoulder away from the vaginal wall in an attempt to shorten the diameter of the baby's axial skeleton.

Signs and Symptoms

Shoulder Dystocia

- "Turtle sign" of infant's head protruding and withdrawing into the birth canal

Transport Management

Shoulder Dystocia

- Perform the McRoberts maneuver.
- With a gloved hand, rotate the baby's arm toward its sternum.
- Provide rapid transport.

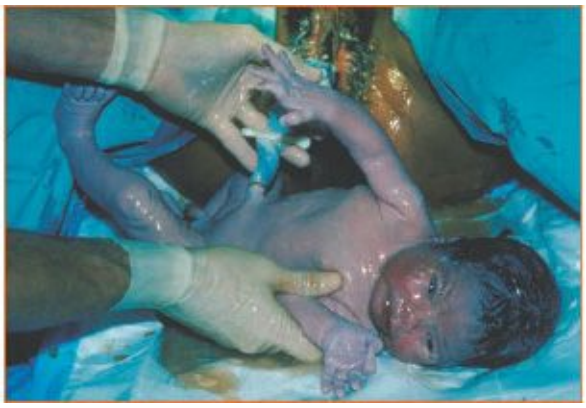


Figure 21-22 Tie or clamp the cord in two places (approximately 3″ and 4″ from its insertion into the baby). Cut the cord between the two ties or clamps.

CCTPs need to be aware that they are limited to what their scope of practice dictates. In extreme cases of shoulder dystocia, obstetricians perform episiotomies, perform shoulder dislocations, or push the head back into the uterus and perform a cesarean section. None of these procedures are performed by CCTPs, so it is important to have a plan of action such as diverting to the nearest obstetric facility if faced with an extreme case that cannot be corrected within your scope of practice.

Multiple Births

Single gestations are most common, although invariably many CCTPs will encounter a multiple-gestation pregnancy and potential delivery. The incidence of twin gestation is 1:100; and triplets, 1:10,000. The presence of a multiple-gestation pregnancy should be identified by history obtained from the patient. Twins may share a placenta and a gestational sack or they may each have their own individual sack and placenta.

After the first infant is delivered, his or her cord should be double-clamped to prevent bleeding (in the case of a shared placenta) and allow for identification of each infant's cord. Place one to two clamps close to the infant and one to two clamps 3 cm from the first set of clamps, then cut the infant's cord between the four clamps **Figure 21-22**.

There is usually a delay of about 15 minutes between deliveries. During this delay, transport should continue and should only be interrupted if the next infant begins to emerge from the birth canal. If the subsequent infant is malpresenting, take the appropriate measures previously described and continue transport. The orientation of each infant at the time of birth should be accurately documented and relayed to the hospital. The presentation is documented as, for example, vertexbreech, with the first term referencing the first infant. Be sure to monitor each infant separately once delivered.

Postdelivery Care

Once delivery is complete, care for both the mother and infant should continue. The newborn's ABCs and APGAR score should be determined (further discussed in Chapter 22). The mother's perineum should be examined for tears. If large tears are actively bleeding, apply direct pressure. Significant postpartum hemorrhage is defined as greater than 500-mL blood loss within 24 hours after delivery of the infant. Often the source of bleeding is not easy to identify; thus, the uterus should be massaged and one or two large-bore IVs established with a crystalloid solution running open. High-flow oxygen should be applied, and, if extended transport time is anticipated, orders for pitocin may be given by medical control. Pitocin will aid in uterine contraction and should be given as 10 to 20 U/L of fluid.

The infant should be thoroughly cleaned and dried, wrapped in warm blankets, and placed next to the

mother if possible. The APGAR score should be taken at 1 and 5 minutes after delivery. If the APGAR score is below 7, repeat every 5 minutes. Continue with any supportive measures applied as needed while transporting.

Postdelivery Complications

Amniotic Fluid Embolism

Amniotic fluid embolism, although rare, can present antepartum and postpartum in 1 of every 8,000 patients and occurs when fetal tissue crosses over the placental barrier into maternal circulation. The most frequent routes of entry include lacerations to the placenta site, the endocervical vein, or areas in the lower portion of the uterus. Amniotic fluid leakage into maternal circulation can cause obstruction to the mother's organs with organ failure or respiratory and cardiovascular compromise. Causes of this pathophysiology are abdominal trauma with amniotic and uterine vein rupture, twins, older gestational periods (longer than 40 weeks), and older patients (older than 40 years). The mother presents with sudden onset of shortness of breath and hypotension. Other signs and symptoms include chest pain, restlessness, anxiety, coughing, vomiting, pulmonary edema with pink, frothy sputum, seizures, coma, and sudden death.

Signs and Symptoms

Amniotic Fluid Embolism

- Sudden onset of shortness of breath
- Sudden onset of hypotension
- Chest pain
- Restlessness
- Anxiety
- Coughing
- Vomiting
- Pulmonary edema with pink, frothy sputum
- Seizures
- Coma
- Sudden death

Transport Management

Amniotic iluid Embolism

- Manage the airway.
- Provide fluid resuscitation.
- Provide rapid transport.
- Administer positive end-expiratory pressure.
- Administer blood.

Amniotic fluid embolism carries a 50% mortality rate; therefore, the CCTP must recognize the signs

and symptoms early and treat with airway management, fluid resuscitation (it is recommended to establish two IV lines), and immediate transport. Additionally, positive end-expiratory pressure may be required.

Postpartum Hemorrhage

Bleeding is common during delivery; blood loss of over 500 mL, however, is considered to be serious and is the leading cause of maternal death. Causes of postpartum hemorrhage are listed as follows:

- **Trauma:** Tearing away of the placenta prematurely or a ruptured placenta
- **Tone:** Inability of the uterus to contract, leading to extended bleeding
- **Tissue:** Any unexpelled fetal product or placenta can cause increased bleeding
- **Thrombin:** Bleeding disorders of the mother can prevent clotting

Management should include padding to produce clotting (do *not pack*, just place a pad), fundal massage **Figure 21-23**, and, after the placenta has been delivered, administering an oxytocin IV drip. If bleeding persists, the CCTP can perform bimanual uterine compression, providing it is within the local scope of practice. In this procedure, one hand, wrapped in a dressing, is clenched as a fist in the vagina while the other hand presses down on the uterus. After about 2 to 5 minutes, release pressure and re-examine for continued bleeding. Repeat as needed.

Signs and Symptoms

Postpartum Hemorrhage

- Severe bleeding after delivery

Transport Management

Postpartum Hemorrhage

- Place padding to absorb the blood.
- Perform fundal massage.
- Administer oxytocin after the placenta has delivered.

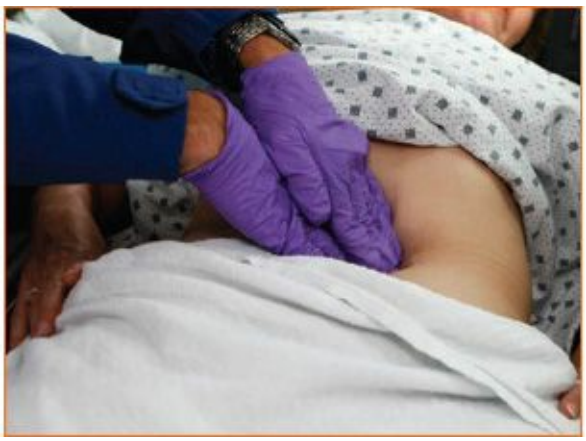


Figure 21-23 Fundal massage should be performed if the patient experiences postpartum hemorrhage.

Rh Incompatibility and Erythroblastosis Fetalis

In some cases, Rh factors in red blood cells in the mother and child can be different. Although this is a well-managed condition prenatally, during delivery blood may mix, resulting in an antibody reaction and inflammatory response. Signs and symptoms include maternal fever, nausea, vomiting, and shortness of breath. Treatment of the mother includes oxygenation and administration of antipyretics.

Erythroblastosis fetalis, a condition caused by the mother's antibodies attacking fetal red blood cells, can develop in the fetus, leading to in utero anemia. Upon delivery, newborns with this condition can appear jaundiced, swollen, and pale. Treatment includes addressing the anemia with blow-by oxygen and admission to a neonatal intensive care unit for treatment of the high bilirubin count.

Signs and Symptoms

Rh Incompatibility in a Mother

- Fever
- Nausea
- Vomiting
- Shortness of breath

Transport Management

Rh Incompatibility in a Mother

- Administer oxygen.
- Administer antipyretics.

Uterine Inversion

Usually 10 to 30 minutes after delivery, the placenta will detach from the uterine wall and deliver vaginally. In rare cases, the placenta will remain attached and as the uterus contracts to expel the afterbirth, the uterus inverts and protrudes from the vagina. Risk factors for this condition include pulling on the cord to induce placental detachment and use of magnesium sulfate during the delivery. Surgical intervention is the most definitive treatment, but CCTPs (if within the local scope of practice) can attempt to correct this complication by flushing the vagina with saline. This procedure, known as hydrostatic correction, will cause the uterus to re-inflate and return to the proper position. Oxytocin can also be given to prevent any further contractions and more profound inversion and protrusion.

Transport Management

Uterine Inversion

- Flush the vagina with saline (hydrostatic correction).
- Administer oxytocin.

Ectopic Pregnancy

A n **ectopic pregnancy** is an extra-utero conception and is not considered a viable fetus, because development in the tubules is a life-threatening condition to women. Most ectopic pregnancies occur in the fallopian tubes, although a small percentage can gestate in the peritoneum, in an ovary, in the cervix, or interstitially. When the fertilized egg implants on the uterine wall, it will begin to burrow by degrading tissue on the wall lining. In the tubules, where there is no build-up of tissue that would be normally found in the uterus, this degradation can lead to vessel rupture and bleeding. For this reason, all women of childbearing years with sudden onset of abdominal pain or uncommon vaginal bleeding must be considered to have an ectopic pregnancy. The greatest risk factor for the occurrence of an ectopic pregnancy is pelvic inflammatory disease. Severe pelvic inflammatory disease can cause tubule scarring, which destroys the cilia hairs that guide the ovum to the uterus. Other risk factors include tubal ligation, reversed ligation, current intrauterine device use, and smoking.

Signs and Symptoms

Ectopic Pregnancy

- Abdominal pain (sudden onset)
- Vaginal bleeding

Differential Diagnosis

Ectopic Pregnancy

- Abruptio placenta
- Placenta previa
- Preterm labor
- Spontaneous abortion

Transport Management

Ectopic Pregnancy

- Continue supportive therapy.
- Continue the administration of methotrexate (for specific cases that meet defined criteria).

Treatment for ectopic pregnancy is primarily surgical; however, early inhospital treatments include the antimetabolite methotrexate in certain cases that meet defined criteria (less than 2 cm in size, and an hCG of less than 15,000 mIU/mL). Methotrexate disrupts the growth of the embryo and causes pregnancy termination.

Pelvic Inflammatory Disease

Pelvic inflammatory disease (PID) causes inflammation to the reproductive system, including the uterus, fallopian tubes, and ovaries, and is most commonly the result of sexually transmitted diseases. If left

untreated, the inflamed tissue scars and loses much of its functionality. Of the one million women who are diagnosed with PID each year, 10%, or 100,000, will become infertile as a result.

Patients with PID will complain of back and abdominal pain with painful urination and have a low-grade fever. In addition, the inflammation sometimes makes it difficult to ambulate without pain, and patients sometimes present with signs and symptoms that mimic appendicitis.

Treatment is done with IV antibiotics; CCTPs must be aware of any drug interactions when transporting patients on an antibiotic regimen.

Signs and Symptoms

Pelvic Inflammatory Disease

- Back and abdominal pain
- Painful urination
- Low-grade fever
- Difficulty ambulating without pain

Differential Diagnosis

Pelvic Inflammatory Disease

- Appendicitis
- Urinary tract infection

Transport Management

Pelvic Inflammatory Disease

- Continue the administration of IV antibiotics.

Toxic Shock Syndrome

During menstruation, the presence of protein-rich blood and a neutral pH creates a fertile environment for the gram-positive bacteria *Staphylococcus aureus* and *Streptococcus pyrogenes*. Super-absorbent tampons can aid in bacteria colonization, as was first discovered in the 1980s. The Centers for Disease Control and Prevention now reports that only 10 in every 100,000 menstruating people will have bacterial infections leading to toxic shock syndrome. Signs and symptoms include high fever, body rash, headache, and hypotension. Treatment includes airway management, fluid resuscitation, and IV antibiotics such as cephalosporins or vancomycin.

Signs and Symptoms

Toxic Shock Syndrome

- High fever

- Body rash
- Headache
- Hypotension

Transport Management

Toxic Shock Syndrome

- Manage the airway.
- Provide fluid resuscitation.
- Continue the administration of IV antibiotics.

Ovarian Cysts

Ovarian cysts occur in about 7% of the female population annually, typically as a result of follicular maturation during the normal female menstrual cycle. Ovarian cysts are often unde-tectable and self-resolving, although a small percentage may continue to grow and may cause lower abdominal pain. These functional cysts are divided into two categories: follicular cysts (also called graafian cysts) that result from nondegenerated graafian follicles, and corpus luteal cysts from nondegenerated corpus luteum. Follicular cysts commonly occur during the first 2 to 3 weeks of the menstrual cycle.

Most ovarian cysts are benign and asymptomatic and are usually discovered only in a routine checkup. If the cyst exceeds a diameter of 4 cm, however, pelvic discomfort begins, either from distention of the pelvic and lower abdominal structure or from ovarian torsion. Symptoms of a cyst include abdominal pain, nausea, vomiting, and frequent urination from bladder pressure.

Pain management with nonsteroidal anti-inflammatory drugs or, in severe cases, morphine and fentanyl is recommended; however, CCTPs need to remember that all abdominal pain must be considered to be caused by an ectopic pregnancy until definitively diagnosed otherwise.

Laboratory Assessment

There is no specific laboratory technique to test for an ovarian cyst **Figure 21-24**. Instead, diagnosis is made through ultrasonography and Doppler flow studies that can monitor blood flow as well as cyst size and location. A computed tomographic scan and magnetic resonance imaging are useful alternatives, although usually not required.

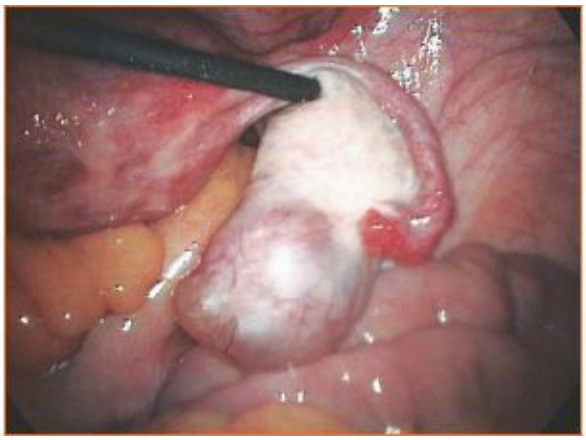


Figure 21-24 An ovarian cyst.

Underlying and resultant conditions related to the cyst may be detected with blood tests. A complete blood count, for example, can detect leukocytosis relating to an infectious process. A decreased hematocrit value may indicate anemia, and prolonged PT and PTT times can detect clotting abnormalities that may complicate the healing process. Cancer antigen 125 (CA-125) levels greater than 35 U/mL suggest carcinoma as the origin of the cyst. CA-125 is a surface protein that is found on normal ovarian tissue cells and carcinomas; its level increases following abnormal excessive tissue growth. Blood and cervical cultures should be monitored for signs of infection. Last, a urinalysis is helpful in determining whether any blood is present in the urine.

Ruptured Ovarian Cyst

Occasionally, ovarian cysts may rupture spontaneously or following trauma. Mild to severe hemorrhaging frequently occurs when a cyst ruptures. If a rupture with hemorrhaging has occurred, patients may present with increased lower abdominal and pelvic pain, vaginal bleeding, and hypovolemia leading to shock. The rupture of an ovarian cyst causes severe **mittelschmerz pelvic pain** to the affected side. Corpus luteal cysts occur during the final 1 to 2 weeks of the menstrual cycle, and their rupture can lead to pelvic inflammation and hemorrhage.

Treatment for patients with a ruptured ovarian cyst includes controlling any immediate life threats. Evaluation for hypovolemic shock is paramount and should include testing for orthostatic changes. Isotonic fluid boluses through large-bore IVs are indicated to maintain an adequate blood pressure for patients in shock.

Signs and Symptoms

Ruptured Ovarian Cyst

- Increased lower abdominal and pelvic pain
- Vaginal bleeding
- Hypovolemia leading to shock

Differential Diagnosis

Ruptured Ovarian Cyst

- Hematuria
- Appendicitis
- Diverticulitis
- Bowel obstruction
- Ovarian tumor
- Ovarian torsion
- Ectopic pregnancy
- Salpingitis (inflammation of the fallopian tube)
- Inflammatory bowel disease
- Pelvic inflammatory disease

Transport Management

Ruptured Ovarian Cyst

- Control any immediate life threats.
- Evaluate for hypovolemic shock.
- Test for orthostatic changes.
- Administer isotonic fluid boluses through large-bore IVs if patient is in shock.

Definitive management occurs at the hospital and normally involves laparoscopic surgery to control bleeding and remove the cyst.

Pathologic Cysts

Neoplasmic cysts of the ovary arise from unchecked tissue growth and are related to a carcinoma, most frequently to ovarian cancer. Endometriomas, another type of mass, fill with blood from ectopic endometrium. Ovarian carcinomas occur in about 15 of every 100,000 patients; treatment involves removal of the cyst or ovaries, or a complete hysterectomy.

Signs and Symptoms

Pathologic Cysts

- Abdominal pain
- Vaginal bleeding
- Infertility

Transport Management

Pathologic Cysts

- Manage pain (administer morphine or fentanyl).

Another more common type of malignant cyst is a polycystic ovary; this is the case of an enlarged ovary with multiple cysts noted on the exterior. Insulin resistance (leading to diabetes), obesity, and hirsutism are associated signs and symptoms of polycystic ovary syndrome. Other signs of polycystic ovary syndrome include abdominal pain, vaginal bleeding, and infertility. Patients have little tolerance for extended transports, and CCTPs should consider pain management with morphine or fentanyl.

Ovarian Torsion

In some cases, an enlarged ovary, either by itself or as a result of another condition, will twist into a nonfunctional state; this is called **ovarian torsion**. Ovarian torsion is often a cause of lower abdominal pain in females that initially presents similar to appendicitis or an ectopic pregnancy **Figure 21-25**. Possible causes include increased ovarian weight secondary to blood retention from impaired venous drainage and congenital deformities. When not rapidly treated, this condition can lead to ovarian

infarction and loss of function. Pregnant, nonpregnant, and postmenopausal women are all at risk for ovarian torsion.

A gradual onset of diffuse lower abdominal pain, biased toward the affected side, is often seen in cases of ovarian torsion. Nausea and vomiting are common, along with either constipation or diarrhea. In approximately 50% of cases, a tender mass can be palpated at the torsion site.

It is important to immediately rule out the possibility of pregnancy and ovarian cysts prior to proceeding with specific treatment. The CCTP can initiate this process by taking a thorough history, including sexual history and the date of the patient's last menstrual cycle. Some clinicians may prefer that pain medications be withheld until diagnosis. Whether the CCTP can administer pain medication based on field impression will depend on local protocols. Pelvic ultrasonography is the preferred method of diagnosis, in which an enlarged ovary is typically observed. A computed tomographic scan and magnetic resonance imaging can serve as secondary diagnostic techniques when a sonogram is nonspecific. Laparoscopic oophoropexy is the most common treatment, in which the ovary is moved out of the distorted position.

Transport Management

Ovarian Torsion

- Manage pain (administer morphine or fentanyl).

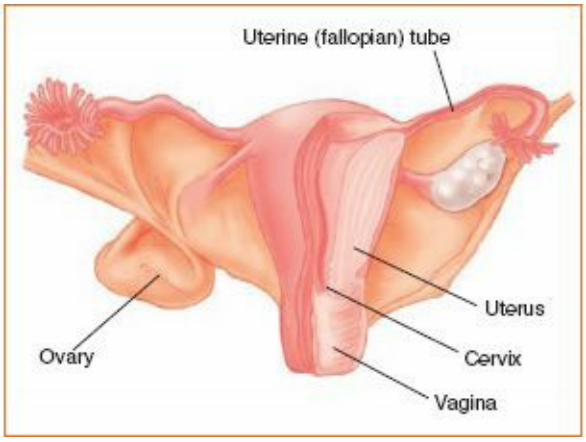


Figure 21-25 Ovarian torsion.

Pain management with morphine or fentanyl should be considered for long transports.

Gynecologic Trauma

Vulvular Hematoma

A hematoma can result from blunt trauma to the vulva, also known as a straddle injury. Purplish swelling occurs that is treated with ice and compression. Foley catheterization should be considered if the hematoma expands to compress the urethra, causing bladder outlet obstruction.

Signs and Symptoms

Vulvular Hematoma

- Purplish swelling of the vulva

Transport Management

Vulvular Hematoma

- Administer ice.
- Provide compression.
- Monitor the indwelling catheter, if one is placed.

Sexual Assault

After the patient's ABCs have been addressed, CCTPs must treat patients who have been sexually assaulted as a living crime scene. The focus should be on evidence preservation; do not allow patients to void or shower and limit any unnecessary treatments. Cover the patient with a sheet to capture trace evidence and ensure that a rape kit is requested.

Transport Management

Sexual Assault

- Address the ABCs.
- Preserve evidence: do not allow patients to void or shower and limit unnecessary treatments.
- Cover the patient with a sheet.

Flight Considerations

Delivery of an infant is absolutely the last procedure that you want to perform while in flight; therefore, a full patient assessment must be performed prior to launch to ensure that the patient is not showing signs of imminent delivery. In addition, the stress and noise of flight can cause anxiety to a pregnant patient and also result in long-term variability and fetal distress. Flight CCTPs should consider analgesics for these patients to ease the anxiety.

Summary

The dynamic changes that occur with pregnant patients are challenging for the CCTP. For a pregnant patient, each day is different physiologically as inside her a new life grows. Gynecologic emergencies do not require significantly different treatment during critical care transport, but as always, are important to treat properly and with sensitivity.

Case Study

A PREGNANT WOMAN AT 25½ WEEKS' GESTATION PRESENTED to a local hospital with a headache and complaints of extreme sensitivity to light and sound. The patient's vital signs were as follows: blood pressure, 154/104 mm Hg; pulse rate, 104 beats/min; and respiratory rate, 18 breaths/min. The patient was placed on the fetal heart monitor, an IV line was inserted, routine lab values were obtained, and a

24-hour urine protein test was ordered. The laboratory results were obtained and were unremarkable with the exception of the uric acid level, which was 7.2 mg/dL (normal, 2.5 to 6.2 mg/dL). The 24-hour urine protein determination was alarming at a total protein count of 333 mg/d (normal, 44 to 225 mg/d). The attending obstetrician informed the expectant mother that he thought she had a moderate case of preeclampsia. Because of its early onset, the patient needed to be transferred to a tertiary care center that specializes in high-risk obstetrics for further evaluation.

Your mobile intensive care unit is dispatched to the referring hospital to transport the patient to a tertiary care center located 70 miles away. Your report from the referring facility indicates that the patient's condition is stable. The patient has no complaints, has a saline lock in place, and is currently on room air. The hospital requests that you bring a fetal heart monitor with you because the fetus has experienced sporadic episodes of bradycardia. Your expected total transport time to the tertiary center is 90 minutes.

Upon your arrival at the referring facility, you are met by the staff nurse outside the patient's room. The nurse gives you a more detailed patient report. Your partner enters the room to begin assessment of the patient while you obtain more information from the nurse. The nurse tells you that this patient is a gravida 1, para 0, who is currently 25½ weeks' pregnant. At 18 weeks' gestation, mild elevations in her blood pressure developed accompanied by swelling in her face, feet, and hands. She was admitted to the obstetrics unit 24 hours ago after complaining of a headache associated with a blood pressure of 154/104 mm Hg, which was the highest reading so far during her pregnancy. Urinary output, between 35 and 55 mL/h, was positive for proteins and negative for blood. The glucose level was normal. The attending obstetrician initially suspected PIH. The patient was given one dose of dexamethasone intramuscularly shortly after admission to the labor and delivery unit to help develop the lungs of the fetus in the event of a preterm delivery. The fetal ultrasound showed an anterior placenta and no placenta previa. The patient reported having no drug allergies, taking only prenatal vitamins, and having a medical history of *Helicobacter pylori*, cytomegalovirus infections, and leukopenia.

Patient assessment reveals marked edema of the face, hands, ankles, and feet, which the patient reports has gotten progressively worse since the onset. The abdominal exam reveals a symmetric abdomen that is free from surgical scars with minor striae gravidarum noted and a normal fundal height of 26 cm. The patient tells you that a decrease in her urinary output began around the 20th week of pregnancy. The edema to the lower extremities is symmetric, and the patient denies having any pain associated with the swelling. An 18-gauge IV catheter is noted in the patient's left antecubital fossa with a saline lock. Deep tendon reflexes are normal without any signs of clonus. The fetal heart rate is noted to be 162 beats/min with the last deceleration 3 hours ago. Maternal vital signs are as follows: blood pressure, 121/70 mm Hg (mean arterial pressure, 87 mm Hg); heart rate, 78 beats/min with sinus rhythm; respiratory rate, 20 breaths/min; and SpO_2 98% on room air. Lung sounds are clear and equal bilaterally with symmetric chest wall expansion. Peripheral pulses are easily palpated bilaterally. The patient has been accepted by the obstetrician at the tertiary care center, and bed availability has been confirmed. You and your partner place the patient on the ambulance stretcher and prepare for transport. Prior to departure, you contact your online medical control to discuss the case. Your medical control physician is comfortable with the current treatment and does not recommend additional interventions. If changes are noted in the patient's condition or if frequent fetal decelerations are noted, the crew is to contact medical control for orders.

Approximately 25 minutes into the 90-minute transport, the patient begins to complain of nausea associated with mild frontal headache and dizziness. Vital signs are as follows: blood pressure, 98/52 mm Hg (mean arterial pressure, 67 mm Hg); pulse rate, 84 beats/min with sinus rhythm; respiratory rate, 20 breaths/min; and SpO_2, 98% on room air. You instruct your patient to lie on her left side and you place

her on 3 L/min of nasal oxygen while your partner administers 4 mg IV of ondansetron (Zofran) and begins a crystalloid infusion of 0.9% normal saline at 100 mL/h. Approximately 10 minutes later your patient reports that the nausea has subsided, but she still complains of a mild headache and is resting quietly in no apparent distress on the stretcher. No significant changes are detected on the fetal heart monitor.

Approximately 60 minutes into the 90-minute transport, your patient begins to complain of a sharp stabbing pain in the center of her chest that radiates to the right upper quadrant of her abdomen. She rates the severity of pain at onset as only a 5/10; however, you notice that she is getting anxious and is difficult to calm. Repeat vital signs are as follows: blood pressure, 164/110 mm Hg (mean arterial pressure, 128 mm Hg); pulse rate, 112 beats/min with sinus tachycardia; respiratory rate, 24 breaths/min; and Spo_2, 99% on 3 L of nasal oxygen. No obvious changes are noted on the fetal heart monitor, with a current heart rate of 170 beats/min. You place your patient on 15 L of oxygen via a nonrebreathing mask. Approximately 10 minutes later, your patient is writhing in pain and is barely able to stay secured to the stretcher as a result of her extreme anxiety. Your patient says, "It hurts so bad.… I just can't get comfortable." She now rates the severity of her headache and her chest and abdominal pain as a 10/10. Vital signs are as follows: blood pressure, 210/112 mm Hg (mean arterial pressure, 144 mm Hg); pulse rate, 121 beats/min with sinus tachycardia; respiratory rate, 28 breaths/min; and Spo_2, 98% on 15 L of oxygen via nonrebreathing mask.

You immediately contact online medical control and give them an update on your patient's condition. The physician requests the administration of 20 mg of nifedipine orally, but you advise him that nifedipine is not available to you. Orders are then received for 10 mg IV of labetalol with another dose of 20 mg IV in 10 minutes if the mean arterial pressure remains greater than 130 mm Hg. You are also ordered to administer 4 g of magnesium sulfate IV over 5 to 10 minutes and then begin an infusion of 2 g/h. All orders are read back and verified. You advise online medical control that you are 20 minutes from your destination, and you request that a patient update is passed along to the receiving facility.

Five minutes after administering labetalol, your patient's condition remains unchanged. She is still extremely anxious with the following vital signs: blood pressure, 222/120 mm Hg (mean arterial pressure, 154 mm Hg); pulse rate, 124 beats/min with sinus tachycardia; respiratory rate, 28 breaths/min; and Spo_2, 97% on 15 L of oxygen via nonrebreathing mask. Your partner establishes a second IV line with an 18-gauge needle in the right antecubital fossa and begins the magnesium sulfate loading dose of 4 g. It is now 10 minutes after her first dose of labetalol was administered and her condition remains unchanged. Her vital signs are as follows: blood pressure, 218/122 mm Hg (mean arterial pressure, 154 mm Hg); pulse rate, 128 beats/min with sinus tachycardia; respiratory rate, 28 breaths/min; and Spo_2, 97% on 15 L of oxygen via nonrebreathing mask. You administer 20 mg IV of labetalol for persistent hypertension related to severe preeclampsia; after 10 minutes, there is no change in your patient's condition.

You arrive at the receiving facility where a team of obstetricians and neonatologists who specialize in high-risk pregnancies are waiting for you. You and your partner provide a detailed report to the awaiting team along with a copy of the patient's health care record. The patient is quickly assessed by the team, and it is determined that an emergency cesarean section is needed.

1. What was the likely cause of this patient's deteriorating clinical picture and severe pain during transport?
2. What is the most common complication associated with preeclampsia?
3. Does the presence of pregnancy-induced hypertension in this patient make her more likely to have a recurrence in future pregnancies?

Analysis

The patient was diagnosed with preeclampsia after testing positive for proteinuria, hypertension, and elevated uric acid levels. The conditions of a small percentage of women (10%) who are diagnosed with preeclampsia worsen, resulting in a condition that is referred to as HELLP syndrome, which carries a high mortality rate. The patient's symptoms developed rapidly without warning and required an emergency cesarean section. The only cure for HELLP syndrome is the immediate delivery of the fetus.

In this case, the transport crew immediately contacted the medical control physician to obtain orders for the patient's worsening condition. Most transport programs should already have an aggressive set of protocols available that allows them to begin treatment of this condition without first contacting medical control. In order to save time during transport, the transport personnel should have asked for "what if" orders from the control physician while at the referring facility to eliminate the delay in treating the patient, therefore giving the patient and fetus a better chance of survival.

The goal of preeclampsia treatment is to avoid the progression and worsening of the symptoms related to the disease process. The patient's symptoms became markedly worse, requiring immediate treatment of life-threatening hypertension. Prompt treatment of the patient's signs and symptoms is the key as disease progression increases morbidity and mortality rates. The interventions performed were only temporary measures because an immediate cesarean delivery was indicated.

The most commonly seen complication associated with preeclampsia is eclampsia. Eclampsia is a disorder marked by the occurrence of seizures during pregnancy that are directly related to hypertension. Eclampsia is also known for having a high mortality rate and often requires a magnesium sulfate infusion to assist in stopping the seizure activity. In the case study, a magnesium sulfate infusion was initiated prophylactically in order to prevent seizures from occurring and was continued throughout the remainder of the transport. The magnesium sulfate infusion should be maintained until the blood pressure returns to normal and the threat of seizures has diminished.

The presence of pregnancy-induced hypertension does make a woman more susceptible for recurrence in future pregnancies. After this patient recovered from her emergency cesarean section, an array of laboratory tests were performed on an outpatient basis to determine a cause for the preeclampsia. Studies have shown that the exact cause of preeclampsia is unknown, but some of the likely suspects are clotting disorders and chromosomal abnormalities. The detailed laboratory studies revealed that the patient had a thrombophilia disorder called factor V Leiden. This disorder often causes small clots to form inside the placenta, which in turn causes the HELLP syndrome and accounts for a 60% recurrence of preeclampsia in future pregnancies.

Prep Kit

Ready for Review

- The physiologic changes induced by pregnancy and the presence of potential life-threatening pathologic conditions can make management of obstetric patients difficult.
- The female reproductive system, regulated by hormonal secretions, undergoes an approximately 28-day menstrual cycle.
- The placenta provides the fetus with oxygen and vital nutrients. It grows with the fetus and includes a thin membrane that separates maternal and fetal blood.
- As the fetus grows, it is more vulnerable to trauma as the ratio of fetal size to amniotic fluid decreases.
- Pregnancy alters the normal physiology of females, affecting nearly every organ system.

- During pregnancy, cardiac output increases, circulating blood volume expands, and peripheral venous pressure increases. Blood pressure and levels of serum albumin and serum protein decrease as pregnancy progresses.
- The fetus increases the demand for oxygen and reduces the oxygen reserve in the pregnant woman.
- Gastroesophageal reflux is more common during pregnancy, and the likelihood of aspiration in a pregnant patient with an altered level of consciousness is greater.
- Pregnancy places increased strain on the renal and endocrine systems.
- Hormonal changes in the pregnant patient can cause dermatologic changes, including acne, linea negra, chloasma, and rashes.
- Obstetric patients require a complete physical exam, including maternal and fetal hemodynamic monitoring and, if in labor, external vaginal evaluation for the presence of crowning.
- If delivery is imminent, CCTPs should choose to assist in the delivery at the hospital rather than during transport.
- To ease anxiety caused by the stress and noise of transport, analgesics may be administered to patients in labor.
- When necessary, CPR may be performed on pregnant patients, following standard guidelines with constant chest compressions and limited interruptions for ventilation and gas exchange.
- If defibrillation is necessary, fetal monitors should be removed before administering a shock.
- The developing fetus has only a small reserve of oxygen, usually only 1 to 2 minutes, if the supply from the mother is cut off or diminished. If the heart of the fetus does not receive enough oxygen, the heart rate decreases.
- The well-being of the fetus may be assessed by auscultation for heart rate and movement, use of a hand-held Doppler or fetal stethoscope, and electronic fetal monitoring.
- Electronic fetal monitoring records the fetal heart rate and uterine contractions on a fetal monitoring strip. Late or variable decelerations in the heart rate are abnormal and may indicate fetal distress.
- The normal heart rate of a fetus is between 120 and 160 beats/min. Fetal heart tones should be at least 30 seconds long and performed and recorded every 15 minutes during labor. During high-risk transports, electronic fetal monitoring should be performed continuously.
- Any vaginal bleeding during the third trimester of pregnancy must be regarded as an emergency.
- Bleeding may indicate a spontaneous abortion (miscarriage), ectopic pregnancy, abruptio placenta (premature separation of the placenta from the uterine wall), placenta previa (placenta that covers the cervical canal), or uterine rupture.
- CCTPs should be aware of the warning signs of dangerous medical conditions, such as pregnancy-induced hypertension, preeclampsia, eclampsia, and HELLP syndrome, and the appropriate treatment protocols.
- Labor includes three distinct stages as the cervix dilates, the baby descends through the birth canal and is born, and the placenta is delivered.
- During delivery, a crucial fetal transition occurs as the baby begins to breathe independently of an oxygen supply from the mother. Anything that delays the decline in pulmonary pressure during fetal transition can lead to delayed transition, hypoxia, brain damage, and death.
- Preterm labor and fetal immaturity are the leading causes of neonatal mortality.
- Tocolytic agents such as terbutaline and magnesium sulfate may be used to help stop preterm labor.

- The baby may present in vertex, Frank breech, complete breech, incomplete breech, or footling breech positions for birth. Only with vertex and footling breech presentations should CCTPs consider delivery in the field.
- In the event of a prolapsed cord, the pregnant patient should be given oxygen, placed in the knee to chest position, and transported rapidly.
- If shoulder dystocia occurs (the infant is too large to travel through the birth canal unassisted), a rapid treatment plan is needed, which may include positioning the pregnant patient using the McRoberts maneuver or rotating the infant's arm toward its sternum during delivery.
- During the delay between multiple births (such as twins or triplets), transport should continue. Transport should only be interrupted if the next infant begins to emerge from the birth canal.
- Once delivery is complete, care for both the mother and infant should continue by examining the mother's perineum for tears and monitoring the infant's APGAR score.
- CCTPs must examine the antepartum patient for possible postpartum hemorrhage, amniotic fluid embolism, complications arising from Rh incompatibility, or uterine rupture.
- All women of childbearing age with sudden onset of abdominal pain or uncommon vaginal bleeding must be considered to have a possible ectopic pregnancy.
- Pelvic inflammatory disease causes inflammation to the reproductive system, including the uterus, fallopian tubes, and ovaries, and, if untreated, may cause infertility.
- CCTPs should be prepared to treat gynecologic emergencies such as toxic shock syndrome, ovarian cysts, ruptured ovarian cysts, pathologic cysts, and ovarian torsion.
- Ovarian torsion is often a cause of lower abdominal pain in females that initially presents similar to appendicitis or an ectopic pregnancy.
- Ovarian cysts are typically a result of follicular maturation during the normal female menstrual cycle and are often undetectable and self-resolving. A small percentage may continue to grow and may cause lower abdominal pain.
- Symptoms of a cyst include abdominal pain, nausea, vomiting, and frequent urination from bladder pressure.
- Ovarian cyst diagnosis is made through ultrasonography and Doppler flow studies that can monitor blood flow as well as cyst size and location.
- An ovarian cyst may rupture spontaneously or following trauma, resulting in hemorrhage. If hemorrhage is present, patients may present with increased lower abdominal and pelvic pain, vaginal bleeding, and hypovolemia leading to shock.
- Treatment for a patient with a ruptured ovarian cyst includes controlling any immediate life threats, including evaluation for hypovolemic shock and maintaining adequate blood pressure with isotonic fluids if the patient is in shock.
- Gynecologic trauma includes vulvular hematoma (straddle injury) and sexual assault. The scene of an assault should be treated as a living crime scene, with efforts made to preserve evidence and limit unnecessary treatments.
- Gynecologic emergencies do not require significantly different treatment during critical care transport but are important to treat properly and with sensitivity.

Vital Vocabulary

amniotic fluid embolism A rare condition that can occur antepartum and postpartum, in which fetal tissue crosses over the placental barrier into maternal circulation, thereby causing obstruction to the mother's organs with organ failure or respiratory and cardiovascular compromise.

breech A delivery presentation in which the buttocks emerge first.

complete breech A delivery presentation in which the buttocks present first and the legs are crossed inside the uterus, oriented transversely to the birth canal; in this case, the mother is unable to vaginally deliver.

dilation Opening of the cervix to a maximum of 10 cm, at which point it is considered complete.

dilation and curettage (D&C) A surgical procedure in which the cervix is opened and the uterus scraped of tissue.

eclampsia The condition that exists once a patient with preeclampsia develops seizures; warning signs of the transition include severe headache, scotomata, hyperreflexia, epigastric pain, and anxiety; the condition causes serious maternal complications.

ectopic pregnancy A pregnancy in which the ovum implants somewhere other than the uterine endometrium.

endometrium The inner mucous membrane of the uterus.

external fetal monitoring Electronic heart monitoring of the fetus while in utero, performed via electrodes on the pregnant woman's abdomen.

footling breech A delivery presentation in which both feet extend into the birth canal; this is the only malpresentation in which field delivery may be considered.

frank breech A delivery presentation in which the buttocks present first, both of the infant's hips are flexed, and the feet are near the fetus' head.

gestation Period of time elapsed from conception to birth. For humans, the full period is normally 40 weeks.

gravida The number of pregnancies a woman has had, including those not carried to term.

HELLP syndrome A hemolytic disorder that occurs during the latter stages of gestation, usually after the 20th week, and whose clinical findings include hemolytic anemia, elevated liver enzyme levels, and a low platelet count; patients will present with blurred vision, abdominal pain, headache, and edema.

hydroureteronephrosis Dilation and obstruction of a ureter.

hyperreflexia A condition of overactive reflexes that can involve excessive twitching or spasms.

incomplete breech A delivery presentation in which the buttocks present first and one foot extends into the birth canal.

long-term variability A pattern of changes in the baseline fetal heart rate occurring within 30-second intervals rather than from beat to beat; due to the parasympathetic-sympathetic interplay in the fetal heart rate.

mittelschmerz pelvic pain Abdominal pain occurring midway between menstrual periods, at the time of ovulation and from the ovulation site.

ovarian cyst A fluid buildup within an outcropping of tissue from the ovary. If such a cyst is ruptured, significant bleeding can occur.

ovarian torsion Twisting of an ovary about its ligaments, resulting in ischemia and possibly necrosis.

parity The number of pregnancies a woman has had that were carried to more than 28 weeks' gestation.

partial thromboplastin time (PTT) A value that represents the intrinsic coagulation pathway's clotting ability; also known as activated partial thromboplastin time (aPTT).

pathologic edema Systemic swelling, which may have several causes, including preeclampsia.

pelvic inflammatory disease (PID) An infection of the female upper organs of reproduction, specifically the uterus, ovaries, and fallopian tubes.

preeclampsia A condition of late pregnancy that involves gradual onset of hypertension, headache, visual changes, and swelling of the hands and feet; also called toxemia of pregnancy.

pregnancy-induced hypertension (PIH) The occurrence of hypertension in a pregnant woman, usually after 20 weeks' gestation, defined as a blood pressure of 140/90 mm Hg or greater in a woman with a previously normal blood pressure, or a rise of 30 mm Hg systolic and/or 15 mm Hg diastolic in a woman with baseline hypertension; can lead to preeclampsia if uncontrolled.

premature Underdeveloped; refers to infants born before 36 weeks from the first day of the last menstrual period.

preterm labor Labor occurring before 36 weeks' gestation, leading to early birth.

proteinuria Protein levels detected in the urine.

prothrombin time (PT) A value that represents the extrinsic coagulation pathway's clotting ability by taking into account various clotting factors, fibrinogen, the prothrombin ratio, and the international normalized ratio.

shoulder dystocia A condition in which the infant's shoulders are too large to travel through the birth canal without additional clinical maneuvering of the infant.

spontaneous abortion Expulsion of the fetus that occurs naturally; also called miscarriage.

supine hypotensive syndrome Low blood pressure resulting from compression of the inferior vena cava by the weight of the pregnant uterus when the mother is supine.

term Used to describe an infant delivered at 37 to 42 weeks of gestation.

thrombocytopenia Reduction in the number of platelets.

tocolytics A group of medications used to suppress preterm labor.

vasospastic disease Vascular constriction or spasm.

vertex A fetal position in which the head is the lowest part of the fetus, resulting in the head presenting first during delivery; this is the normal delivery presentation.

Bibliography

Anderson JM, Etches D. Prevention and management of postpartum hemorrhage. *Am Fam Phys*. 2007;75(6):875–882.

Ayres S, Grenvik A, Holbrook P, Shoemaker W. *Textbook of Critical Care* , 3rd ed. Philadelphia, PA: WB Saunders Co; 1995.

Capeless E. Cardiovascular changes in early phase of pregnancy. *Am J Obstet Gynecol*. 1989;161(6):1449–1453.

Caroline N. *Nancy Caroline's Emergency Care in the Streets* , 6th ed. Sudbury, MA: Jones and Bartlett Publishers; 2008.

Centers for Disease Control and Prevention. Pelvic Inflammatory Disease—CDC Fact Sheet. Available at: http://www.cdc.gov/std/PID/STDFact-PID.htm. Accessed December 31, 2008.

Chames M, Pearlman M. Trauma during pregnancy: outcomes and clinical management. *Clin Obstet Gynecol*. 2008;51:398-408.

Clark SL, Cotton DB, Lee W, et al. Central hemodynamic assessment of normal term pregnancy. *Am J Obstet Gynecol*. 1989;161(6):1439–1442.

Cleary V, Wilson P, Super G. *Prehospital Care: Administrative and Clinical Management*. Rockville, MD: Aspen Publishers; 1987.

Cunningham F, MacDonald G, Paul C, Gant NF. *Williams Obstetrics*, 8th ed. Norwalk, CT: Appleton & Lang; 1989.

Datner E, Promes S. Resuscitation in pregnancy. In: Tintinalli JE, Kelen GD, Stapczynski MD, eds. *Emergency Medicine: A Comprehensive Study Guide*. New York, NY: McGraw-Hill; 2006:254.

eMedicineHealth. Ovarian cysts. Available at: http://www.emedicinehealth.com/ovarian_cysts/article_em.htm. Accessed December 31, 2008.

Farrell R. *OB/GYN Emergencies: The First 60 Minutes*. Rockville, MD: Aspen Publishers; 1986.

Guyton A, Hall J. *Textbook of Medical Physiology,* 11th ed. Philadelphia, PA: WB Saunders Co; 2006.

Gyetvai K, Hannah ME, Hodnett ED, et al. Tocolytics for preterm labor: a systematic review. *Obstet Gynecol*. 1999;94(5) (part 2):869–877.

Kaaja RJ, Greer IA. Manifestations of chronic disease during pregnancy. *JAMA*. 2005;294:2751–2757.

Kass EH. Asymptomatic infection of urinary tract. *Trans Assoc Am Phys*. 1956;69:56–63.

Kish K, Collea JV. Malpresentation and cord prolapse. In: DeCherney AH, Nathan L, eds. *Current Obstetric & Gynecologic Diagnosis & Treatment*, 9th ed. New York, NY: Lange/McGraw-Hill; 2003:369–386.

Lattanzi DR, Cook WA. Urinary calculi in pregnancy. *Obstet Gynecol*. 1980;56:462–466. Mastrobattista J. Therapeutic agents in preterm labor: steroids.

Clin Obstet Gynecol. 2000;43(4):802–808. Marx JA, Hockberger RS, Walls RM. *Rosen's Emergency Medicine*

Concepts and Clinical Practice, 5th ed. St. Louis, MO: Mosby, Inc; 2002.

Moore J, Baldisseri MR. Amniotic fluid embolism. *Crit Care Med*. 2005;33(10 suppl):S279–S285.

Moore K. *The Developing Human: Clinically Oriented Embryology*, 4th ed. Philadelphia, PA: WB Saunders Co; 1988.

Oxorn H. *Human Labor & Birth*, 5th ed. Norwalk, CT: Appleton-Century-Croft; 1986.

Peters CW, Layon AJ, Edwards RK. Cardiac arrest during pregnancy. *J Clin Anesth*. 2006;50:27–28.

Sodre PM. Obstetrics and gynecology: maternal physiology changes during pregnancy. Available at: http://www.medstudents.com.br/ginob/ginob5.htm. Accessed February 12, 2009.

Stanton SL, Kerr-Wilson R, Grant Harris V. The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynecol*. 1980;87:897–900.

Strassmann BI. The evolution of endometrial cycles and menstruation. *Q Rev Biol*. 1996;71(2):181–220.

Tintinalli J, Kellen G, Stapczynski J. *Emergency Medicine: A Comprehensive Study Guide*, 5th ed. New York, NY: McGraw-Hill; 2000.

Vegetti W. FSH and follucogenesis: from physiology to ovarian stimulation. *Reprod Biomed Online*.

2006;12(6):684–694.

Whittaker P, Macphail S, Lind T. Serial hematologic changes and pregnancy outcome. *Obstet Gynecol*. 1996;88(1):33–39.


REGA 1414

Neonatal Emergencies

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Objectives

1. Define the terms newborn, neonate, and term newborn (p 876).
2. Discuss the roles of the CCTP when caring for a neonate (p 876).
3. Recognize anatomy and physiology unique to a neonate, including differences in thermoregulation, respiratory structure and function, oxygen transport, cardiovascular function, renal function, fluid and electrolyte balance, central nervous system, and the skeletal system (p 877).
4. Understand how problems with transitional circulation can result in neonatal emergencies (p 877–879).
5. Describe developmental aspects of pain in the neonate (p 879).
6. Discuss anatomic and physiologic differences in the premature infant and how these relate to management (p 881).
7. Discuss medical complications for which late preterm infants are at risk (p 881).
8. Identify important antepartum and intrapartum factors that can affect labor, delivery, and the neonate, and understand the pathophysiology that is associated with these factors (p 880).
9. Identify when a CCTP would use the APGAR score in caring for a newborn (p 880).
10. Discuss neonatal assessment, stabilization, and management (p 880).
11. Discuss how to prepare for and provide neonatal resuscitation, and identify situations in which it should be performed (p 881).
12. Explain the initial steps in resuscitation of a neonate (p 881).
13. Describe methods that can be used to improve airway and breathing in a neonate with inadequate respiration (p 882).
14. Determine when vascular access is indicated for a neonate and appropriate fluid selection (p 888–889).
15. Describe appropriate assessment technique for examining a neonate (p 879).
16. Describe free-flow oxygen delivery and appropriate assisted ventilation for a neonate (p 882).
17. Describe appropriate endotracheal intubation technique for a neonate (p 885–886).
18. Determine when an orogastric tube should be inserted, and describe how to insert an orogastric tube in a neonate (p 886).
19. Describe appropriate chest compression and ventilation technique for a neonate (p 888).
20. Discuss indications for medications, dosage, and routes of administration for a neonate (p 890–891).
21. Discuss the use of ventilators during neonatal transports (p 889).

22. Distinguish between primary and secondary apnea, and list causes, assessment, and management (p 892).
23. Describe how to perform needle thoracentesis for pneumothorax decompression for a neonate (p 895).
24. Discuss the pathophysiology, assessment findings, management, and treatment plan of the following respiratory emergencies in a neonate: meconium aspiration, apnea, pneumonia, pneumothorax, respiratory distress, and respiratory failure (p 893–896).
25. Recognize congenital anomalies that may lead to compromise of the neonate (p 897).
26. Discuss the pathophysiology, assessment findings, and management of the following cardiovascular emergencies in a neonate: cyanosis, tachyarrhythmias, and bradyarrhythmias, bradycardia, cardiac arrest, persistent pulmonary hypertension, acidosis, shock, and anemia (p 896–902).
27. Describe how to perform umbilical vein catheterization in a neonate (p 900).
28. Discuss the pathophysiology, assessment findings, and management of the following gastrointestinal emergencies in a neonate: gastroschisis, omphalocele, gastrointestinal obstruction and vomiting, acute intestinal perforation, hematemesis and bleeding from the rectum, volvulus, intussusception, and diarrhea (p 902–907).
29. Discuss management of infectious diseases and sepsis in the neonate (p 907).
30. Discuss management of hyperthermia and hypothermia in the neonate (p 908).
31. Discuss management of toxic exposure in the neonate, including the appropriate treatment for the neonate with narcotic depression (p 908).
32. Discuss the pathophysiology, assessment findings, and management of the following trauma/birth injuries in the neonate: head and neck injuries, nerve injuries, bone injuries, and abuse/maltreatment (p 908–910).
33. Discuss the pathophysiology, assessment findings, and management of the following neurologic conditions in the neonate: seizures, hypoxic ischemic encephalopathy, and lethargy (p 910–911).
34. Discuss the pathophysiology, assessment findings, and management of the following metabolic conditions in the neonate: hypoglycemia, hypocalcemia, and inborn errors of metabolism (p 912–913).
35. Discuss the use of an incubator (p 913).
36. Recognize the emotional impact and the need for information, empathy, and compassion for the parent/guardian during a critical care transport (p 913–914).
37. Discuss risks associated with critical care transport, including factors unique to air transport of a neonate (p 914–915).

Introduction

The care of a newborn or neonate must be tailored to meet the unique needs of this population. A **newborn** refers to an infant within the first few hours after birth; a **neonate** refers to an infant within the first 28 days after birth **Table 22-1**. A **term newborn** is one delivered between 37 and 42 weeks' **gestation**. This chapter reviews challenges that the CCTP may face when transporting a neonate, including recognizing anatomic differences in a neonate, understanding the physiologic changes that occur in a newborn during transition from fetal circulation after birth, thermoregulation, and the special needs of

infants delivered prematurely or births complicated by other factors. The steps involved in neonatal resuscitation, patho-physiology of common congenital anomalies, medical emergencies encountered during the first month after birth, and how these factors affect the process of transporting a critically ill infant are also discussed.

Additional skilled care intervention (intubation, obtaining peripheral IV access, placement of umbilical venous and arterial lines, needle aspiration of the chest, and chest tube placement) is needed for approximately 6% of neonates, with the rate of complications increasing as the neonate's birth weight and gestational age decrease. In the United States, approximately 80% of the 30,000 babies born each year weighing less than 3 lb (1,500 g) require resuscitation. Because both short- and long-term outcomes in neonates have been linked to initial stabilization efforts, it is imperative that CCTPs anticipate problems with neonates, are knowledgeable about how to deal with them, have the appropriate resuscitation equipment readily available, and carefully consider the neonate's ultimate transport plan.

TABLE 22-1 Infant Age Terminology

Term	Age
Newborn	Within the first few hours after birth
Neonate	Within the first 28 days after birth
Infant	Age 1 month to 1 year
Term newborn	Delivered between 37 and 42 weeks' gestation
Preterm	Delivered before 37 weeks' gestation

Anatomy and Physiology of a Neonate

Neonates are not small children. Newborn infants have anatomic differences and undergo unique physiologic changes at birth, placing them at special risks.

Thermoregulation

Neonates become cold easily due to a variety of factors. The potential for heat loss is greater due to a high body surface area-to-body weight ratio and limited subcutaneous fat. Neonates also lose heat to the environment through the mechanisms of thermal conduction, convection, radiation, and increased evaporative heat loss through the skin. Heat production is limited due to low glycogen stores and brown fat, especially in premature infants (brown fat is used in older individuals as a heat energy source), and limited capacity to generate heat by shivering. Neonates have a narrow range of *neutral thermal environment*, the temperature at which maintenance of normal body temperature requires only minimal metabolic expenditure. The term newborn is able to increase heat production through brown fat metabolism (nonshivering thermogenesis); however, this is at the expense of increased oxygen consumption. Hypothermia is associated with hypoxia, prolonged coagulation time with reduced platelet function and increased risk of intraventricular hemorrhage, reduced drug metabolism, cerebral depression, myocardial depression, acidosis, decreased immunity, patient discomfort, and increased mortality. The preterm infant is particularly vulnerable because the immature skin is thin, allowing significant heat (and evaporative fluid) losses. To help maintain normothermia, CCTPs should use prewarmed blankets and equipment, including a prewarmed transport **incubator** Figure 22-1, place a cap on the infant's head, and minimize the infant's exposure to cold. It is also important to avoid

environmentally induced hyperthermia because this condition is associated with increased energy expenditure, morbidity, and mortality.

Respiratory Structure and Function

Lung development starts early in embryonic life, but the lungs are unable to sustain life *ex utero* until later in the canalicular stage of development, after approximately 23 weeks' gestation or 500 g. The neonatal lung is small relative to the body size, with little respiratory reserve. Surfactant is present by 23 weeks of gestation, but surfactant deficiency is common before 32 weeks; babies born prematurely may benefit from exogenous surfactant delivered endotracheally. The neonate has a respiratory rate of 30 to 60 breaths/min with a tidal volume of about 5 to 7 mL/kg. Their heads are large in relation to their bodies, resulting in neck flexion and obstruction in the supine position. Placing a rolled towel under the neonate's shoulders helps open up the airway. The infant's tongue occupies a larger intraoral volume and can lead to airway obstruction. Newborn infants are obligate nose breathers and may present with obstructive apnea when the nasal passages are blocked by a developmental defect (choanal atresia), edema of the nasal mucosa by vigorous suctioning, or secretions. Placing and maintaining an oral airway is often a sufficient intervention to stabilize a neonate with a fixed nasal obstruction during transport. Another important airway anatomic feature of neonates is a relatively more cephalad and anterior larynx, which needs to be considered when locating the vocal cords during intubation. When positive-pressure ventilation (PPV) is needed, attempts should be made to maintain physiologic tidal volume breaths because volutrauma, hypercapnia, and hypocapnia have been associated with bleeding in the brain (intraventricular hemorrhage), hearing loss, and chronic lung disease or bronchopulmonary dysplasia.

Figure 22-1 A transport incubator.

Oxygen Transport

Fetal hemoglobin (HbF) forms 70% to 80% of the total hemoglobin in the neonate. Fetal hemoglobin delivers oxygen to the tissues in the hypoxic condition *in utero*, but tends to hold on to oxygen in the normal conditions after birth (the oxygen dissociation curve is shifted to the left). Neonates have a high oxygen requirement (6 to 8 mL/kg/min vs 4 to 6 mL/kg/min in adults). Tissue oxygenation is achieved by a relatively high cardiac output (300 mL/kg/min vs 60 to 80 mL/kg/min in adults), high heart rate (120 to 160 beats/min), and high respiratory rate (30 to 40 breaths/min). Neonates do not tolerate interruption in oxygen delivery for any length of time and become hypoxic and bradycardic quickly.

Cardiovascular Function

The heart rate, normally 120 to 160 beats/min in neonates, is an important determinant of cardiac output because the cardiac muscle is immature. Neonates respond to careful volume loading (fluid bolus of 10 to

20 mL/kg) with an increase in cardiac output, but they do not tolerate fluid overload. The two most common inotropic agents used in neonates are dopamine, used when an infant has cardiac compromise and is peripherally vasodilated from septic shock, and dobutamine, which also improves cardiac function but can exacerbate hypotension due to peripheral vasodilation. **Table 22-2** summarizes newborn, neonate, and term newborn vital signs.

Transitional Circulation

In utero the pulmonary vasculature has a high resistance, so most of the blood returning to the right atrium from the placenta (oxygenated blood) passes to the left atrium via the foramen ovale **Figure 22-2**. The oxygenated blood enters the left atrium, mixes with the small pulmonary return, and is pumped via the left ventricle and the aorta to the coronary arteries and the brain. Deoxygenated blood returns from the lungs to the left atrium and mixes with the oxygenated blood in the left atrium. Of the ejected right ventricular output, most blood flows to the aorta via the ductus arteriosus, mixing with the left ventricular output. Blood carried by the descending aorta perfuses various organs and the lower body. Umbilical arteries, which branch from the internal iliac arteries, lead to the placenta where blood is oxygenated.

With the clamping of the umbilical cord at birth (removal of the low resistance placenta), there is a sudden rise in the systemic vascular resistance. Cardiorespiratory adaptation at birth results in an increase in pulmonary blood flow and physiologic closure of the fetal shunts that allowed the blood to bypass the lungs—the foramen ovale and ductus arteriosus.

TABLE 22-2 Normal Infant Vital Signs

Age	Respiratory Rate (breaths/min)	Pulse Rate (beats/min)	Minimal Systolic Blood Pressure (mm Hg)
Newborn	30-60	120-160	Varies (target mean blood pressure equals gestational age)
Neonate	30-40	120-160	> 60
Infant	25-50	70-160	> 70

With extrauterine breathing and expansion of the lungs, tissue oxygenation increases, the pulmonary vascular resistance decreases, and pulmonary blood flow increases. The increased pulmonary venous return to the left atrium raises the left atrial pressure above the right, closing the flap valve of the foramen ovale. With increasing left ventricular pressure and a fall in right ventricular pressures, there is a decrease or reversal of flow through the ductus arteriosus. Anatomic closure may occur as early as 6 hours after birth, but complete occlusion may take up to 6 weeks. Pulmonary vascular resistance may increase with hypoxia, acidosis, cold stress, primary pulmonary disease such as meconium aspiration or pneumonia, or hypercarbia, causing persistent pulmonary hypertension, the return of left-to-right shunting through the ductus arteriosus and/or foramen ovale, profound hypoxia and cardiovascular compromise, and sometimes death. This process is worsened if the patient has systemic hypotension. Optimizing a neonate's condition during transport, with special attention given to maintaining normothermia, optimal oxygenation, fluid status, and systemic blood pressure, and minimizing acidosis and noise decreases the risk of developing severe persistent pulmonary hypertension.

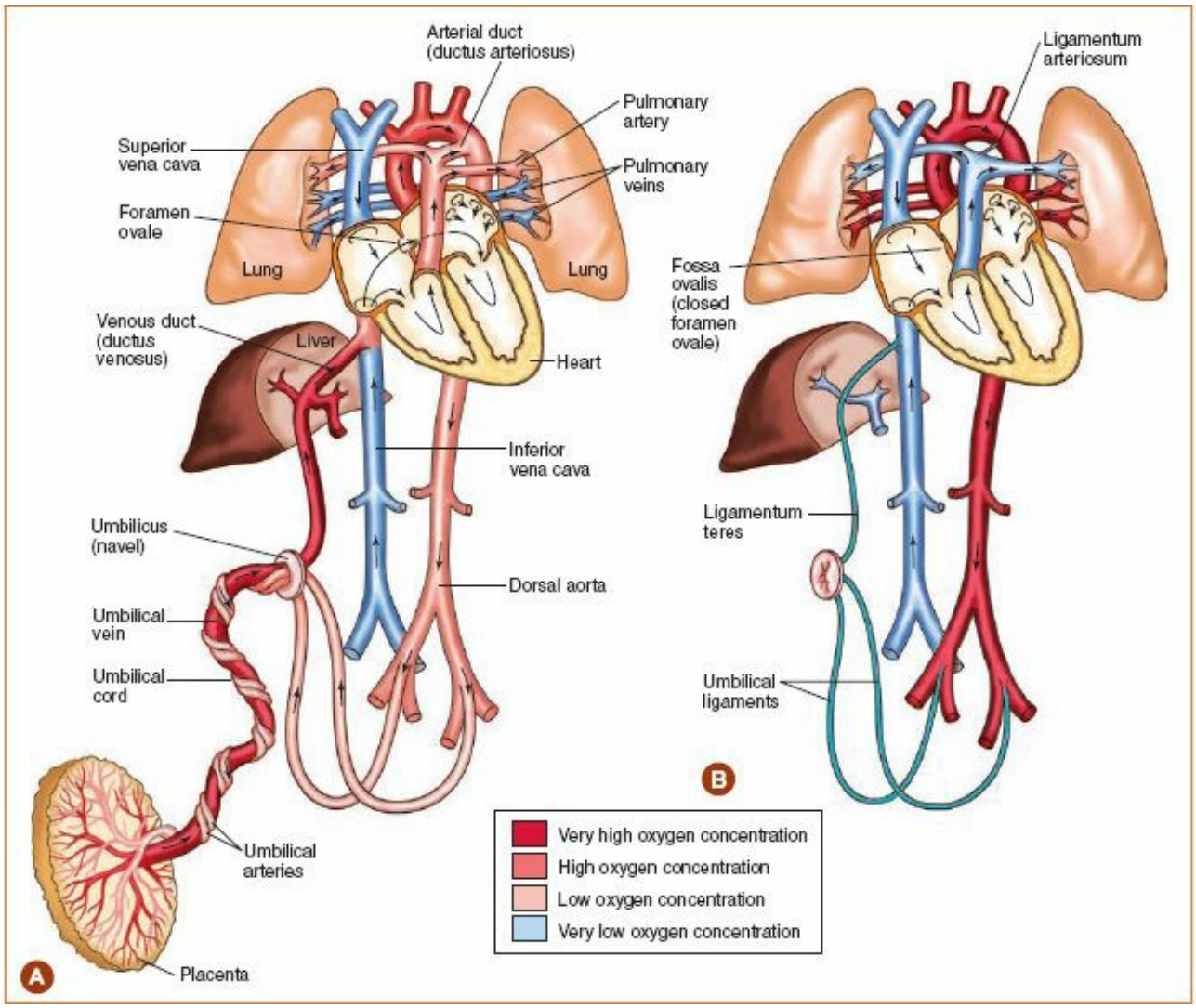


Figure 22-2 Fetal circulation. **A.** Oxygenated blood from the placenta reaches the fetus through the umbilical vein. Blood returns to the placenta via two umbilical arteries. Right-to-left shunts occur at the foramen ovale and the ductus arteriosus. **B.** Fetal circulation following transition.

Patent ductus arteriosus is seen in 50% of extremely premature infants (< 26 weeks' gestation) and is a risk factor for intraventricular hemorrhage and necrotizing enterocolitis. Whereas immediate treatment for a patent ductus arteriosus during critical care transport is not indicated, minimizing fluid overload during transport can decrease the risk of a persistent symptomatic patent ductus arteriosus.

Some cardiac abnormalities depend on a patent ductal flow, and circulatory collapse occurs with the closure of the ductus, often at 2 to 7 days of age. Maintenance of ductal patency with prostaglandins during transport can be lifesaving and will be discussed further in the section on cyanotic congenital heart disease.

Renal Function

Nephrogenesis is completed at 36 weeks' gestation and no further nephrons are produced. Additional increases in renal mass are the result of the growth of tubules. The glomerular filtration rate at term is low and reaches adult indexed values only at 2 years of age. The newborn's creatinine level at birth reflects the mother's creatinine level and falls to reflect renal function by 1 week of age. Tubular function matures over the first few months of life; infants usually produce urine that is isotonic to plasma, but if required, can concentrate their urine to achieve an osmolality of 500 to 700 mOsm/kg H_2O. Adult values (urinary osmolality, typically 1,200 to 1,400 mOsm/kg H_2O) are reached by 1 year of age. Infants tolerate fluid

restriction poorly and become dehydrated quickly.

Fluid and Electrolyte Balance

The extracellular fluid compartment is expanded in neonates, with total body water representing 85% of body weight in premature babies, and 75% of body weight in term babies, compared to 60% of body weight in adults. Contraction of the extracellular fluid compartment and weight loss in the first few days after birth is a normal physiologic process, due in part to diuresis induced by atrial natriuretic peptide secondary to increased pulmonary blood flow and stretching of the left atrial receptors. After this period of negative water and sodium balance, water and sodium requirements increase to match those of the growing infant. Therefore, fluids should be restricted until the postnatal weight loss has occurred. Liberal fluid regimens in the first few days of life have been shown to be associated with worse outcomes in premature infants (increased patent ductus arteriosus, necrotizing enterocolitis, and death). Fluid requirements increase incrementally from day 1 of life (60 to 100 mL/kg/d for term to extremely low birth weight infants, respectively) to 150 mL/kg/d at 1 week of life (up to 180 mL/kg/d by 1 week in a premature neonate with high evaporative losses). $D_{10}W$ is commonly used for IV fluid maintenance in a newborn for the first 24 hours. After 24 hours, once adequate urine output is established, 10% dextrose in 0.25 normal saline is used as a maintenance fluid in a neonatal transport; 10 mEq of potassium chloride in 500 mL may be added unless findings show abnormal electrolyte levels or renal function, necessitating adjustment. In general, a full-term infant needs 60 mL/kg/d, a very low birth weight infant (< 1,500 g) needs 80 mL/gk/d, and an extremely low birth weight infant (< 1,000 g) needs 100 mL/kg/d on day 1 of life, increasing by 10 mL/kg/d each day until reaching total fluids of 150 mL/kg/d.

Central Nervous System, Nociception, and the Stress Response

The lower limit for cerebral autoregulation in neonates is not known, but it is thought to be near a cerebral perfusion pressure of 30 mm Hg. In general, neonates are considered to have pressure passive cerebral circulation (as mean arterial pressure falls, cerebral blood flow falls, and vice versa). Appropriate mean arterial blood pressures for extreme premature neonates have not been determined but it is generally acknowledged that an acceptable mean arterial pressure equates to the gestational age of the newborn. It is also important to minimize stress and use appropriate sedation for procedures to minimize blood pressure peaks that can lead to intraventricular hemorrhage.

Developmental Aspects of Pain

Neonates, including premature neonates, show well-developed responses to painful stimuli. Pain in neonates should be treated using appropriate analgesic measures, such as bundling and a pacifier dipped in $D_{10}W$ for blood draws or IV placement, or local anesthetics or opiates for more invasive procedures.

Skeletal Development

Neonates have incomplete ossification of bones and tissues are more fragile than in older children and adults, making them more prone to soft-tissue and bony injuries. This should be considered when providing CPR because neonates are at higher risk of rib fractures and liver laceration during chest compressions. Optimal technique for chest compressions in neonates is reviewed later in the section on neonatal resuscitation.

Neonatal Assessment and Stabilization

Steps taken to assess, stabilize, and optimally transport an ill neonate can make a significant difference in

long-term outcome. For this reason, it is critical for CCTPs to recognize neonatal emergencies and know how to respond to them. Optimally, as soon as a transport need is identified, or at the latest on arrival at the referring institution, the CCTP should review relevant history, including prenatal issues, neonatal symptoms, vital signs (including blood pressure), physical exam findings, chest or abdominal radiographs, if obtained, and laboratory values (including blood glucose). Communication among all health care providers remains critical throughout the transport to optimize care. Specifics regarding assessment and stabilization are described below.

Common Risk Factors

The risk for complications in neonates increases as birth weight and gestational age decrease. Risk also increases in term infants when additional complications such as maternal infection, diabetes, hypertension, or meconium are present. Antepartum and intrapartum risk factors for complications are listed in **Table 22-3** and **Table 22-4**.

The APGAR Score

The **APGAR score** is a standardized numeric expression of a baby's condition after birth **Table 22-5**. Traditionally, 1- and 5-minute scores are recorded, although extended scores (every 5 minutes up to 20 minutes if the score is < 7) may need to be recorded if the infant's condition remains depressed. Each of the five signs is awarded a score of 0, 1, or 2, with a maximum score of 10.

Factors that affect the APGAR score include physical maturity (APGAR scores are often lower in premature infants), maternal medication intake or narcotic use, and neuromuscular or cardiorespiratory conditions. The score is a limited indicator of the severity of asphyxia. The correlation of the APGAR score with adverse neurologic outcome increases when the score remains 0 to 3 at 20 minutes.

Laboratory Assessment

Neonatal laboratory values assist in managing a variety of critical conditions. Critical neonatal laboratory values are listed in **Table 22-6**.

Stabilization and Management

Stabilization of the neonate is needed if there is acute airway obstruction, ineffective respiration, or insufficient cardiovascular circulation. The neonate must be evaluated for respiratory effort, heart rate, and color. The first steps in stabilizing a neonate are airway management and ventilation. To optimize adequate oxygenation and ventilation, suction the neonate's airway thoroughly.

Free-flow (blow-by) supplemental oxygen is required if peripheral cyanosis is present despite adequate respiratory effort and a pulse rate greater than 100 beats/min. Ventilations are required if respiratory effort is inadequate, ineffective, or absent, or if the pulse rate is less than 100 beats/min. Gasping respirations usually indicate a significant problem and require the same intervention as no respiratory effort or apnea. Ventilations are provided at a rate of 40 to 60 breaths/min, with a tidal volume sufficient to expand the neonate's chest, keeping in mind that a physiologic tidal volume for a neonate is 5 to 7 mL/kg. Intubation will be required if bag-mask ventilations are ineffective, tracheal suctioning is required, or prolonged PPV is necessary (see the section on neonatal resuscitation). Chest compressions are indicated if the neonate's pulse is less than 60 beats/min without improvement despite assisted ventilations with bag-mask ventilations (3 compressions followed by 1 breath, with 90 compressions and 30 breaths delivered each minute). Volume expansion may be necessary (10- to 20-mL/kg bolus of normal saline) as well as pharmacologic medications (epinephrine for persistent

bradycardia < 60 beats/min despite optimal respirations and chest compressions, dextrose [$D_{10}W$] for blood glucose levels < 40 mg/dL, or sodium bicarbonate for documented or suspected metabolic acidosis).

TABLE 22-3 Antepartum (Before Birth) Risk Factors
Multiple gestation
Pregnant woman's age < 16 y or > 35 y
Postterm (> 42 weeks') gestation
Toxemia, hypertension, diabetes
Polyhydramnios (excessive amount of amniotic fluid)
Premature rupture of the membrane and fetal malformation
Inadequate prenatal care
History of perinatal morbidity or mortality
Use of drugs/medications
Fetal anemia
Oligohydramnios (decreased volume of amniotic fluid during a pregnancy)

TABLE 22-4 Intrapartum (During Birth) Risk Factors
Premature labor
Rupture of membranes > 24 h before delivery
Abnormal presentation
Prolapsed cord
Chorioamnionitis
Meconium-stained amniotic fluid
Use of narcotics within 4 h of delivery
Prolonged labor or precipitous delivery
Bleeding
Placenta previa

TABLE 22-5 The APGAR Score

Sign	Score 0	1	2
Appearance (color)	Blue or pale	Body pink, extremities blue	Completely pink
Pulse (heart rate)	0	0-100	> 100
Grimace (irritability)	No response	Grimace	Cries
Activity (muscle tone)	Limp	Some flexion	Active movement
Respirations	Absent	Slow or irregular	Strong cry

TABLE 22-6 Critical Neonatal Lab Values

Name	Normal	Critically Low	Critically High
Blood Hematology and Coagulation Studies			
Hematocrit (% vol), neonate	44-64	< 33	> 70
Hemoglobin (g/dL), neonate	14.5-22	< 9.5	> 22
Chemistry			
Bilirubin (mg/dL), neonate	1-10	None	> 13
Glucose (mg/dL), neonate	40-65	< 40	> 200
Microbiology (Qualitative Results) and Serology			
Group B streptococcus antigen (delivery and nursery)	None	None	Positive
Urinalysis			
Note exceptions:	None	Urine glucose in patient < 2 y	Positive
	None	Urine ketones in patient < 2 y	Positive

Note: There is variance in unpublished lists—this is not an official list.

Special care needs to be taken when stabilizing and transporting a preterm neonate. Infants born before 34 weeks of gestation are at increased risk for bleeding into the brain (intraventricular hemorrhage). Avoiding rapid fluctuations in blood pressure, temperature, fluid volume, and pH status minimizes this risk. Preterm infants needing oxygen therapy and mechanical ventilation are also at risk of long-term lung damage, or bronchopulmonary dysplasia. Minimizing oxygen exposure and avoiding excessive pressures from assisted ventilation minimizes this risk. Even late preterm infants, those born at 34 to 36 weeks of gestation, are at increased risk of jaundice, dehydration, hypothermia, hypoglycemia, and sepsis.

It is important for the CCTP to communicate with the receiving facility as soon as possible. Important information includes relevant history, vital signs, physical exam findings, diagnostic studies done such as labs and radiographic results, relevant social issues, anticipated needs on arrival (ventilator, etc), and estimated time of arrival.

Provide psychological support and communicate with the neonate's parents. Encourage maternal bonding. Explain to the parents what is happening to their infant and what steps are being done to optimize the infant's outcome. Do not provide false hope and avoid discussing the neonate's chances of survival.

Determining the Need For and Providing Neonatal Resuscitation

The ABCs of neonatal resuscitation are the same as those applied to adults:

- Airway (position and clear)
- Breathing (stimulate to breathe)

- Circulation (assess pulse rate and color)

The most important and effective action in neonatal resuscitation is getting oxygen into the infant's lungs. In addition, maintenance of normal body temperature is important during neonatal resuscitation. The initial evaluation of the neonate begins with the evaluation of breathing, color, and pulse rate (counted for 6 seconds at the base of the cord or by auscultation and multiplied by 10).

In an infant who is cyanotic, first ensure patency of the airway by bulb suctioning the mouth and then the nose, and gently stimulating the infant. Stimulation is provided by flicking the soles of the feet and rubbing the back. It is important not to be too rough with the rubbing and to avoid slapping the infant because these actions may lead to traumatic injury.

In an emergency situation where it may not be possible to weigh the infant or child, CCTPs should be familiar with the length-based/color-coded resuscitation tape (for example, the Broselow tape) to help provide an estimate of body weight based on the child's crown-to-heel length **Figure 21-3**. This is helpful because most neonatal and pediatric (up to 40 kg) drug doses are based on body weight. It will also help determine the appropriate size of resuscitation supplies for the patient. The sizes for pediatric supplies are organized and listed according to the length-based/color-coded classification scheme.

Figure 22-3 Use of a length-based resuscitation tape is one way to estimate a child's weight and identify the correct size for pediatric equipment and appropriate medication doses. A child is shown here, but the tape also can be used on an infant.

Airway/Breathing Management

Free-Flow Oxygen

If an infant is cyanotic or pale, provide supplemental oxygen. Oxygen should be warmed and humidified if it needs to be provided for more than a few minutes. If PPV is not indicated (pulse rate > 100 beats/min and adequate respiratory effort), oxygen initially can be delivered through an oxygen mask held close to the infant's face. Alternatively, if a flow-inflating bag is set up, CCTPs can deliver oxygen by holding the mask close to the infant's face. If a self-inflating bag is connected to an oxygen source, the oxygen reservoir can be held close to the infant's face to provide oxygen, or CCTPs can disconnect the oxygen tubing from the self-inflating bag, and hold the tubing within a hand cupped loosely over the mouth and nose to facilitate delivery of supplemental oxygen. Holding the face mask over the neonate's face without squeezing the bag does not deliver oxygen from a self-inflating bag. The oxygen flow rate should be less than or equal to 5 L/min to minimize hypothermia. Remember that 5 g/dL of deoxygenated hemoglobin is needed before clinical cyanosis is observed. A severely anemic hypoxic neonate will be pale, but not cyanotic. Studies have demonstrated that even short exposure to supplemental oxygen may be associated with adverse oxidant stress in the neonate, both directly to the lungs and indirectly to premature neonates' eyes, and that resuscitation with room air may be an acceptable alternative. Oxygen, while life sustaining, can have negative side effects. Excessive oxygen in premature infants can lead to blindness. Oxygen toxicity, from a direct oxidant stress, can cause and exacerbate lung disease. For these reasons, once resuscitation has been achieved, an oxygen saturation monitor should be used to adjust the amount of

oxygen to the target range appropriate for each condition. In general, a premature infant's oxygen saturation should be maintained at 88% to 92%, and a term infant's oxygen saturation should be maintained at 95% to 98% saturation.

Oral Airways

Oral airways are rarely used for neonates, but can be lifesaving if airway obstruction leads to respiratory failure. Bilateral **choanal atresia** (a bony or membranous obstruction at the back of the nose preventing airflow) can be rapidly fatal, and usually responds to the placement of an oral airway (or a gloved finger until an adequate sized oral airway is located). The **Pierre Robin syndrome** is a series of developmental anomalies including a small chin and posteriorly positioned tongue that frequently leads to airway obstruction. Positioning the patient prone (chest down) may relieve the obstruction. If not, an oral airway should be placed.

Bag-Mask Ventilation

Signs of respiratory distress that suggest a possible need for bag-mask ventilation include gasping or periodic breathing, intercostal retractions (sucking in between the ribs), nasal flaring, and grunting on expiration. Respiratory distress occurs in approximately 8/1,000 live births and accounts for approximately 15% of neonatal deaths. Bag-mask ventilation is indicated when an infant is apneic, has inadequate respiratory effort, or has a pulse rate of less than 100 beats/min (this constitutes bradycardia in a newborn) after clearing the airway of secretions, relieving obstruction from the tongue, and stimulation. Bag-mask ventilation is also required when there is persistent **central cyanosis** despite breathing 100% oxygen.

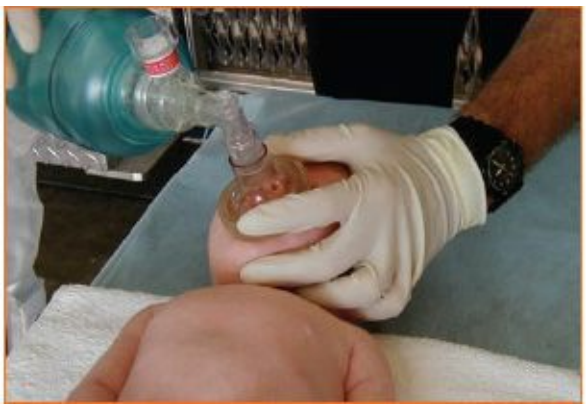


Figure 22-4 Bag-mask ventilation of the newborn. Hold the mask securely to the face with your thumb and index finger. Apply countertraction under the bony part of the chin with your middle finger.

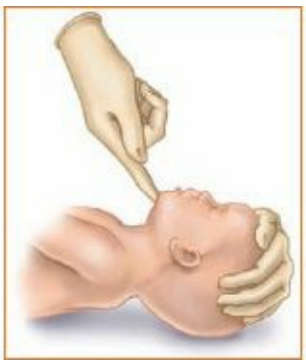


Figure 22-5 The sniffing position.

Adequate bag-mask ventilation with 100% oxygen necessitates the use of appropriately sized

equipment with techniques modified for the unique needs of the neonate. The face mask needs to provide an airtight seal, fitting over the infant's mouth and nose, extending down to the chin but not over the eyes **Figure 22-4**. The infant needs to have a patent airway, cleared of secretions, with the neck slightly extended in the sniffing position **Figure 22-5**. The first few breaths of a neonate after birth frequently require higher pressures (sometimes as high as 30 cm H_2O pressure or higher for brief periods of time) because the lung is not yet expanded and is still filled with fluid. To deliver these initial breaths, the pop-off valve (usually set by the manufacturer at 30 to 40 cm H_2O) on the bag may need to be disabled. Subsequent breaths should be delivered with sufficient pressure to result in visible but not excessive chest rise. The neonate should appear to be taking an easy breath, with a pressure of about 15 to 20 cm H_2O for normal lungs.

Three devices are used to deliver bag-mask ventilation to a neonate. The first device is a self-inflating bag with an oxygen reservoir **Figure 22-6A**. For this method, an oxygen source is not necessary to provide PPV but is necessary to provide supplemental oxygen. The second device is a flow-inflating bag that needs a gas source to provide PPV **Figure 22-6B**. The third device is a T-piece resuscitator that minimizes the risk of excessive pressures, pneumothorax, and lung injury in the neonate, and is becoming more widely available **Figure 22-6C**.

CCTPs should be familiar with available equipment in advance and ensure that the equipment is in working order. CCTPs will most likely be using a self-inflating bag. It is important to use the infant size (240 mL) if available. Since the tidal volume of a neonate is only 5 to 7 mL/kg, even when using a 240-mL bag, only one tenth the volume is used for each breath. If a neonatal bag is not available and the infant has severe respiratory distress, apnea, or bradycardia, a bag designed for adults or older children (750 mL or greater volume) may be used if the delivered breath size is kept appropriately small. In this situation, it is even more critical to monitor chest rise to avoid an excessive volume of delivered breaths. The algorithm for managing a distressed newborn is shown in **Figure 22-7**.

The most common reasons for ineffective bag-mask ventilation are inadequate mask seal and incorrect head position. Other causes such as mucous plug, pneumothorax, or equipment malfunction need to be considered. The correct timing is 40 to 60 breaths/min in a neonate. It is easy to deliver breaths at a much higher rate, which can increase risks such as hypocapnia, air trapping, and pneumothorax. Counting "breathe-two-three-breathe-two-three" as you ventilate (give a breath on "breathe," release while you say "two-three") can help with timing. PPV is continued as long as the pulse rate remains less than 100 beats/min or respiratory effort is ineffective. If prolonged PPV is needed, using a pressure manometer is important to help with monitoring and minimizing excessive pressures (usually less than 25 cm H_2O pressure in term infants, less in preterm infants).

Whereas newborn resuscitation with 100% oxygen is standard in the United States and is currently recommended by the American Academy of Pediatrics, a growing number of studies suggest that newborn resuscitation with room air is a safe alternative, including a recent meta-analysis published that showed increased survival in newborns initially resuscitated with room air compared to those resuscitated with 100% oxygen. Regardless, if oxygen is not readily available, bag-mask ventilation can be initiated with room air while an oxygen source is established. Remember to continuously monitor the effectiveness of ventilation and keep the infant warm throughout his or her care. Ventilation of the lungs is the single most important and effective step in cardiopulmonary resuscitation of the compromised neonate. Signs of effective ventilation include rapid rise in heart rate, improvement in color and tone, audible breath sounds by auscultation, and visible chest rise.

Table 22-7 lists equipment and medications needed for preparation for neonatal resuscitation.

Intubation

Most neonatal resuscitations can be successfully completed with bag-mask ventilation. However, intubation is indicated if:

- Meconium-stained fluid is present at birth and the infant is significantly depressed. (Intubation should occur prior to stimulating the infant, for the purpose of suctioning the trachea before the infant takes his or her first breath.)
- Congenital **diaphragmatic hernia** (congenital defect where abdominal organs herniate through an opening in the diaphragm into the chest cavity) is suspected and respiratory support is indicated.
- The infant is not responding to bag-mask ventilation and chest compressions leading to the need for epinephrine administration in a neonate when IV access is being obtained.
- Prolonged PPV is needed.

Figure 22-6 One of three devices can be used to deliver bag-mask ventilation to a neonate. **A.** A self-inflating bag. **B.** A flow-inflating bag. **C.** A T-piece resuscitator.

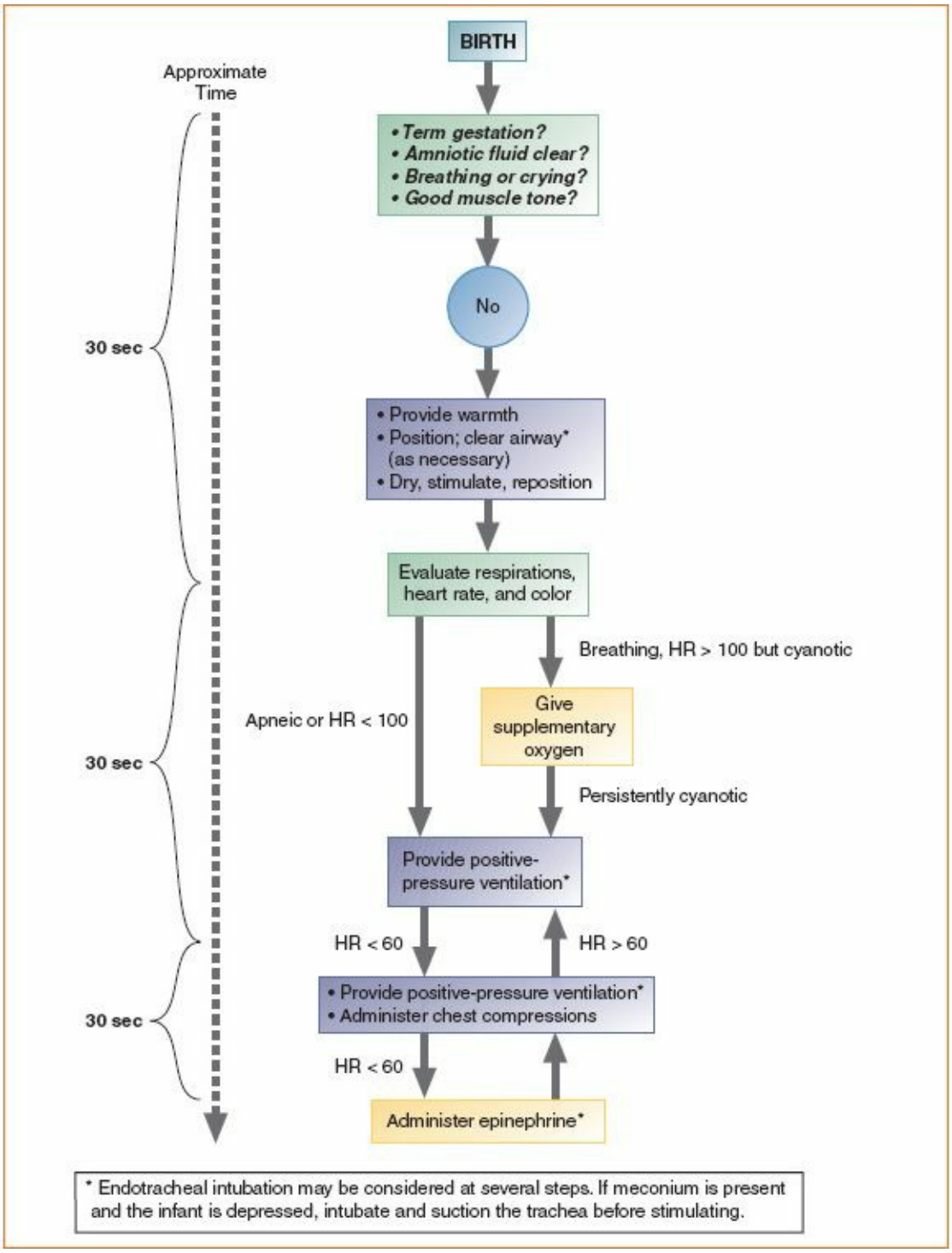


Figure 22-7 Resuscitation algorithm for the distressed neonate. Used with permission of the American Academy of Pediatrics, *Textbook of Neonatal Resuscitation, Fifth edition*. © American Academy of Pediatrics.

TABLE 22-7 Preparation of Area for Neonatal Resuscitation	
Resuscitation equipment and supplies	Suction equipment Bulb syringe, mechanical suction and tubing, suction catheters, 5F or 6F 8F feeding tube and 20-mL syringe Meconium aspirator as indicated
	Device for delivering positive-pressure ventilation, capable of delivering 90% to 100% oxygen

Bag-mask equipment	Face masks, neonatal and premature infant size (cushioned-rim masks preferred) Oxygen source with flow meter (flow rate up to 10 L/min)
Intubation equipment	Laryngoscope with straight blades, size 0 (preterm) and size 1 (term) Extra bulb, batteries for laryngoscope Endotracheal tubes size 2.5, 3.0, 3.5, and 4.0 Stylet (optional) Scissors and tape for securing endotracheal tube CO_2 detectors (optional) Laryngeal mask airway (optional)
Medications	Epinephrine 1:10,000 (0.1 mg/mL), 3- or 10-mL ampules Isotonic crystalloid (normal saline or lactated Ringer's solution), 100- or 250-mL bag Sodium bicarbonate 4.2% (5 mEq/10 mL) Naloxone hydrochloride, 0.4- or 1.0-mg/mL ampule Dextrose 10%, 250 mL
Umbilical catheterization equipment	Sterile gloves Scalpel or scissors Antiseptic solution Umbilical tape Umbilical catheters, 3.5F, 5F (a sterile 3.5F feeding tube can be used in an emergency) Three-way stopcock Syringes, 1, 3, 5, 10, 20, and 50 mL Needles, 25, 21, and 18 gauge
Miscellaneous	Gloves and appropriate BSI protection Radiant warmer or other heat source Firm, padded resuscitation surface Clock with second hand, timer optional Towels, linens Stethoscope, neonatal or pediatric preferred Cardiac monitor or saturation monitor Oropharyngeal airway (0, 00, and 000 sizes or 30-, 40-, and 50-mm long)

Adapted from American Academy of Pediatrics, *Textbook of Neonatal Resuscitation, Fifth edition*. American Academy of Pediatrics, 2006.

TABLE 22-8 Neonatal Endotracheal Tube Sizes

Size	Recommended Age, weeks
2.5	< 28
3.0	28-34
3.5	34-38

4.0	> 38

Before beginning to intubate the neonate, ensure the appropriate equipment is at hand. Use the straight blade #1 for a term infant and #0 for a preterm infant. Endotracheal (ET) tubes for neonates are available in size 2.5 to 4.0 mm **Table 22-8**. Again, a length-based/color-coded resuscitation tape or Broselow tape can be used to estimate ET tube size. A stylet may be used to provide rigidity to the ET tube. If a stylet is used, it is imperative that it is secured in its position (bending it over at the top of the ET tube so it can't advance) and not extended beyond the ET tube, or tracheal perforation may occur.

Skill Drill 22-1 shows intubation of the neonate, which is discussed in the following steps.

1. Ensure the neonate is preoxygenated by bag-mask ventilation with 100% oxygen prior to an intubation attempt **Step 1**.
2. Suction the oropharynx to ensure removal of secretions **Step 2**. This is a vagal stimulus, so pay close attention to pulse rate; bag-mask ventilation may again be needed prior to the intubation attempt if bradycardia results.
3. Place the laryngoscope blade in the oropharynx and then visualize the vocal cords, keeping in mind the vocal cords are more cephalad and anterior relative to adults **Step 3**. Take care to avoid applying torque to the blade, which increases the risk of trauma. Place the ET tube between the vocal cords until the black line on the tube is at the level of the cords. A general rule of thumb is that for term babies, the ET tube usually is advanced until it is at 9 cm at the lip. ET tube depth of insertion for premature infants is generally determined using this formula:

   Weight in kg + 6

   An extremely low birth weight baby (< 1,000 g) may only need to have the ET tube advanced to 6½ to 7 cm at the lip. Intubation attempts should be limited to 20 seconds, followed by bag-mask ventilation if unsuccessful or if significant bradycardia develops.
4. Confirm placement **Step 4** by observing chest rise when applying positive pressure through the ET tube, auscultating laterally and high on the chest, noting the absence of significant air sounds over the stomach, noting mist in the ET tube (will be seen when patient exhales through the tube from condensation of humidified air leaving the lungs), and clinical improvement of the patient. A CO_2 detector should be used if available.
5. Tape the ET tube in place on the face to minimize the risk of the tube dislodging **Step 5**. Once you place an ET tube, the infant needs to be monitored closely for complications such as tube dislodgement, tube occlusion by mucous plug, or pneumothorax.

Complications of ET tube placement include oropharyngeal or tracheal perforation, esophageal intubation with subsequent persistent hypoxia, and right mainstem intubation that can lead to atelectasis, persistent hypoxia, and pneumothorax. These risks can be minimized by optimal placement of the laryngoscope blade and carefully noting how far the ET tube is being advanced. Premedication is often not needed in depressed neonates. Consider atropine to avoid vagal-induced bradycardia. Administer sedation if agitation is present. Avoid paralytics since neonatal airways are very small, and may be difficult to maintain once the neonate is paralyzed.

Gastric Decompression

Gastric decompression using an orogastric tube is indicated for prolonged bag-mask ventilation (more than 5 to 10 minutes), if abdominal distention is impeding ventilation, or in the presence of a known or suspected diaphragmatic hernia. Many diaphragmatic hernias are diagnosed prenatally by routine ultrasound. They are suspected clinically if the following are noted: decreased breath sounds (90% of diaphragmatic hernias are on the left), a scaphoid or concave abdomen because many of the abdominal contents are in the chest, and increased work of breathing.

Skill Drill 22-2 shows neonatal orogastric tube placement, which is discussed here:

1. To determine the length of tube to insert, use an 8F feeding tube and measure the length from the bottom of the earlobe to the tip of the nose to halfway between the xiphoid process (lower tip of sternum) and the umbilicus **Step 1**.
2. Insert the tube through the mouth to this distance **Step 2**.
3. Attach a 20-gauge syringe and suction the stomach contents **Step 3**. Tape the tube to the neonate's cheek. Remove the syringe from the feeding tube to allow venting of air from the stomach, and intermittently suction the feeding tube.

Circulation

If the heart rate remains below 60 beats/min despite positioning, clearing the airway, drying and stimulation, and 30 seconds of effective PPV, chest compressions are indicated. Two techniques are used to perform chest compressions: the thumb technique and the two-finger technique. In the thumb technique, two thumbs are placed side by side over the sternum between the nipples and the hands encircle the torso. In the two-finger technique, the tips of two fingers are placed over the sternum between the nipples, and the sternum is compressed between the fingers **Figure 22-8**.

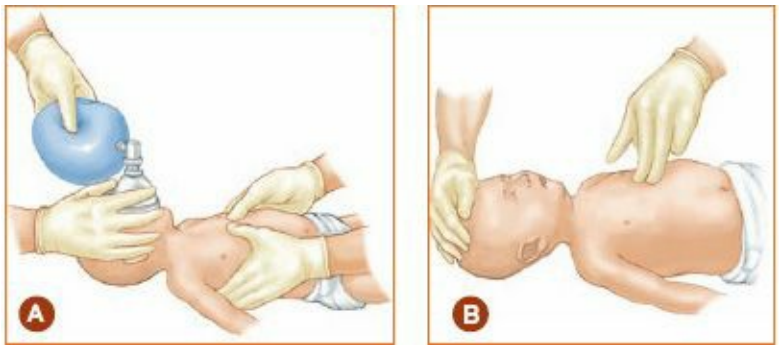
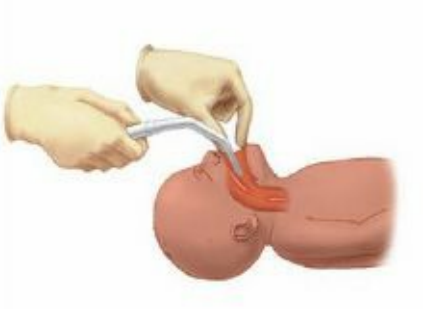


Figure 22-8 Chest compressions in the neonate. **A.** When there are two providers, use your thumbs side by side, placed just below an imaginary line drawn between the two nipples. **B.** When working alone, or when the infant is large, use two fingers to depress the sternum.

Skill Drill 22-1

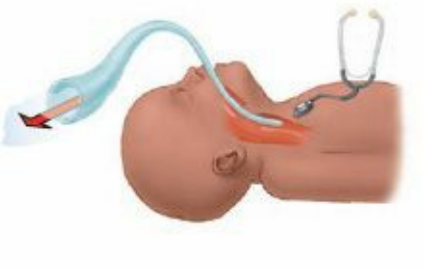
Intubation of a Neonate

1 Preoxygenate the infant by bag-mask ventilation with 100% supplemental oxygen.

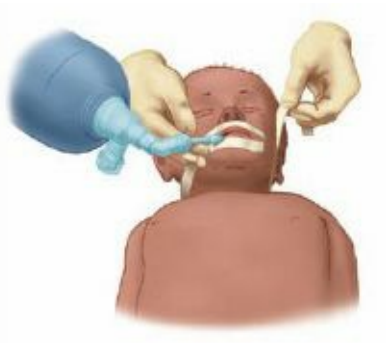


2 Suction the oropharynx. Provide bag-mask ventilation if bradycardia results.

3 Place the laryngoscope blade in the oropharynx. Visualize the vocal cords. Place the ET tube between the vocal cords until the black line on the tube is at the level of the cords.



4 Confirm placement. Observe chest rise, auscultate laterally and high on the chest, note the absence of significant air sounds over the stomach, note mist in the ET tube, and use a CO_2 detector.



5 Tape the ET tube in place. Monitor the neonate closely for complications.

Skill Drill 22-3 reviews the steps for resuscitating a neonate, which are listed here:

1. Lay the neonate flat (Trendelenburg position can cause intracranial hemorrhage especially in preterm infants) **Step 1**.
2. Suction the mouth and nose with a bulb syringe (if available) after delivery of the shoulders. Drying and suctioning are usually enough stimulation to induce breathing. If these do not suffice, gently flick your fingers against the soles of the neonate's feet and/or rub the neonate's back **Step 2**. Rough handling is not needed. *Do not shake the baby*.
3. If the neonate is still not breathing effectively, begin bag-mask ventilation **Step 3**. If the neonate begins breathing on his/her own, support and assist respirations and recheck the airway to be sure it remains clear.
4. If the neonate is still not breathing, continue bag-mask ventilation and check for an umbilical, femoral, or brachial pulse **Step 4**.
5. If you cannot feel a pulse or if the heart rate is less than 60 beats/min, begin closed-chest cardiac compressions **Step 5**.

Skill Drill 22-2

Inserting an Orogastric Tube in the Neonate

1 Measure for correct depth—from the bottom of the earlobe to the tip of the nose to halfway

between the xiphoid process (lower tip of sternum) and the umbilicus.

2 Insert the tube to the appropriate depth.

3 Remove the gastric contents with a 20-mL syringe. Tape the tube to the neonate's cheek. Remove the syringe from the feeding tube to allow air to vent from the stomach.

The depth of compression is one third of the anterior-posterior diameter of the chest. The fingers should remain in contact with the chest at all times to minimize risk of trauma. Time the compressions in synchrony with artificial ventilation, which continues during chest compressions. (In neonates, even if respirations are being supported by bag-mask ventilation, the ventilation is still timed with the chest compressions.) The person delivering the chest compression counts aloud, "one and two and three and breath and." Downward strokes of chest compressions should be delivered while saying "one, two, three." Release of the strokes should occur while saying "and." The person ventilating delivers a breath during "breath-and." This results in 90 compressions and 30 breaths/min. Note that this rate applies to one-rescuer infant CPR and it is best to use two rescuers for a faster compression rate. Heart rate is assessed at 30-second intervals and chest compressions stopped when the heart rate is greater than 60 beats/min. Liver laceration and rib fractures are possible risks of delivering chest compressions.

If the neonate is not responding to resuscitation, other causes must be considered. Generally, the difficulties relate to the following: technical/mechanical/equipment problems, unrecognized pulmonary complications, severe metabolic derangements, congenital abnormalities of organs systems, or severe anemia.

Pharmacologic Interventions

Medications are rarely needed in neonatal resuscitation because most infants can be resuscitated with

effective venti-latory support. Epinephrine is indicated when the heart rate remains below 60 beats/min despite PPV and chest compressions. Normal saline bolus, lactated Ringer's, or blood may be indicated for hypovolemia, blood loss, or for suspected metabolic acidosis while awaiting blood gas results. Sodium bicarbonate is only indicated when the degree of metabolic acidosis is known from a blood gas. Naloxone is generally not used during resuscitation but may be used if the infant continues to have depressed respirations and there is a history of narcotic use in labor. It is contraindicated if the mother is a chronic narcotics user. Medications in neonates are based on weight, so you may need to estimate the infant's weight for dosing. A term infant usually weighs 3 to 4 kg (approximately 6½ to 8½ lb). An infant born at 28 weeks of gestation averages 1 kg (2.2 lb), and an infant born at 34 weeks of gestation averages 2 kg (4½ lb). Medications that may be used are listed in **Table 22-9**.

Epinephrine

If the infant has a pulse rate of less than 60 beats/min after 30 seconds of effective ventilation and 30 seconds of chest compressions, administration of epinephrine is indicated. The recommended concentration for neonates is 1:10,000. The recommended dose is 0.1 to 0.3 mL/kg of 1:10,000 epinephrine IV, equal to 0.01 to 0.03 mg/kg, administered rapidly, and followed by a 0.5- to 1-mL normal saline flush to clear the line. While IV is the optimal route to deliver epinephrine, if IV access is not yet established, consider starting with the higher dose of 0.3 to 1.0 mL/kg of 1:10,000 epinephrine via the endotracheal tube. Epinephrine dosing may be repeated every 3 to 5 minutes for persistent bradycardia.

Skill Drill 22-3

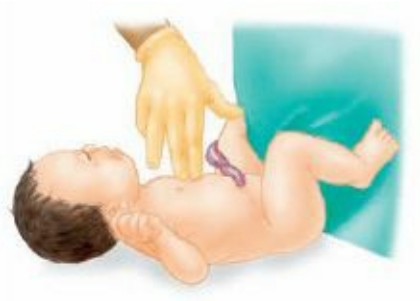
Resuscitating a Neonate

1 Lay the neonate flat. Suction the mouth and nose with a bulb syringe.

2 If drying and suctioning do not induce breathing, gently snap your fingers on the soles of the neonate's feet.

3 Begin bag-mask ventilation if the neo-nate is not breathing effectively.

4 If the neonate is still not breathing, continue bag-mask ventilation and check for an umbilical, femoral, or brachial pulse. The brachial location is shown here.



5 If you cannot feel a pulse or if the heart rate is less than 60 beats/min, begin chest compressions.

Volume Replacement

If the infant's intravascular volume is significantly depleted from conditions such as placental abruption or septic shock, fluid resuscitation may be needed. In a newborn, a low umbilical venous catheter can be placed in the large vein in the umbilical cord, advancing the line just far enough to allow blood return, but not far enough to enter the liver. In an infant older than a few days, a peripheral IV or intraosseous (IO) line will need to be placed. It is important to note that while the technique for an IO line is similar to that for older children or adults, a smaller needle needs to be used to avoid exiting the far side of the bone. A fluid bolus in an infant is 10 to 20 mL/kg of normal saline IV over 5 to 10 minutes. More than one bolus may be needed if the patient is still clinically hypovolemic. Signs of hypovolemia include pallor, delayed capillary refill, and weak pulses despite a good heart rate or high-quality chest compressions (defined as compressions delivered at a depth of approximately one third of the anterior-posterior diameter of the chest that allow for full chest recoil at a rate of 90 compressions per minute with minimal interruptions. Note that in the neonate, the specific depth is important since the size of the neonate is variable; compressing too little leads to ineffective compressions, while compressing too deeply leads to trauma).

Ventilation During Transport

Neonatal transport incubators are typically fitted with a ventilator that provides synchronized intermittent mandatory ventilation. Settings are adjusted relative to the patient's condition. Peak inspiratory pressure is adjusted to provide a comfortable breath (typically 14 to 20 mm Hg, lower range for more premature infants, higher for term infants with decreased lung compliance such as seen in pneumonia). Peak end expiratory pressure is typically 3 to 4 mm Hg, rate 20 to 60 breaths/min, inspiratory time 0.4 seconds. If volume ventilation is used, a tidal volume of 6 to 8 mL/kg is targeted. Ventilator tubing can affect the measured tidal volume on some ventilator circuits, so close observation of chest rise and peak inspiratory pressure is critical, especially when first placing a neonate on tidal volume ventilation. For patients with very severe lung disease, a standard ventilator may not provide adequate support. High-frequency ventilation, a respiratory frequency of 400 to 800 breaths/min and optimal PEEP (usually higher than that used with conventional ventilation), relies primarily on convection and molecular diffusion of gas rather than tidal volume ventilation to exchange oxygen and carbon dioxide. Some transport incubators can be fitted with a high-frequency ventilator, in which case comparable ventilator settings can be used for transport. Frequently only a conventional ventilator is available for transfer. When switching a neonate from high-frequency ventilation back to a conventional ventilator, the neonate usually tolerates the fastest rate that the conventional ventilator can deliver; inspiratory pressures need to be kept relatively low and expiratory times long (I:E at least 1:2) to minimize the risk of air trapping. If the neonate is critically ill and oxygen saturations cannot be maintained on a conventional transport ventilator, hand bagging may provide acceptable ventilation and oxygenation until arrival at a tertiary care center. In the case of term infants with respiratory failure, some centers can initiate inhaled nitric oxide to optimize pulmonary vasodilation or extracorporeal membrane oxygenation in the field and transport on an inhaled nitric oxide or extracorporeal membrane oxygenation circuit. It is important to have discussions with the regional referral center in advance to know the technology that is available in the area.

TABLE 22-9 Medications for Neonates

Drug Name	Dose/Route	Concentration	Indications/Comments	Side Effects
Ampicillin	50 mg/kg IV over 5–10 min	Reconstitute with sterile water	1st line antibiotic for sepsis workup Use with gentamicin Use 100 mg/kg if meningitis suspected	None
Atropine	0.01–0.03 mg/kg IV/IM; may repeat every 5 min × 3 PO: 2–3 × IV dose; minimum dose of 0.1 mg in infants > 5 kg		Preintubation medication to minimize vagal-induced bradycardia Standard dose of 0.02 mg/kg used PALS	Tachycardia, arrhythmia Fever, rash, urinary retention
Cefotaxime	50 mg/kg q 6–12h IV depending on weight/age	In D_5W, $D_{10}W$, NS; infuse over 30 min	Broad spectrum antibiotic used for suspected/documented gram negative sepsis	Potential increased risk of fungal infection due to broad spectrum of coverage.
Ceftriaxone	50 mg/kg/dose q 24h IV/IM	In D_5W, $D_{10}W$, NS; IV infusion given over 30–60 min	Can be used to complete antibiotic course at home Do not use in a newborn nor a neonate with total bilirubin > 5 mg/dL since it displaces bilirubin from albumin and can lead to kernicterus	Cases of fatal reactions with calcium-ceftriaxone precipitates in lung and kidneys in both term and premature neonates have been described.
Dextrose	2 mL/kg IV bolus over 5–10 min	$D_{10}W$; if $D_{25}W$, dilute to < 12.5% to give via peripheral IV	Symptomatic hypoglycemia (blood glucose level < 40 mg/dL) May use for hydration May repeat bolus or follow with 5 mg/kg/min Incompatible with blood products	None
Dobutamine	3–5 μg/kg/min IV; increase 3–10 μg/kg/min to max 40 μg/kg/min	12.5 μg/mL; reconsitute in D_5W/$D_{10}W$/NS	Cardiogenic shock, hypotension, increased myocardial contractility, cardiac failure Try volume first Incompatibile with sodium bicarbonate	Hypoxemia, tachycardia, arrhythmia
Dopamine	5 μg/kg/min IV; increase 2–3 μg/kg/min to max 40 μg/kg/min for effect	40 μg/mL; reconstitute in D_5W/$D_{10}W$/NS	Nonhypovolemic shock, increased cardiac output Try volume first	Hypoxemia, tachycardia, arrhythmia
Epinephrine	0.01–0.03 mg/kg or 0.1–0.3 mL/kg ET or IV q 3–5 min; may titrate IV dose up to max 1 μg/kg/min for effect	1:10,000 (0.1 mg/mL); dilute 1:1 for ET administration	Resuscitation medication for asystole and bradycardia Bronchodilator Standard dose used in PALS and recommended by the AAP is 0.01 mg/kg IV	Arrhythmia Hypertension, IVH Renal failure Hypokalemia
Gentamicin	4 mg/kg IV/IM (IV preferred) over 30 min	Compatible w/TPN, NS, D_5W, $D_{10}W$	Part of sepsis workup Obtain cultures before administering Do not give with cephalosporin	None

Inhaled nitric oxide	20 ppm starting dose		Selective decrease in pulmonary vascular resistance Used for PPHN Started with oxygenation index > 25 Needs specific delivery device	Methemoglobinemia Need daily methemoglobinemia levels
Morphine sulfate	0.05–0.1 mg/kg IM/IV over 15–20 min (may push over 4–5 min if needed), then q 4 h or 10–15 μg/kg/h infusion	Compatible with D_5W, $D_{10}W$, NS	Analgesia and sedation Do not stop suddenly. Do not use in shock and increased intracranial pressure. Use naloxone as antidote for respiratory depression.	Respiratory depression, hypotension, bradycardia
Naloxone hydrochloride	0.1 mg/kg IV push/IM; may repeat in 3–5 min	1.0 mg/mL or 0.4 mg/mL; compatible with D_5W and NS	Narcotic-induced respiratory depression Avoid in newborns with narcotic use in mother.	Hypertension, arrhythmia, Pulmonary edema Vomiting
Normal saline-volume expander	10 mL/kg IV; may repeat × 1 if needed	Alternatives include whole blood or lactated Ringer's, 10 mL/kg	First line for volume resuscitation, shock, hypotension, hemorrhage	None
Phenobarbital	10–20 mg/kg IV, infuse over 10–15 min; may repeat for continued seizures	Compatible with $D_5W/D_{10}W/NS$	Anticonvulsant First line for seizures	Hypotension, repiratory depression Stevens-Johnson syndrome
Prostaglandin E_1	0.05 μg/kg/min infusion; may titrate down when stable, with good effect	Compatible with D_5W/NS; 1 ampule (500 μg) + 500 mL IV fluids = 1 μg/mL PGE solution; not compatible with other solutions or medications	Keeps ducts arteriosus patent Recommend intubating patient for long transports and obtain second IV access prior to infusion.	Apnea, hyperthermia, hypotension, bradycardia, hypoglycemia, seizures, hypocalcemia
Sodium bicarbonate	2 mEq/kg IV push over 2 min for resuscitation; repeat as needed	0.5 mEq/mL (4.2% solution); also available in 1 mEq/mL; compatible with $D_5W/D_{10}W/NS$	Resuscitation Metabolic acidosis Dose in mEq = base deficit on blood gas × 0.3 × weight in kg, over 15 min When the patient has a high serum sodium level, tromeThamine (Tham) may be used at a comparable dose to treat metabolic acidosis.	Rapid infusion may cause IVH

Abbreviations: AAP, American Academy of Pediatrics; D_5W, 5% dextrose in water; $D_{10}W$, 10% dextrose in water; $D_{25}W$, 25% dextrose in water; ET, endotracheal(ly); IM, intramuscularly; IVH, intraventricular hemorrhage; MI, myocardial infarction; NS, normal saline; PALS, pediatric advanced life support; PGE, prostaglandin E; PO, orally; PPHN, persistent pulmonary hypertension.

Additional Transport Considerations

While PPV and chest compressions can be performed in a moving emergency vehicle, it is key to ensure a secure airway before beginning the transport. The neonate is best stabilized at the referral center. Evaluation during air transport is difficult because of vibration, noise, and poor lighting. CCTPs must rely on cardiorespiratory equipment to determine the stability of vital signs. Procedures such as intubation or needle aspiration of the chest can be particularly challenging in flight because of limited available space and inability to maintain a stable environment for needle aspiration. An airway must be secured prior to a helicopter flight.

If additional interventions such as intubation or needle aspiration are needed during ambulance transport, it is recommended that the CCTP consider pulling the ambulance to the side of the road while performing the procedure. If in flight, the CCTP needs to rely more on nonauditory assessments (visual, tactile, electronic monitoring) to provide care and perform necessary procedures. If necessary, bag-mask ventilation can be performed during air transport if the ET tube becomes dislodged and is unable to be replaced readily.

Respiratory Conditions

Clearing the airway and providing stimulation establish adequate cardiorespiratory function in most neonates. However, a small subset of neonates need assisted ventilation, as discussed earlier. Conditions leading to respiratory compromise in the neonate include anatomic anomalies leading to obstruction, such as choanal atresia and Pierre Robin syndrome. Both of these conditions respond to placement of an oral airway. The next sections discuss specific conditions, their causes, and management.

Apnea

Apnea is defined as a pause in respirations of more than 20 seconds. It is often associated with cyanosis, pallor, **hypotonia**, or bradycardia.

There are two types of apnea: primary and secondary apnea. **Primary apnea** is present at birth. This is the initial response to asphyxia at birth. It responds well to stimulation and oxygen supply. If asphyxia continues beyond the primary apnea period, **secondary apnea** sets in after a few deep gasping breaths. It only responds to assisted ventilation and supplemental oxygen. Though these two types of apnea seem clinically indistinguishable, it is critically important to recognize the difference between them to provide appropriate management.

In the neonate, there are multiple causes of apnea. Respiratory causes include fixed anatomic obstruction, obstruction from position (head on chin obstructing airway), secretions, or reflux with aspiration. Metabolic causes include hypoglycemia, inborn errors of metabolism that cause high ammonia levels, and hypocalcemia. Cardiovascular causes include poor tissue oxygenation from arrhythmia, congenital heart disease, shock, anemia/hemorrhage, patent ductus arteriosus, and hypotension. Infectious etiologies include necrotizing enterocolitis, pneumonia, sepsis, respiratory syncytial virus, *Chlamydia*, and pertussis. Neurologic causes include seizures, encephalopathy, intraventricular hemorrhage, congenital hypoventilation, increased intracranial pressure, and apnea of prematurity. Hypothermia or hyperthermia, trauma, child abuse, and illicit drugs vs delivered or prescribed medications need to be considered as well.

Signs and Symptoms

Apnea

- Cyanosis
- Pallor
- Hypotonia
- Bradycardia

Differential Diagnosis

Apnea

- Fixed anatomic obstruction
- Obstruction from position, secretions, or reflux with aspiration
- Hypoglycemia
- Inborn errors of metabolism
- Arrhythmia

- Congenital heart disease
- Shock
- Anemia/hemorrhage
- Patent ductus arteriosus (PDA)
- Hypotension
- Necrotizing enterocolitis
- Pneumonia
- Sepsis
- Respiratory syncytial virus
- *Chlamydia*
- Pertussis
- Seizures
- Encephalopathy
- Intraventricular hemorrhage
- Congenital hypoventilation
- Increased intracranial pressure
- Apnea of prematurity
- Hypothermia
- Hyperthermia
- Trauma
- Child abuse

Transport Management

Apnea

- Manage the ABCs.
- Keep the patient warm.

Management of apnea includes managing the ABCs as needed. Administer free-flow oxygen at 2 L/min for cyanosis and then assisted ventilation if needed. Next, obtain the neonate's history and examine the neonate: Check the fontanelle for a bulge that may indicate increased intracranial pressure. Check the nose and oropharynx for obstruction. During the cardiac exam, listen for murmurs; listen for crackles on pulmonary auscultation. Check capillary refill to assess perfusion. Obtain a set of vital signs. Check the blood glucose level immediately. Then ensure that a complete blood count (CBC), cultures, chest radiograph, a blood gas analysis, and electrolyte levels are obtained if possible. Administer 2 mL/kg of 10% dextrose bolus if the glucose level is less than 40 mg/dL. Keep the patient warm. Ensure that a sepsis workup, including a CBC and appropriate cultures (urine, blood, cerebrospinal fluid), is performed. Administer antibiotics (eg, ampicillin, gentamicin) if these haven't been started. (The sepsis workup is often started at the sending facility and antibiotics chosen by the sending physician.) Administer naloxone if there is positive acute (not chronic) exposure to narcotics. Treatment of apnea often resolves associated symptoms such as cyanosis, bradycardia, and hypotonia. If apnea persists, intubate the patient prior to transport.

Causes of Respiratory Distress in a Neonate

Meconium-Stained Amniotic Fluid

Meconium is the first stool of an infant, and is usually expelled after birth. However, it may be expelled *in utero* in term infants during fetal stress. Post-term infants are at even higher risk for meconium-stained amniotic fluid due to relatively impaired placental function. Meconium-stained amniotic fluid is present in 10% to 15% of deliveries. Meconium aspiration occurs when infants inhale the meconium into their lungs before or during the delivery, when hypoxia or acidosis causes the infant to take a gasping breath. If they inhale the meconium-stained amniotic fluid either *in utero* or at delivery, plugging of the airways and hypoxia can follow. The respiratory distress caused by meconium aspiration is termed meconium aspiration syndrome. It can lead to atelectasis in the lungs, persistent pulmonary hypertension (delayed transition from fetal to neonatal circulation), pneumonitis, and pneumothorax. The differential diagnosis for meconium aspiration syndrome includes respiratory distress syndrome, **transient tachypnea of the newborn** (transient respiratory distress lasting a few hours and which is secondary to retained fetal lung fluid), sepsis, pneumonia, and congenital respiratory or cardiac malformation. Patchy/fluffy infiltrates may be seen on the chest radiograph **Figure 22-9**.

If meconium is present in the amniotic fluid of a depressed newborn at birth, tracheal intubation and aspiration with a meconium aspirator is performed before other interventions. Even when tracheal suctioning is performed, meconium aspiration syndrome may occur. When transporting an infant who has been exposed to meconium-stained amniotic fluid and has respiratory symptoms after delivery, it is important to monitor for the development of complications from meconium aspiration syndrome such as worsening lung compliance and pneumothorax and intervene as symptoms indicate. Consider PPV on transport if progressive hypoxia develops. Short inspiratory time and long expiratory time is commonly used to provide adequate ventilation and minimize air trapping in the neonate. High positive end expiratory pressure values may also be used. A needle set-up should be available in the event the infant develops a significant pneumothorax en route. If a significant pneumothorax develops, as suspected by acute cyanosis, increased work of breathing, and decreased breath sounds, clean the area just over the nipple on the affected side, insert a small (22-gauge) butterfly needle in the second intercostal space, midclavicular line, over the rib, while pulling back on a syringe to evacuate the extrapleural air and relieve the intra-thoracic pressure. This is discussed further in the section on pneumothorax and shown in **Figure 22-11**.

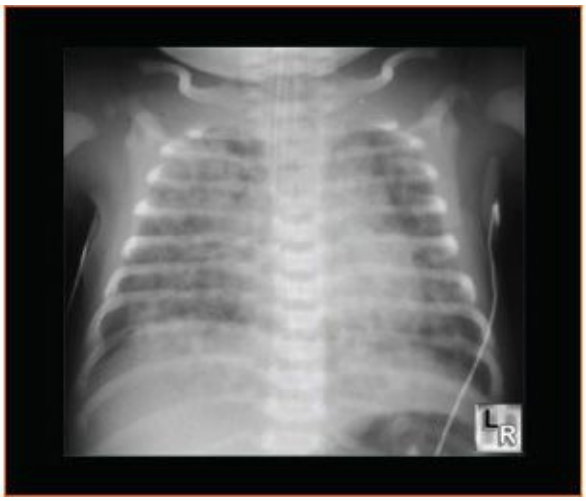


Figure 22-9 A chest radiograph with patchy/fluffy infiltrates, indicating meconium aspiration syndrome.

Signs and Symptoms

Meconium Aspiration Syndrome

- Respiratory distress
- Hypoxia

Differential Diagnosis

Meconium Aspiration Syndrome

- RDS
- Transient tachypnea of the newborn (TTN)
- Sepsis
- Pneumonia
- Congenital respiratory malformation
- Congenital cardiac malformation

Transport Management

Meconium Aspiration Syndrome

- Monitor for development of complications.
- Intervene as necessary.
- Consider PPV if progressive hypoxia develops.
- Perform needle thoracentesis if a significant pneumothorax develops.

Pneumonia

Neonates have an impaired immune response and as a result are at increased risk of pneumonia. Signs and symptoms of pneumonia include tachypnea (respiratory rate > 60 breaths/min), increased work of breathing, and hypothermia. Initial assessment should include a chest radiograph, blood gas measurements in infants with significant respiratory symptoms, CBC, and blood culture. Management includes antibiotics (eg, ampicillin and gentamicin), maintaining the infant with nothing given by mouth until respiratory symptoms resolve (because of the increased risk of aspiration), and administration of IV fluids to maintain hydration and blood glucose levels. Respiratory support such as supplemental oxygen and PPV needs to be provided based on the infant's clinical condition and oxygen saturations.

Signs and Symptoms

Pneumonia

- Tachypnea
- Increased work of breathing
- Hypothermia

Differential Diagnosis

Pneumonia

- Airway foreign body
- Asthma
- Bronchiectasis
- Bronchitis
- Heart failure
- Cystic fibrosis
- Empyema
- Gastroesophageal reflux
- Human immunodeficiency virus infection
- Pertussis
- Pneumococcal infections

Transport Management

Pneumonia

- Maintain the infant with nothing given by mouth (NPO).
- Provide IV fluids.
- Provide respiratory support.

Respiratory Distress Syndrome

Infants delivered prematurely, especially at less than 32 weeks of gestation, have a high risk of respiratory distress syndrome (RDS) as the result of insufficient **surfactant**, a surface-active agent naturally occurring in the lungs that minimizes surface tension in the lung. Within a few hours of birth, neonates with RDS present with grunting, retractions, nasal flaring, tachypnea, and, often, cyanosis. Male infants and infants of diabetic mothers are at increased risk of RDS. Surfactant inactivation can also occur due to interactions with abnormal alveolar proteins such as meconium or blood or in the case of pneumonia, albumin. Both surfactant deficiency and dysfunction may respond to exogenous surfactant, administered in a controlled clinical setting by providers experienced in its delivery. When transporting an infant after surfactant administration, the CCTP needs to recognize improving compliance and the potential need to decrease respiratory support (eg, wean oxygen as saturations improve, decrease peak pressure as chest excursion improves). Peak pressures and oxygen concentration should be minimized as appropriate throughout transport to minimize the risk of pneumothorax or chronic lung disease. An oxygen saturation of 85% to 92% should be targeted during transport of premature infants with RDS to minimize oxygen toxicity. Continuous positive airway pressure can be helpful in mild RDS to minimize alveolar collapse during transport. Ensure that a sepsis workup has been performed prior to transport, since pneumonia in the newborn is indistinguishable from RDS. Prevent cold stress by keeping the neonate warm and bundled.

Signs and Symptoms

Respiratory Distress Syndrome

- Grunting
- Retractions
- Nasal flaring
- Tachypnea
- Cyanosis

Differential Diagnosis

Respiratory Distress Syndrome

- Sepsis
- Pneumonia
- Wet lung
- Lung malformations

Transport Management

Respiratory Distress Syndrome

- Monitor for improving compliance and need to decrease respiratory support.
- Minimize peak pressures and oxygen concentration.
- Target oxygen saturation of 85% to 92% to minimize oxygen toxicity.
- Provide continuous positive airway pressure (CPAP).
- Keep the neonate warm and bundled.

Pneumothorax

Simple pneumothorax is a collection of gas in the pleural space, causing lung collapse. Tension pneumothorax involves air in the pleural space under pressure and is a life-threatening condition. Causes of pneumothorax include meconium aspiration, pneumonia, neonatal resuscitation, trauma, and aggressive assisted ventilation. A pneumothorax may present with increased oxygen requirements, tachypnea, increased work of breathing, agitation, bradycardia if mild/moderate, tachycardia if a large pneumothorax is causing tamponade, cyanosis, and hypotension if the pneumothorax is large. Management of pneumothorax includes transillumination, thoracentesis, and possible intubation, recognizing that PPV may cause an enlargement of the pneumothorax. With transillumination, increased transmission of light on one side of the chest suggests pneumothorax. A chest radiograph will confirm the diagnosis. Findings include an ipsilateral edge of the lung parallel to the chest wall, lucency, and a deep sulcus sign (deep lateral costophrenic angle). Mediastinal shift to the contralateral side is seen in cases of tension pneumothorax. If a symptomatic pneumothorax is suspected, a needle thoracentesis can be lifesaving. On the side of the suspected pneumothorax, clean the area around the second intercostal space, midclavicular line (usually just above the nipple), with alcohol **Figure 22-10**. Prepare the equipment needed: a 22-gauge butterfly

needle attached to extension tubing, a three-way stopcock, and a 20-mL syringe. Insert the needle above the third rib (in the second intercostal space) as a second provider pulls back on the syringe (which is open to the patient). The nerves and blood vessels run below the ribs, so avoid piercing this area **Figure 22-11**. Continue to slowly advance the needle until air is recovered. The butterfly needle is rigid, so be gentle to avoid further tearing the lung. If the 20-mL syringe becomes filled with air, turn the stopcock off to the neonate, push out the air from the syringe, open the stopcock to the neonate, and continue withdrawing air. When no more air can be withdrawn, remove the needle. If there is a symptomatic ongoing air leak, a 22-gauge angiocatheter can be inserted in a similar location, the introducer needle removed, and the angiocatheter attached to the extension tubing. Note that the angiocatheter may further tear the lung during its initial placement and it is more likely to kink than the butterfly needle. Remove as much air as possible with the syringe. At this point, the tubing may be briefly occluded while you place the end of the tubing that had been attached to the syringe in a small bottle of sterile water and release the tubing occlusion. This relieves the pressure buildup from the pneumothorax until the patient can be transferred to a facility skilled in placement of a chest tube in a neonate. Intubate the infant if respiratory distress continues.

Signs and Symptoms

Pneumothorax

- Increased oxygen requirement
- Tachypnea
- Increased work of breathing
- Agitation
- Bradycardia
- Tachycardia
- Cyanosis
- Hypotension

Transport Management

Pneumothorax

- Perform needle thoracentesis if necessary.

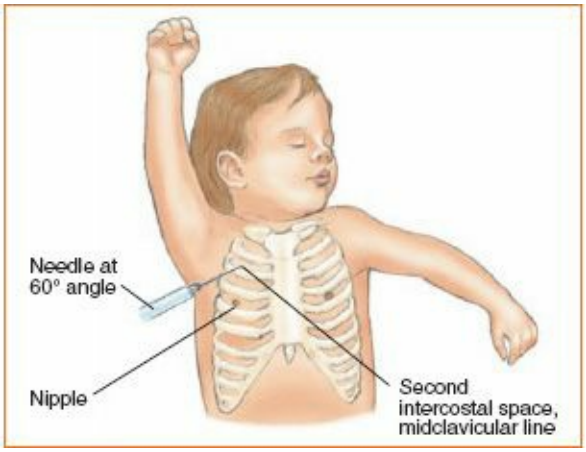


Figure 22-10 For needle thoracentesis to treat a tension pneumothorax or a symptomatic pneumothorax, position the child and identify the entry site.

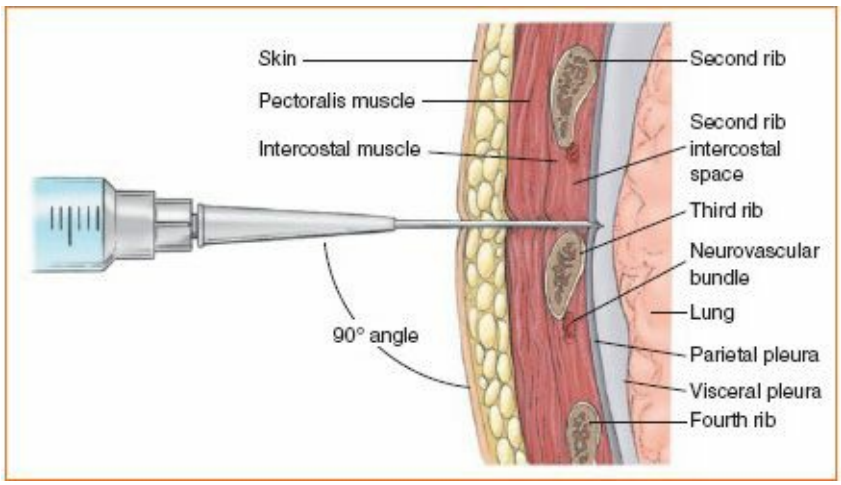


Figure 22-11 Insert the needle over the top of the rib margin in the second intercostal space at the midclavicular line. Nerves and blood vessels run below the ribs, so avoid piercing this area.

Resuscitation in a Neonate Beyond the Newborn Period

The neonate must be assessed using a systematic approach. Begin a rapid visual assessment of the child's overall appearance, work of breathing, and circulation. This can generally be completed within the first few seconds of the patient encounter.

- A rapid assessment to evaluate cardiopulmonary and neurologic function includes assessment of vital signs and pulse oximetry.
- Obtain a focused medical history and a thorough head-to-toe physical exam. Categorize the clinical condition by type and severity (respiratory to include upper/lower airway obstruction, lung disease, or disorder of control of breathing) and cardiovascular to include hypovolemia or cardiogenic shock.

The following is a modification of the pediatric advanced life support as applicable to the newborn and neonate.

A general assessment includes assessment of appearance (muscle tone, interaction, consolability, gaze, cry), work of breathing (nasal flaring, reduced or absent respiratory effort), and abnormal breath sounds (wheezing, grunting, stridor). Evaluation of circulation includes abnormal skin color (pallor, mottling) or bleeding. Determine if the condition is life threatening.

Primary Assessment Includes Airway, Breathing, and Circulation

Assessment of the airway is essential to determine if it is patent. Look for movement of the chest or abdomen. Listen for breath sounds and feel the movement of air at the nose and mouth. Increased inspiratory effort with retractions, snoring or high-pitched stridor, and periods where no breath sounds are heard suggest upper airway obstruction. Attempt to open and maintain the airway by use of the head tilt-chin lift or jaw-thrust maneuver. Suction the nose and pharynx. Attempt foreign body removal by back slaps and chest thrusts in a child younger than 1 year. Intubation may be required if these measures do not work. A respiratory rate of greater than 60 breaths/min is abnormal at any age in a child. A decreasing or irregular respiratory rate may suggest worsening of the clinical condition, especially if associated with a worsening level of consciousness. A decreased respiratory rate or an irregular respiratory rate in an acutely ill child is an ominous clinical sign because it often signals impending cardiopulmonary arrest.

Assessment of circulation includes both cardiovascular function and end-organ function.

Cardiovascular function is assessed by skin color, temperature, heart rate, rhythm, blood pressure, peripheral and central pulses, and capillary refill time. End-organ function is assessed by evaluation of mental status (brain perfusion), skin perfusion, and renal output. A systolic blood pressure of less than 60 mm Hg in a term infant up to 1 month of age and less than 70 mm Hg up to 6 months of age is an ominous sign. Hypotension represents a state of shock in which physiologic compensatory mechanisms (tachycardia, vasoconstriction) have failed. Hypotension with hemorrhage is consistent with about a 20% to 25% loss in circulating volume, whereas hypotension in septic shock represents inappropriate vasodilation rather than loss of circulating volume. Aggressive fluid resuscitation along with management of airway and breathing are needed to prevent cardiac arrest.

TABLE 22-10 Management of Respiratory Distress and Failure

Assessment Parameter	Action as Indicated
Airway	Support the airway. Clear the airway (suction the nose and mouth as needed). Insert an oropharyngeal airway if indicated.
Breathing	Assist ventilation (bag-mask ventilation or intubation). Provide oxygen (humidified if available). Begin with 100% and wean as needed based on oximetry. Continuously monitor oxygen saturation by pulse oximetry. Prepare for intubation as indicated.
Circulation	Monitor heart rate and rhythm. Establish vascular access for fluid therapy and medications.

Alteration in neurologic signs may be caused by conditions other than cerebral hypoxia. Some drugs and metabolic conditions such as rising ammonia from an inborn error of metabolism or increased intracranial pressure may cause neurologic signs. Careful evaluation of pallor, mottling, and cyanosis may indicate inadequate oxygen delivery to the tissues. Acrocyanosis (bluish discoloration of hands and feet) is often seen in healthy newborns, and unlike central cyanosis, is not associated with hypoxia.

Management of Respiratory Distress and Failure

Respiratory problems are a major cause of cardiac arrest in newborns and infants. Clinical deterioration in respiratory function may progress rapidly; therefore, prompt recognition and effective management are fundamental to pediatric life support. The primary goal is to restore adequate oxygenation and ventilation. **Table 22-10** lists measures for ensuring airway, breathing, and circulation.

Cardiovascular Conditions

Cyanosis

Cyanosis (blue coloration of gums and face) is caused by decreased arterial oxygenation. **Acrocyanosis**, or cyanosis of the extremities, with pink color centrally is a benign, common condition that presents as bluish coloration of the hands and feet and is often seen in newborns secondary to cold stress and

peripheral vasoconstriction.

Cyanosis often presents in association with poor perfusion and congestive heart failure. It may also present with shock or the result of either a pulmonary or cardiac process. Cardiac causes of cyanosis include cyanotic congenital heart disease, arrhythmia, cardiomyopathy, patent ductus arteriosus, and myocarditis. Noncardiac causes of cyanosis include persistent pulmonary hypertension (PPHN), sepsis/pneumonia, RDS, aspiration pneumonitis (meconium/blood), severe anemia, and arteriovenous malformation.

Signs and Symptoms

Cyanosis

- Blue coloration of gums and face

Differential Diagnosis

Cyanosis

- Congestive heart failure
- Shock
- Cyanotic congenital heart disease
- Arrhythmia
- Cardiomyopathy
- PDA
- Myocarditis
- Pulmonary hypertension (PPHN)
- Sepsis/pneumonia
- RDS
- Aspiration pneumonitis (meconium/blood)
- Severe anemia
- Arteriovenous malformation

Transport Management

Cyanosis

- Provide oxygen.
- Ventilate with bag-mask device as needed.
- Consider transfusion as needed.

Cyanotic Congenital Heart Disease

Cyanotic congenital heart disease (CHD) is caused by developmental anomalies of the heart or major vessels leading to abnormal blood flow. Deoxygenated blood from the right side of the heart is shunted to the left side, causing cyanosis. The cyanotic heart lesions, known as “The 5 Ts” are truncus arteriosus,

transposition of the great vessels, tetralogy of Fallot, tricuspid atresia, and total anomalous pulmonary venous return. A hypoplastic left heart also leads to cyanosis.

Management of cyanosis in a neonate suspected of CHD includes assessment of the ABCs, monitoring blood pressure and oxygen saturation (preferably upper and lower saturations to follow for PPHN), optimizing oxygenation to prevent acidosis, and supporting cardiovascular function. Administer oxygen to the infant, using a nonrebreathing mask if needed, to keep the oxygen saturation level > 70%. Hyperoxygenation can be harmful because it reduces pulmonary vascular resistance and diverts blood from systemic circulation to pulmonary circulation. For the same reason, hyperventilation can be harmful to the infant as well. Consider intubation if respiratory distress is severe. Obtain IV access as soon as possible. Obtain a CBC with platelet count and differential count, and perform electrolyte and blood gas determinations. Measuring arterial blood gas helps determine the degree of oxygenation/perfusion and ventilation. Ensuring that laboratory tests for lactate and hemoglobin with blood gases have been obtained is a quick way of determining the extent of acidosis and anemia. Simultaneous determinations of blood gases from a preductal and postductal extremity, ie, right arm (preductal) and left leg (postductal), are also useful. A difference in Pa_{O_2} (preductal–postductal) of greater than 15 mm Hg indicates a diagnosis of PPHN. Sodium bicarbonate may be needed if poor perfusion has caused significant metabolic acidosis (check base excess on the blood gas). Obtain four limb blood pressures to help determine the level of stenosis/obstruction to blood flow. If possible, perform a hyperoxia test because it is a very important test to distinguish cardiac from noncardiac (lung disease) causes of cyanosis. This test is performed by placing the infant in an **oxygen hood** with 100% oxygen for 20 minutes. Obtain a blood gas measurement. A Pa_{O_2} measurement greater than 150 mm Hg on blood gas analysis essentially rules out cardiac causes of cyanosis because deoxygenated blood bypasses the lungs and is shunted to the left side of heart in the case of CHD. The exception to this rule is total anomalous pulmonary venous return, in which oxygenated blood returns to the right side of the heart, leading to progressive pulmonary edema. Because results from a blood gas analysis may not be readily available, upper and lower oxygen saturations can be obtained. A preductal (right upper extremity) upper saturation more than 10 points higher than a postductal (lower extremity) saturation suggests pulmonary hypertension. Hyperoxia with hyperventilation supports PPHN as the cause of cyanosis. Correct any other metabolic abnormalities that may be found on lab analysis. Consider prostaglandin administration in discussions with the supervising physician. In the case of ductal-dependent congenital heart lesions (transposition of the great arteries, left ventricular outflow obstruction), timely infusion of prostaglandin E_1 is crucial. It keeps the ductus arteriosus open. Prostaglandin is not indicated in nonductal-dependent lesions. An echocardiogram is used to assess the type of CHD and determine the presence of pulmonary hypertension; however, this is often not available when transferring a neonate from a community hospital to a higher level facility. If the diagnosis of CHD is suspected but uncertain during transport and the patient is severely acidotic, hypoxic, hypotensive, or poorly perfused, discuss a trial of prostaglandins with the supervising physician. The infant may need to be intubated for the infusion since prostaglandins can cause apneas. Other side effects of prostaglandins are seizures, bradycardia, hyperthermia, and hypotension. Follow vital signs closely throughout the transport. The infant may have congestive heart failure and low blood pressure/delayed capillary refill in addition to cyanosis. Management of hypotension includes a normal saline bolus as a first line of therapy. After ensuring adequate volume expansion, consider inotropes or pressors. Dobutamine and dopamine are agents for supporting blood pressure. The dose is 5 to 20 μg/kg/min. Administer sodium bicarbonate for severe acidosis.

Signs and Symptoms

Cyanotic Congenital Heart Disease

- Cyanosis
- Respiratory distress

Differential Diagnosis

Cyanotic Congenital Heart Disease

- Truncus arteriosus
- Transposition of great vessels
- Tetralogy of Fallot
- Tricuspid atresia
- Total anomalous pulmonary venous return
- Hypoplastic left heart

Transport Management

Cyanotic Congenital Heart Disease

- Discuss a trial of prostaglandins with the supervising physician if the infant is severely acidotic, hypoxic, hypotensive, or poorly perfused.
- Follow vital signs closely.
- Manage hypotension, if present.
- Manage acidosis.

Recognition and Management of Tachyarrhythmias and Bradyarrhythmias

Sinus tachycardia is defined as a heart rate faster than normal for the child's age. Heart rate generally varies with activity and other factors influencing oxygen demand (ie, the infant's temperature). Common pathologic causes for tachycardia include tissue hypoxia, hypovolemia, fever, metabolic stress, pain, anxiety, drugs, and anemia.

Supraventricular tachycardia (SVT) is an abnormally fast rhythm originating above the ventricles. It is the most common cause of tachyarrhythmia producing cardiovascular compromise in infancy. It is commonly caused by a reentry mechanism that involves an accessory pathway, atrioventricular nodal reentry, or ectopic atrial focus. Wolff-Parkinson-White syndrome, in which ventricular preexcitation produces a delta wave on electrocardiogram, is an example of a condition that produces SVT via an accessory pathway. SVT is often tolerated well in infants. They often present with symptoms of congestive cardiac failure, poor feeding, rapid breathing, irritability, pale or blue color, and vomiting. Initial management includes determination of pulse and signs of adequate perfusion, supporting the ABCs and oxygenation as needed, obtaining an electrocardiogram to determine the rhythm, and obtaining vascular access and establishing monitoring by a cardiopulmonary monitor and/or pulse oximeter. Specific emergency intervention is dictated by the condition of the infant and varies depending on the width of the observed QRS complex on the electrocardiogram. Vagal maneuvers, cardioversion, and pharmacologic interventions are among the options available, but should be performed under the

supervision of a credentialed provider of such care. Children with tachyarrhythmias need to be evaluated by a pediatric cardiologist, but this should not delay appropriate emergency treatment.

Signs and Symptoms

Supraventricular Tachycardia

- Symptoms of congestive heart failure
- Poor feeding
- Rapid breathing
- Irritability
- Pale or blue color
- Vomiting

Differential Diagnosis

Supraventricular Tachycardia

- Wolf-Parkinson-White syndrome
- Sinus tachycardia

Transport Management

Supraventricular Tachycardia

- Support ABCs and oxygenation as needed.
- Monitor by cardiopulmonary monitor and/or pulse oximeter.
- Perform vagal maneuvers, cardioversion, and pharmacologic interventions based on protocol.

Bradycardia in a neonate is usually a result of tissue hypoxia and resolves when tissue oxygenation is restored. The most common neonatal bradyarrhythmia is due to maternal lupus, resulting in heart block. If the resting fetal heart rate is low enough, hydrops (edema) develops. Postdelivery cardiac pacing may need to be initiated, but it is usually nonurgent.

Recognition and Management of Cardiac Arrest

In contrast to cardiac arrest in adults, sudden cardiac arrest in children is uncommon. Cardiac arrest is more often caused by progression of respiratory distress, respiratory failure, or shock, typically associated with hypoxemia and acidosis, than from cardiac arrhythmias. Sudden infant death syndrome has decreased significantly in recent years with the “back to sleep” campaign, which instructs parents to place the infant on its back to sleep. Trauma is an important cause of cardiopulmonary collapse in infants older than 6 months. Details of infant resuscitation, including the importance of adequate ventilation, have already been discussed.

Persistent Pulmonary Hypertension

Persistent pulmonary hypertension (PPHN) is persistence of elevated pressures in pulmonary vasculature after birth. It is associated with failure to transition from fetal circulation to postpartum or normal newborn circulation. The elevated pulmonary blood pressure causes ineffective pulmonary perfusion, hypoxemia, and right-to-left shunting of blood via the patent foramen ovale and/or patent ductus arteriosus. PPHN presents mostly in term or post-term neonates, within the first few hours of life. PPHN frequently presents with a combination of cyanosis, tachypnea, hypoxemia (often labile/fluctuating, respiratory distress, and differential oxygen saturations [upper > lower]).

The exact cause of PPHN is not understood. However, several factors contribute to the failure of normal circulatory transition at birth. Pulmonary vasoconstriction often occurs secondary to perinatal events. The most potent vasoconstrictors of the pulmonary artery are hypoxia and acidosis. Meconium aspiration is the most common cause of PPHN, often causing perinatal distress. Additional conditions and risk factors associated with PPHN are RDS, asphyxia, pneumonia, hypothermia, hypoglycemia, and sepsis.

Abnormal development of the pulmonary vasculature is more frequently seen if the mother has taken NSAIDs during pregnancy and in infants of diabetic mothers. It is also common in chronic fetal distress/hypoxia resulting in hypertrophy of the vascular muscles of the pulmonary artery (characterized by idiopathic or black lung PPHN, because there is no parenchymal disease seen on a chest radiograph), aspiration, hypoxic ischemic encephalopathy, and congenital diaphragmatic hernia.

It is important to have a low threshold of suspicion for PPHN. Care should be taken to optimize oxygenation, minimize stress, maintain a normal blood pressure, and avoid or correct acidosis. Obtain a CBC, differential count, platelet count, blood gas analysis for hyperoxia, and follow upper and lower oxygen saturations while treating the underlying cause of the PPHN.

Signs and Symptoms

Persistent Pulmonary Hypertension

- Cyanosis
- Tachypnea
- Hypoxemia (often labile/fluctuating)
- Respiratory distress
- Differential oxygen saturations

Differential Diagnosis

Persistent Pulmonary Hypertension

- Meconium aspiration
- RDS
- Asphyxia
- Pneumonia
- Hypothermia
- Hypoglycemia
- Sepsis

Transport Management

Persistent Pulmonary Hypertension

- Optimize oxygenation.
- Minimize stress.
- Maintain normal blood pressure.
- Avoid or correct acidosis.

Acidosis

Respiratory acidosis may be caused by maternal drug use that results in respiratory depression in the neonate or a primary pulmonary or neurologic cause for suboptimal gas exchange. Respiratory acidosis frequently presents with visually obvious hypoventilation or increased work of breathing, and often responds to assisted ventilation. Metabolic acidosis results when cations, frequently lactic acid from poor tissue perfusion or toxic byproducts in inborn errors of metabolism, build up in the bloodstream. Causes of metabolic acidosis include asphyxia, congenital heart disease, sepsis, inborn errors of metabolism, hypovolemia, seizures, bradycardia, or hypotension. The presentation of metabolic acidosis may include compensatory tachypnea, mottled/gray skin with delayed capillary refill (normally < 3 seconds) apnea, lethargy, hypertonia or hypotonia, feeding intolerance, seizures, or emesis. Management of metabolic acidosis includes managing the ABCs, providing adequate oxygenation, ventilation, and hydration, and treating the underlying cause. If adequate ventilation and fluid resuscitation fail to clear the metabolic acidosis, sodium bicarbonate can be given at a dose of 0.3 × the base deficit on the blood gas analysis × the neonate's weight in kilograms. If the exact deficit is not known, a dose of 2 mEq/kg can be given. In premature neonates, the dose should be given over at least 30 minutes to minimize the risk of intraventricular hemorrhage. If inborn error of metabolism is suspected, enteral feeding should be stopped, and IV dextrose should be initiated to minimize production of toxic byproducts. Critically ill neo-nates often have a mixed metabolic and respiratory acidosis.

Signs and Symptoms

Metabolic Acidosis

- Compensatory tachypnea
- Mottled/gray skin with delayed capillary refill
- Apnea
- Lethargy
- Hypertonia/hypotonia
- Feeding intolerance
- Seizures
- Emesis

Differential Diagnosis

Metabolic Acidosis

- Asphyxia
- Congenital heart disease
- Sepsis
- Inborn errors of metabolism
- Hypovolemia
- Seizures
- Bradycardia
- Hypotension

Transport Management

Metabolic Acidosis

- Manage the ABCs.
- Provide adequate oxygenation, ventilation, and hydration.
- Treat the underlying cause.

Shock

Shock is a serious condition where perfusion is inadequate to meet tissue demands. All organ systems can be affected by shock. Both lung ventilation and tissue perfusion are needed to maintain oxygen delivery to the tissues. Hypovolemia is a common cause of shock in a neonate. Causes include hypovolemia from acute blood loss or fluid losses from decreased intake, polyuric renal failure, or diarrhea. Cardiogenic shock can follow congenital heart disease, arrhythmias, myocardial ischemia from asphyxia, cardiac tamponade, pneumothorax, or high intrathoracic pressure from PPV with secondary decreased venous return. Distributive shock includes sepsis, cardiac depression, and vasodilation.

Mean blood pressure is often used in a neonate, especially in the preterm population, where the lowest acceptable mean blood pressure is equal to the gestation age of the neonate (for example, a mean arterial pressure of 28 mm Hg in a neonate at 28 weeks' gestation and a mean arterial pressure of 38 mm Hg in a neonate at 38 weeks' gestation).

Shock presents with some, if not all, of the following: hypotension, tachycardia (or bradycardia in cases of asphyxia), poor perfusion, tachypnea, oliguria/anuria, hypothermia, acidemia, and weak pulses. Hepatomegaly, cardiomegaly, and peripheral edema are seen in cardiogenic shock. Absence of a heart murmur does not rule out cardiogenic shock. Disseminated intravascular coagulopathy may be a late presentation.

The management of shock includes managing the ABCs, checking blood glucose levels, obtaining vascular access, providing fluid resuscitation, and treating the underlying cause. If it is necessary to provide fluids to support circulation or deliver resuscitation medications intravenously to a neonate, emergent access must be obtained. It is usually difficult to establish peripheral access in an infant who needs volume or resuscitation medications. The umbilical vein can be catheterized using an umbilical vein catheter in a newborn using the following steps:

1. Clean the cord with alcohol or another antiseptic.
2. Place a sterile tie firmly but not too tightly around the base of the cord for control of bleeding. If available, place a sterile drape over the site. While the line needs to be placed quickly in a code situation, maintain sterile technique as much as possible.

3. Prefill a sterile 3.5F to 5F umbilical vein line catheter (a comparable sized sterile feeding tube can be used in an emergency) with normal saline using a 3-mL syringe.
4. Using sterile technique and after a tie is wrapped around the base of the cord to control bleeding, cut the cord with a scalpel below the clamp placed on the cord at birth about 1 to 2 cm from the skin (between the clamp and the cord tie).

Signs and Symptoms

Shock

- Hypotension
- Tachycardia (or bradycardia in cases of asphyxia)
- Poor perfusion
- Tachypnea
- Oliguria/anuria
- Hypothermia
- Academia
- Weak pulses
- Hepatomegaly, cardiomegaly, and peripheral edema in cardiogenic shock
- Disseminated intravascular coagulopathy

Differential Diagnosis

Shock

- Hypovolemia from acute blood loss or fluid loss
- Polyuric renal failure
- Diarrhea
- Congenital heart disease
- Arrhythmias
- Myocardial ischemia from asphyxia
- Cardiac tamponade
- Pneumothorax
- High intrathoracic pressure from PPV with secondary decreased venous return
- Sepsis
- Cardiac depression
- Vasodilation
- Profound anemia

Transport Management

Shock

- Manage the ABCs.

- Check blood glucose levels.
- Treat the underlying cause.

5. The umbilical vein is a large thin-walled vessel usually at the 12 o'clock position, as compared to the two thick-walled umbilical arteries usually found at 4 and 8 o'clock **Figure 22-12**. Insert the catheter 2 to 4 cm (less in preterm infants) until blood can be aspirated. If the catheter is advanced too far, infusing hypertonic solutions directly into the liver may lead to irreversible damage **Figure 22-13**. If the catheter is advanced into the heart, arrhythmias may develop.
6. Once access is established, flush with 0.5-mL normal saline and tape in place.

For an older neonate (after the first week), emergent IV access is needed but if peripheral IV access cannot be obtained, an intraosseous line can be placed similar to an older child. Care must be used to choose a small size line and to avoid traversing the bone.

Anemia

Anemia is defined as a hematocrit value of less than 38% in preterm neonates and less than 42% in term neonates. A neonate with anemia may be asymptomatic or present with any of the following signs or symptoms, depending on the cause: tachycardia, pallor, petechiae/purpura, jaundice, hepatosplenomegaly, respiratory distress, hydrops, heart failure, visible bleeding/hematoma, cyanosis, shock, or acidosis.

Causes of anemia in a neonate can be categorized by increased destruction, decreased production, or loss of red blood cells. Causes of increased destruction include hemolysis from red blood cell defects (hereditary spherocytosis, glucose 6-phosphate dehydrogenase or pyruvate kinase deficiency, or thalassemia) or immune processes (Rh incompatibility, ABO incompatibility, maternal lupus, maternal penicillin use). Disseminated intravascular coagulopathy, hemangiomas, vitamin E deficiency, or arterial stenosis can also lead to hemolysis. Decreased production can be seen in infections such as parvovirus, rubella, or cytomegalovirus; congenital leukemia; Diamond-Blackfan syndrome; or trisomy 21 (Down syndrome). Hemorrhage can occur *in utero*, with fetal-maternal hemorrhage or twin-twin transfusion, in the perinatal or neonatal period with abruptio placentae, placenta previa, velamentous insertion of the cord on the placenta, cord rupture, fetomaternal bleeding, cephalohematoma, subgaleal/intracranial hemorrhage, splenic hemorrhage, intracranial hemorrhage, necrotizing enterocolitis, or traumatic injury.

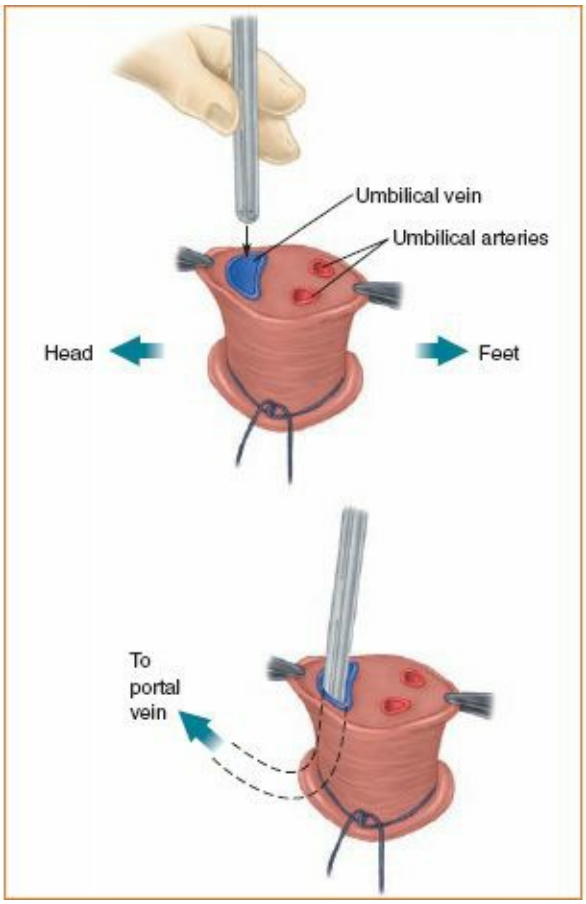


Figure 22-12 Location of the umbilical vein.

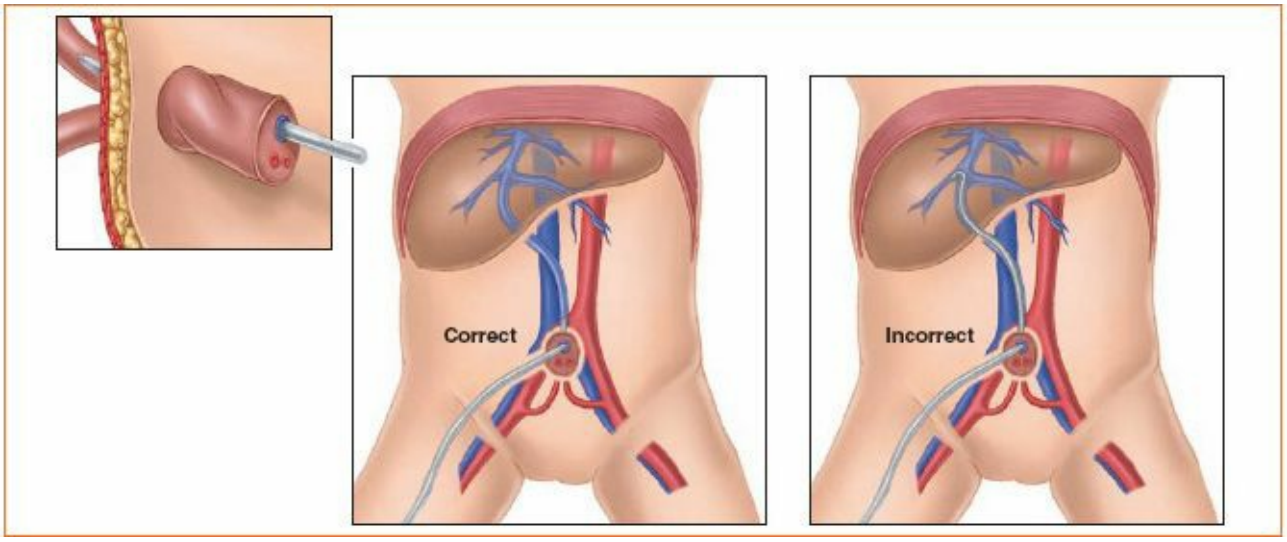


Figure 22-13 Umbilical vein catheterization. Note that if the catheter is advanced too far, infusing hypertonic solutions directly into the liver may lead to irreversible damage.

Rh incompatibility is a serious situation that can have dire consequences. If a pregnant woman's blood type is Rh negative and the fetus's blood type is Rh positive, the woman can be sensitized to the Rh-positive blood, making antibodies against it, when small amounts of fetal blood get into her circulation. Rhogam is routinely given to Rh-negative mothers during and immediately after delivery to minimize this risk, but sensitization still occurs, such as after unrecognized spontaneous early miscarriages. In subsequent pregnancies, if the fetus is Rh positive, maternal antibodies can cross the placental barrier and destroy the Rh-positive fetal red blood cells, leading to severe anemia and ultimately heart failure and hydrops.

Signs and Symptoms

Anemia

- Tachycardia
- Pallor
- Petechiae/purpura
- Jaundice
- Hepatosplenomegaly
- Respiratory distress
- Hydrops
- Heart failure
- Visible bleeding/hematoma
- Cyanosis
- Shock
- Acidosis

Differential Diagnosis

Anemia

- Hereditary spherocytosis
- Glucose-6-phosphate dehydrogenase or pyruvate kinase deficiency
- Thalassemia
- Rh incompatibility
- ABO incompatibility
- Maternal lupus
- Maternal penicillin use
- Disseminated intravascular coagulopathy
- Hemangiomas
- Vitamin E deficiency
- Arterial stenosis
- Parvovirus
- Rubella
- Cytomegalovirus
- Congenital leukemia
- Diamond-Blackfan syndrome
- Trisomy 21
- Hemorrhage *in utero* or in the perinatal or neonatal period

Transport Management

Anemia

- Manage the ABCs.
- Transfuse as ordered/indicated.

Management of anemia includes managing the ABCs and obtaining IV access (optimally two sites). Initially the following lab values should be obtained in consultation with a physician: a blood type and crossmatch, CBC, **peripheral blood smear** (allows the lab to microscopically examine the structure of the blood cells), reticulocyte count, **Coombs' test**, bilirubin level, liver function tests, a **TORCH screen** (a blood test that checks for several infections in a newborn, such as toxoplasmosis, rubella, cytomegalovirus, and herpes simplex), cultures of scrapings from any vesicular lesions, electrolyte levels, and coagulation factors. (TORCH is a set of infections pregnant women are susceptible to that result in fetal deformities.) The neonate may be in shock. Volume resuscitation with a 20-mL/kg bolus of normal saline followed by the administration of blood is crucial. If the anemia is chronic, the infant is at risk of volume overload after transfusion. The supervising physician may need to perform an exchange transfusion to avoid cardiac collapse. If coagulopathy is suspected, use packed red blood cells and fresh-frozen plasma.

Gastrointestinal Conditions

Any part of the gastrointestinal tract may have congenital anomalies. The most common lesions are **atresias** (complete absence of lumen), stenosis (narrowing), duplications of loops of bowel, and functional obstructions.

Presenting features may include increased salivation; choking with feeding; cyanosis; vomiting, especially if bile stained; abdominal distention; blood in stool or failure to stool; lethargy; or irritability. Physical findings may include a distended, discolored (erythematous), tender abdomen; absent bowel sounds; or lethargy and poor perfusion. Important principles include the knowledge that bilious (green or yellow) vomiting is considered an indication of an obstruction until proven otherwise, passage of meconium does not rule out an obstructed bowel, and an obstructed bowel can lead to perforation and peritonitis.

Abdominal Wall Defects

Gastroschisis and **omphalocele** are abdominal wall defects that can be distinguished by their presentation **Table 22-11**. Gastroschisis is a full-thickness defect of the abdominal wall, usually to the right and adjacent to the intact umbilical cord. Variable amounts of edematous intestine, stomach, and fallopian tube extrude from the defect. The liver and spleen are normally situated in the abdomen. The extruded intestine does not have a protective covering. Omphalocele is a herniation of the abdominal contents into the umbilical cord. A protective membrane covers the herniated contents (unless ruptured during the birth process). Gastroschisis is usually an isolated defect, whereas omphalocele has an association with congenital defects, such as a congenital diaphragmatic hernia, and other anomalies.

Ideally, these infants should be delivered at a regional perinatal center that has immediate access to a neonatal intensive care unit and pediatric surgery. However, occasionally these infants deliver at remote locations even when the condition may have been diagnosed prenatally. Under those circumstances, newborn transport should be expedited, and care coordinated between obstetricians, pediatricians, and surgeons. Immediate care includes initial resuscitation, then placing the infant in a clear sterile bag to cover and protect the exposed abdominal contents to limit fluid and protein loss and prevent hypothermia. Handling of the bowel should be minimized to decrease vascular compromise. In a large defect, the infant may be placed right side down to minimize stretching of the mesenteric vessels. The intestines should be

visualized throughout the transport, and if they begin to look dusky, they should be repositioned to optimize blood flow. A nasogastric or orogastric tube should be maintained at low intermittent suction. A wide-bore double-lumen **repogle** tube, if available, is ideal **Figure 22-14**. A repogle tube is a double-lumen tube used for gastric decompression in infants with gastrointestinal issues; for an infant, usually an 8F is used and is connected to low intermittent wall suction to collect gastric secretions. Vascular access should be maintained. Generous IV fluids of $D_{10}W$ are initiated at approximately 120 to 150 mL/kg/d because of increased fluid loss and third space deficits. Provide necessary hemodynamic support with fluids and pressors, if necessary. Broad-spectrum antibiotics should be used before initiating the transport given the increased risk of infection in these infants.

TABLE 22-11 Comparison of Gastroschisis and Omphalocele

	Gastroschisis	Omphalocele
Definition	Centrally located, full-thickness abdominal wall defect	Herniation of abdominal contents into base of cord
Features	2- to 4-cm defect Right side of cord Intact umbilical cord Uncovered intestine, thick, edematous, matted Liver, spleen in normal position	Different sizes Protective membrane covers abdominal contents Elements of umbilical cord course over the sac and come together at the top to form a normal umbilical cord. Large omphalocele may contain the liver and/or spleen.
Associated abnormalities	Isolated defect	25% to 40% of infants have associated chromosomal, cardiac, neurologic, genitourinary, skeletal, or other gastrointestinal defects.

An intact omphalocele is a less urgent surgical problem than a gastroschisis or ruptured omphalocele because the intact membrane conserves heat and protects the intestine. The sac should be carefully protected. Surgical consultation should be obtained as soon as possible, initially by telephone, so that a surgical plan can be developed. Provide urgent transport to an appropriate pediatric surgical facility.

Gastrointestinal Obstruction and Vomiting

The causes of vomiting can be differentiated by the presence or absence of bile, which turns the vomitus green. Presence of bile must be treated as a surgical emergency until obstruction, especially **malrotation**, has been ruled out. Bile-stained vomiting may occasionally be seen without intestinal obstruction, such as with a septic ileus. In these situations, it is often present without abdominal distention and does not continue to recur.

The differential diagnosis of bilious vomiting includes malrotation with volvulus, intestinal atresia, or annular pancreas. The differential diagnosis of nonbilious vomiting includes esophageal atresia, gastroesophageal reflux, overfeeding (excessive volume of feed), milk or formula intolerance, sepsis, or obstructive lesions above the ampulla of Vater such as pyloric stenosis or partial intestinal obstruction.

Congenital gastrointestinal obstruction should be considered when polyhydramnios (extra amniotic fluid) is present. **Esophageal atresia** is suspected when the infant presents with increased salivation and

chokes on feeding. Vomitus with esophageal atresia is never bile stained, and the infant often develops respiratory distress from aspiration. Examine the infant for other abnormalities such as anal atresia (an abnormal or absent anal opening). The diagnosis is further substantiated by inability to pass a nasogastric tube to the stomach because the tube coils in the esophageal pouch. Stomach contents may be obtained when suctioning the ET tube of an intubated baby with esophageal atresia with a tracheoesophageal fistula. When trying to document an esophageal atresia, use as large a tube that will pass through the nares. Atresia can be confirmed by obtaining a chest radiograph that includes the neck and upper abdomen if available. Note the presence or absence of air in the stomach (no air in the stomach suggests that there is an esophageal atresia without a communication between the trachea and the lower esophagus, whereas the presence of air in the stomach suggests the presence of such a connection). Note the presence of other vertebral and/or rib anomalies suggesting Vater syndrome. Diagnosis requires a high index of suspicion. During transport, position the neonate with the head elevated at a 30° angle, place a suction catheter in the upper pouch, and provide intermittent suction. Neonates often do not need to be intubated if the airway is kept clear of secretions, which necessitates frequent oral suctioning.

Figure 22-14 A repogle tube.

Signs and Symptoms

Esophageal Atresia

- Increased salivation
- Choking on feeding
- Nonbilious vomit
- Respiratory distress
- Inability to pass a nasogastric tube to the stomach

Transport Management

Esophageal Atresia

- Position the infant with the head elevated at a 30° angle.
- Place a suction catheter in the upper pouch and provide intermittent suction.
- Keep the airway clear of secretions.

Proximal intestinal obstruction is an obstruction at or above the level of the jejunum. Duodenal

obstruction may be complete (duodenal atresia) or partial (annular pancreas, duodenal atresia, malrotation, and midgut volvulus). The clinical presentation may include bilious vomiting, minimal dilation of the abdomen, or evidence of intestinal ischemia (bloody mucoid stools). There is an increased incidence of duodenal atresia in trisomy 21, where a prenatal ultrasound or abdominal radiograph shows the classic "double-bubble," in which two large collections of gas are present, one in the stomach and the other in the dilated part of the duodenum **Figure 22-15**. The rest of the abdomen is generally gasless in complete obstruction. The exact cause of obstruction may not be evident until laparotomy. Malrotation should be considered, as midgut **volvulus** (twisting of the entire midgut on the superior mesenteric artery pedicle) constitutes a surgical emergency. The ensuing ischemia may result in a nonviable bowel and death, resulting in the phrase known to most pediatricians, "Never let the sun set on bilious emesis." Management includes establishing IV access, fluid resuscitation, broad-spectrum antibiotics, and urgent laparotomy. When volvulus is suspected, surgery should be consulted by telephone before leaving the referring institution to expedite a surgical plan.

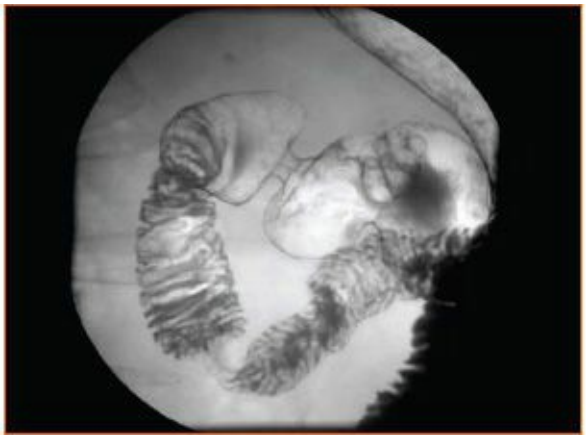


Figure 22-15 An abdominal radiograph showing a collection of gas in the stomach and in the duodenum.

Signs and Symptoms

Proximal Intestinal Obstruction

- Bilious vomiting
- Minimal dilatation of the abdomen
- Evidence of intestinal ischemia (bloody mucoid stools)

Differential Diagnosis

Proximal Intestinal Obstruction

- Duodenal atresia
- Annular pancreas
- Malrotation
- Midgut volvulus

Transport Management

Proximal Intestinal Obstruction

- Maintain thermoregulation.
- Closely monitor vital signs, including oxygen saturation, heart rate, respiratory rate, and blood pressure.
- Ensure strict intake/output.

Distal intestinal obstruction refers to partial or complete obstruction of the distal small bowel (ileum) or large intestine. The differential diagnosis includes ileal atresia, meconium ileus (obstruction of the terminal ileum by inspissated meconium), meconium plug or hypoplastic left colon, colonic atresia, or Hirschsprung's disease (congenital aganglionic colon). The clinical presentation often includes a distended abdomen, failure to pass meconium in the first 24 to 48 hours, and bilious or nonbilious vomiting. An abdominal radiograph often shows multiple dilated loops of bowel **Figure 22-16**. Management considerations include ensuring IV access, hydration, and placement of a nasogastric tube or repogle if available to suction air and avoid further abdominal dilation during transport. Transfer the infant to a pediatric surgical center for specific diagnosis and management.

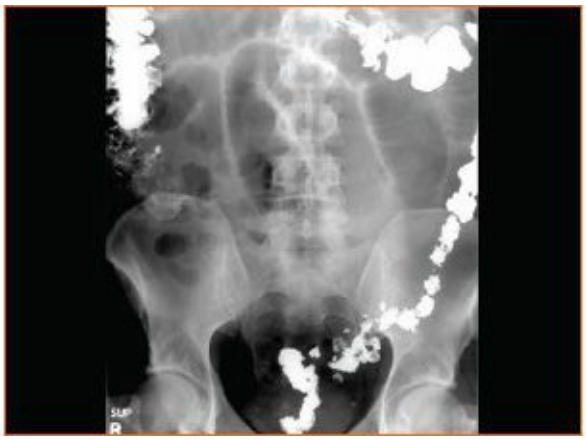


Figure 22-16 An abdominal radiograph after a barium enema showing multiple dilated loops of bowel.

Signs and Symptoms

Distal Intestinal Obstruction

- Distended abdomen
- Failure to pass meconium in the first 24 to 48 hours
- Bilious or nonbilious vomiting

Differential Diagnosis

Distal Intestinal Obstruction

- Ileal atresia
- Meconium ileus
- Meconium plug or hypoplastic left colon
- Colonic atresia

- Hirschsprung's disease

Transport Management

Distal Intestinal Obstruction

- Maintain thermoregulation.
- Closely monitor vital signs, including oxygen saturation, heart rate, respiratory rate, and blood pressure.
- Ensure strict intake/output.

Imperforate anus is a congenital condition where the anal opening is either absent or displaced. Diagnosis is made by inspection of the anal opening and should be made in the delivery room. If the condition is diagnosed shortly after birth, the infant is usually asymptomatic, but a delay in diagnosis can lead to progressive abdominal distention and perforation. On diagnosis, maintain NPO status, establish IV access, administer maintenance IV fluids, and place a nasogastric tube or repogle and provide low intermittent suction to minimize abdominal distention; transfer to a surgical center for evaluation and management should be arranged. These infants have a higher rate of CHD and other anomalies that may further complicate the transport.

In each of these transports, thermoregulation of the neonate should be maintained and close monitoring of vital signs including oxygen saturation, heart rate, respiratory rate, blood pressure, and strict intake/output should occur.

Hirschsprung's disease is a congenital anomaly in which a portion of the intestine lacks ganglion nerve cells. As a result, normal peristalsis does not occur, and stool backs up. If undiagnosed, this can lead to toxic megacolon. Signs include decreased stooling, abdominal distention, and in later stages, shock. Fluid resuscitation and transport to a referral center with pediatric surgery capability can be life saving.

Signs and Symptoms

Hirschsprung's Disease

- Decreased stooling
- Abdominal distention
- Shock

Transport Management

Hirschsprung's Disease

- Fluid resuscitation
- Transport to a referral center with pediatric surgery capability.

Acute Intestinal Perforation

Perforation of the bowel can be caused by an obstructed bowel; necrotizing enterocolitis (NEC)—a disorder seen predominantly in premature infants; iatrogenic perforation (may be caused by suprapubic bladder aspiration or paracentesis); or it may occur spontaneously. It is sometimes seen in preterm infants in association with indomethacin use, especially when combined with hydrocortisone or dexamethasone treatment.

Signs and Symptoms

Acute Intestinal Perforation

- Progressive abdominal distention
- Respiratory distress
- Hypotension
- Acidosis (mixed metabolic and respiratory)
- Feeding intolerance
- Grossly bloody stools

Differential Diagnosis

Acute Intestinal Perforation

- Obstructed bowel
- NEC
- Iatrogenic perforation

Transport Management

Acute Intestinal Perforation

- Maintain NPO status.
- Monitor for development of apnea.
- Intubate the infant if recurrent apnea develops during transport.

The clinical presentation includes progressive abdominal distention, respiratory distress, hypotension, and acidosis (mixed metabolic and respiratory). NEC presents with abdominal distention, feeding intolerance, and grossly bloody stools. The infant often appears septic. The diagnosis is confirmed by an abdominal radiograph of pneumatosis intestinalis (air in the bowel wall), and may be suspected when an abnormal gas pattern, ileus, and a fixed sentinel loop are seen on radiographs. Other features include intra-hepatic portal venous gas. The physical examination and laboratory tests are helpful in making the diagnosis of intestinal perforation. Radiographic studies will show free air in the abdomen that can be confirmed by a cross-table lateral or left lateral decubitus view (right side up). For transport, maintain NPO status. A nasogastric or repogle tube should be placed and low intermittent suction provided to assist decompression of the bowel. These infants are at high risk of shock and severe metabolic acidosis secondary to inflammation and vascular leak. Establish vascular access and provide

10-mL/kg normal saline boluses as needed to support perfusion and blood pressure, in addition to maintenance fluids. Blood cultures and CBC should be drawn and broad-spectrum antibiotics initiated prior to leaving the referring facility. These neonates often develop apnea. A low threshold for intubation before beginning the transport should be maintained. If the infant develops recurrent apnea en route, intubate the infant quickly to avoid further abdominal distention from bag-mask ventilation. Provide transport to an appropriate pediatric surgical facility for further evaluation and surgical intervention. If intestinal perforation is confirmed, notify surgery before leaving the referring institution so a surgical plan can be expedited.

Hematemesis and Bleeding from the Rectum

Bleeding from the upper gastrointestinal tract may be seen in a nasogastric tube aspirate or be associated with vomiting. Blood is generally bright red, though brownish blood may be seen if the bleeding is slow or starts high in the intestines. The differential diagnosis includes idiopathic (no clear cause, generally resolves within a few days), swallowed maternal blood (accounts for about 10% of cases; the infant may swallow blood during a cesarean section, occasionally during a vaginal delivery), nasogastric trauma from a nasogastric tube, a stress ulcer (often seen in sick neonates), coagulopathy (hemorrhagic disease of the newborn [vitamin K deficiency] or disseminated intravascular coagulation), bleeding Meckel's diverticulum, necrotizing entrocolitis, intestinal fissure, formula intolerance (usually cow's milk protein allergy), maternal bleeding nipples in breast-fed infants, or volvulus. Volvulus is the abnormal twisting of the intestine. Volvulus can lead to gangrene and death of the segment of intestine, intestinal obstruction, perforation peritonitis, and death of the patient. Midgut volvulus occurs in infants with malrotation of the bowel during fetal development. Segmental volvulus occurs at any age and can be caused by abnormal intestinal contents (meconium ileus); sigmoid volvulus is usually the result of a dilated rectosigmoid on a narrow pedicle. Volvulus often has a sudden onset. The symptoms and signs may include abdominal pain, nausea, vomiting, and blood in the stool. Intestinal volvulus is a surgical emergency (release of the abnormal twisting and ensuring normal blood supply). If suspected, keep the patient at NPO status, establish IV access, begin maintenance fluids, decompress the intestines with a nasogastric tube or repogle, provide low intermittent suction, and expedite transfer to a center with pediatric surgical services.

Signs and Symptoms

Volvulus

- Abdominal pain
- Nausea
- Vomiting
- Blood in the stool

Differential Diagnosis

Volvulus

- Malrotation of the bowel during fetal development
- Abnormal intestinal contents (meconium ileus)
- Dilated rectosigmoid on a narrow pedicle

Transport Management

Volvulus

- Maintain NPO status.
- Provide maintenance fluids.
- Decompress the intestines with a nasogastric tube or repogle.
- Provide low intermittent suction.

Intussusception

Intussusception is the telescoping of one segment of intestine into another adjacent distal segment of the intestine. Intussusception is the most common cause of intestinal obstruction in children between 3 months and 6 years of age. It is extremely rare in children younger than 3 months of age or in older children and adults, and can present with passage of bloody stool, often described as currant jelly stool due to its resemblance to that food. When suspected, keep the patient at NPO status, establish IV access, begin maintenance fluids, decompress the intestines with a nasogastric tube or repogle, provide low intermittent suction, and expedite transfer to a center with pediatric radiology and surgical services, because the intussusception may be reduced by enema, but frequently needs open reduction.

Signs and Symptoms

Intussusception

- Bloody stools, often described as currant jelly stool

Transport Management

Intussusception

- Maintain NPO status.
- Provide maintenance fluids.
- Decompress the intestines with a nasogastric tube or repogle.
- Provide low intermittent suction.

Diarrhea

Diarrhea in a neonate is unusual because the infant still has maternal immunoglobulins that help aid in minimizing the risk of contracting diarrheal illness in the first few weeks of life. When transferring a neonate with diarrhea, the infant should be maintained at NPO status, IV access established, and fluid resuscitation provided as clinically indicated. Maintenance fluids should be continued en route to minimize further dehydration and hypoglycemia. Vital signs should be monitored throughout, and attention paid to maintaining normothermia.

Transport Management

Diarrhea

- Maintain NPO status.
- Provide maintenance fluids.
- Monitor vital signs.
- Maintain normothermia.

Infectious Diseases/Sepsis

Neonates are particularly prone to becoming compromised by serious infectious disease because their immune system is not fully developed. Signs of infection in a neonate include decreased activity (lethargy), hypothermia, hypoglycemia, poor perfusion, low blood pressure, and apnea. Neonates are at higher risk of infection with group B *Streptococcus* and gram-negative bacteria, which frequently respond to ampicillin and gentamicin. Primary assessment includes a blood culture, urine culture, and lumbar puncture followed by broad-spectrum antibiotics. A chest radiograph is taken if respiratory symptoms are present. Viral illness can also lead to dire illness in a neonate. Regardless of the causative organism, it is important to support the infant's cardiorespiratory state and aggressively fluid resuscitate if signs of shock are present. Monitor vital signs and maintain normothermia while transferring to a center that can manage a critically ill neonate because the infant's condition can rapidly deteriorate.

Signs and Symptoms

Infectious Diseases/Sepsis

- Decreased activity (lethargy)
- Hypothermia
- Poor perfusion
- Low blood pressure
- Apnea

Transport Management

Infectious Diseases/Sepsis

- Monitor vital signs.
- Maintain normothermia.

Hyperthermia/Hypothermia

Hyperthermia (temperature > 99.5°F [37.5°C]) in a neonate is usually caused by overbundling or exposure to excessive heat. It can also be seen with herpes simplex infection. Hypothermia in the neonate, a drop in body temperature to less than 97.5°F (36.4°C), occurs in all climates, but is more common during the winter months. It can also be an early sign of sepsis.

Neonates have increased surface area-to-volume ratio, making them extremely sensitive to environmental conditions, especially when wet after delivery. An increase in metabolic function in an attempt to overcome the heat loss can cause hypoglycemia, metabolic acidosis, pulmonary hypertension, and hypoxemia. Infection should be considered as an etiology in any hypothermic neonate.

Hypothermic neonates are cool to the touch, initially in the extremities; as their temperature drops, however, the skin becomes cool all over. The infant may also be pale and have acrocyanosis. The hypothermic neonate may present with apnea, bradycardia, cyanosis, irritability, and a weak cry. As a neonate's temperature drops, he or she may become lethargic and obtunded. In severely hypothermic babies, the face and extremities may appear bright red. Sclerema—hardening of the skin associated with reddening and edema—may be seen on the back, limbs, or all over the body. Thermal shock, disseminated intravascular coagulopathy, and death may occur in more serious cases.

Preventive measures include warming your hands before touching the baby, using prewarmed blankets and equipment, and placing a cap on the neonate's head since the head is the largest source of heat loss. The critically ill neonate, once stabilized, should be placed in a prewarmed incubator or, if none is available, covered with warm blankets for transport. Recent studies in neonates with hypoxic-ischemic injury indicate improved outcomes when the infant is provided mild therapeutic hypothermia within 6 hours of birth. This approach is not recommended in the field, although it is prudent to prevent hyperthermia. Maintain the infant at the lower margin of normal temperature (axillary temperature no higher than 97.5°F [36.4°C]).

Signs and Symptoms

Hypothermia

- Temperature below 97.5°F (36.4°C)
- Skin that is cool to the touch
- Apnea
- Bradycardia
- Cyanosis
- Irritability
- Weak cry
- Lethargy
- Sclerema
- Thermal shock
- Disseminated intravascular coagulopathy

Transport Management

Hypothermia

- Maintain the infant at the lower margin of normal temperature (axillary temperature no higher than 97.5°F [36.4°C]).

Toxic Exposure

Most toxic exposures in neonates are from transplacental exposure (eg, maternal alcohol or narcotic use), or purposeful administration by another individual such as a well-meaning younger child. The most important principle of managing toxic exposure/ingestion is stabilization of the neonate. Manage the patient's ABCs. Obtain IV access as indicated based on history and physical exam. Be sure to obtain the history from the sending facility. In the case of respiratory suppression from narcotics, which is much more likely to be from chronic maternal exposure, respiratory support should be provided until the infant is transferred to a tertiary hospital. If respiratory depression is suspected to be from acute narcotic exposure, such as when an infant delivers shortly after a dose of maternal narcotic for labor pain control, naloxone, 0.1 mg/kg may be administered to reverse the narcotic effect. While the IV route is preferred, IM administration may be used. If there is a chronic maternal (and therefore newborn) exposure to narcotics, administering naloxone may precipitate seizures in the infant, potentially causing death, and therefore is not recommended.

For additional exposures, including topical exposures, contact your local poison control center where experts will be able to guide you regarding a response to a myriad of exposures.

Signs and Symptoms

Toxic Exposure

- Stabilize the infant.
- Manage the ABCs.

Trauma/Birth Injuries

Injuries to the infant that result from mechanical forces (ie, compression, traction) during the birth process are categorized as birth trauma. Significant birth injury accounts for fewer than 2% of neonatal deaths and stillbirths in the United States. It still occurs occasionally and unavoidably, with an average of 6 to 8 injuries per 1,000 live births. Hypoxic injury may coexist with mechanical injury. Most birth traumas are self-limiting and have a favorable outcome.

Causes/risk factors for birth injuries include macrosomia, prolonged labor, precipitous delivery, breech presentation, instrumentation (forceps/vacuum-assisted delivery), extreme prematurity, and oligohydramnios.

Head and Neck Injuries

Most birth injuries self-resolve with time and are not fatal. Birth injuries to the head and neck include the benign head and neck injuries: **vacuum caput**, which is accumulation of fluid at the site of the vacuum extractor that usually resolves within hours, and **caput succedaneum**, a subcutaneous collection of fluid with poorly defined margins in the scalp, resulting from pressure on the infant's head during delivery, that usually resolves within a few days. Serious head and neck injuries include a subgaleal hematoma, which can spread slowly and result in shock. A subgaleal bleed can present as an ill-defined mass in the dependant part of the head (usually the occipital region). It does not follow suture lines. Significant blood loss may occur and the infant may present with shock and acute renal failure if the condition is not promptly identified and treated. **Cephalohematoma** is a subperiosteal collection of blood and may be accompanied by a linear skull fracture. It is most commonly seen in the parietal region and is limited by suture lines. It resolves within a few months with resulting calcification. A skull fracture may present with a slight depression in the skull; however, this may not be obvious secondary to an overlying hematoma.

In addition to the skull findings, these injuries may present with anemia and jaundice. If the hemorrhage progresses, it may cause hypotension and shock. The hematoma may also become infected. Management of head and neck injuries is based on presentation. Blood transfusion may be needed for hemorrhage and anemia. Phototherapy (light therapy to reduce bilirubin) is needed for jaundice based on the bilirubin level. If the neonate is in hypovolemic shock from excessive blood loss, manage the ABCs and treat for shock, focusing on volume resuscitation.

Signs and Symptoms

Head and Neck Injuries

- Skull fracture
- Anemia
- Jaundice
- Hypotension
- Shock
- Acute renal failure

Transport Management

Head and Neck Injuries

- Manage the ABCs.
- Treat for shock, focusing on volume resuscitation.

Nerve Injuries

Nerve injuries often occur because of hyperextension or overstretching during delivery, especially with breech presentation. The nerve injuries incurred during the birth process can range from peripheral nerve branch injury to major spinal cord injury. Most nerve injuries do not require any immediate intervention or admission to a neonatal intensive care unit (NICU). Nerve injuries that need immediate attention and possible transfer to a facility that can provide a higher level of care include recurrent laryngeal nerve injury, which causes paralysis of the vocal cords, unilaterally or bilaterally. This injury presents with respiratory distress, stridor, or desaturations. It may be associated with intracranial bleeding. Visualize the vocal cords by direct laryngoscopy. Intubate in the event of bilateral vocal cord paralysis. Do not feed the neonate to avoid the risk of aspiration during transport. Vaginal breech delivery is the greatest risk factor for spinal cord injury. It frequently presents with a loud "snap" sound at the delivery. It may be associated with epidural hemorrhage, loss of motor function distal to the injury, temperature instability, and loss of bladder and bowel function. Immobilize the head and restrain the neonate. If the nerve injury impairs spontaneous respiration, intubate and provide PPV until the infant can be transferred to an appropriate health care facility.

Signs and Symptoms

Nerve Injuries

- Respiratory distress
- Stridor
- Desaturations
- Paralysis of vocal cords, unilaterally or bilaterally

Spinal Cord Injury

- Epidural hemorrhage
- Loss of motor function distal to the injury
- Temperature instability
- Loss of bladder and bowel function

Transport Management

Nerve Injuries

- Maintain NPO status.
- Provide PPV if spontaneous respiration is impaired.

Bone and Other Injuries

The clavicle is the most common fracture seen in a newborn, and may be accompanied by a pneumothorax. Risk factors include a large infant, shoulder dystocia, and instrumentation during delivery (especially forceps). A fractured clavicle itself does not need any urgent intervention as it heals with limited motion of the arm within 7 to 10 days. However, a rare complication of a fractured clavicle is a pneumothorax. If the neonate presents with tachypnea or increased work of breathing, consider a pneumothorax. See the section on pneumothorax for management.

Transport Management

Bone and Other Injuries

- Monitor for pneumothorax; treat if present.
- Treat for shock.

Fractures of the humerus or femur are uncommon. Manage these by splinting the arm or leg and transferring to a pediatric orthopaedic center.

Intra-abdominal injuries are rare but often present as shock and bluish discoloration of the abdominal wall. Treat the patient for shock, ensure that a hematocrit, type and cross, and if possible, an abdominal ultrasound have been performed at the referring facility before transferring to a hospital with pediatric surgical support.

Finally, soft-tissue injuries include abrasions, lacerations, and ecchymosis; these injuries mostly need routine care.

Abuse/Maltreatment

Shaken baby syndrome is the most common cause of death in cases of child abuse. Shaking of a baby may cause tearing of bridging veins in the brain, a subdural hematoma, or hypoxic injury. Most often, there are no significant findings on physical exam to indicate this condition. It should be suspected in a previously well infant who presents with nonspecific signs such as feeding intolerance, irritability, and vomiting. Severe cases may even present with seizures or apnea secondary to intracranial hemorrhage. Other maltreatment includes blunt trauma to the abdomen, burns, bruising, and skeletal injuries. The management for these is again based on presentation as follows. Obvious extremity fractures should be stabilized, with attention given to maintaining circulation to the affected extremity. Fluid resuscitation may be needed if hemorrhaging has occurred. Respiratory support may be needed if intracranial hemorrhage or trauma to the upper airway has occurred. With severe burns, the affected areas should be protected with sterile material and IV fluids initiated due to increased insensible water loss through the burned skin. The CCTP's goal is to stabilize the neonate and transport safely to a health care facility where further workup and treatment may be done.

Signs and Symptoms

Shaken Baby Syndrome

- Feeding intolerance
- Irritability
- Vomiting
- Seizures
- Apnea secondary to intracranial hemorrhage

Transport Management

Shaken Baby Syndrome and Other Maltreatment

- Stabilize the neonate.

Neurologic Conditions

Seizures

Seizures represent the most distinctive sign of neurologic disease in the neonatal period. Seizures may be identified by direct observation. A seizure is defined as a paroxysmal alteration in neurologic function, ie, behavioral and/or autonomic function. Apnea is also a common manifestation of seizures in a neonate. It is important to distinguish other motor phenomenon (eg, jitteriness, myoclonic activity) that could be confused with seizures. Seizures represent a relative medical emergency because they are usually a sign of a serious underlying abnormality. It is critical to recognize neonatal seizures and to determine their cause because they may interfere with cardiopulmonary function, feeding, affect metabolic function, and if prolonged, may cause brain injury.

Jitteriness is often confused with a seizure **Table 22-12**. Jitteriness is characteristically a disorder of the newborn and is rarely seen at a later age. Jitteriness is most commonly seen with hypoxic-ischemic encephalopathy, hypocalcemia, hypoglycemia, and drug withdrawal. It is important to recognize that newborns exhibit normal motor activity that could be mistaken for seizures. These myoclonic,

dysconjugate eye movements, or sucking movements are often seen when the infant is drowsy or asleep.

Simple classification of seizure types suggests that four essential types can be recognized: subtle, tonic, clonic, and myoclonic. Subtle seizures are characterized by apnea, eye deviation, blinking, sucking, and pedaling movements of the legs. Tonic seizures are characterized by tonic extension of the limbs. Less commonly, with this type of seizure, flexion of the arms and extension of the legs may also occur. Tonic seizures are more common in premature infants, especially in those with intraventricular hemorrhage. Seizures characterized by clonic localized jerking are classified as focal clonic seizures; these types of seizures can occur in both term and premature infants. Lastly, myoclonic seizures are characterized by flexion jerks of the upper or lower extremities, singly or in a series of repetitive jerks.

Causes of neonatal seizures include hypoxic ischemic encephalopathy, intracranial hemorrhage, intracranial infections (meningitis), development defects, hypoglycemia, hypocalcemia, and other metabolic disturbances, frequently the result of inborn errors of metabolism. You may observe a quiet, often hypotonic infant. The infant may be lethargic and/or apneic. Hypoglycemia must be recognized quickly and treated promptly, within a few minutes, to minimize the risk of brain damage. $D_{10}W$ may be given as an IV bolus (2 mL/kg) if the blood glucose level is less than 40 mg/dL. A blood glucose measurement and administration of dextrose if hypoglycemia is present may be lifesaving. The blood glucose level should be rechecked every 30 minutes after treatment for hypoglycemia, until the blood glucose level is documented to be stable. An IV bolus of dextrose should be followed by an infusion of $D_{10}W$ to maintain normoglycemia during transport. The neonate's vital signs and oxygen saturation should be monitored throughout transport. Apnea, a common symptom of neonatal seizures, may require intubation and ventilatory support during transport. Phenobarbital (Luminal) and phenytoin (Dilantin) are the two commonly used anticonvulsants in neonates. Both drugs may interfere with respiratory and cardiac function and should only be used in consultation with the supervising physician. Because seizures may be a sign of meningitis, the referring facility should have performed a septic workup and begun antibiotics before the transfer.

TABLE 22-12 Jitteriness vs Seizures

Characteristics	Jitteriness	Seizures
Ocular phenomenon (deviation or fixation of the eyes)	Not seen	Commonly associated
Stimulus sensitive (may be triggered by a stimulus)	Yes	No
Dominant movement	Tremor	Clonic jerking
Application of gentle pressure to limb	Stops jitteriness	Does not stop seizures
Autonomic phenomenon	Not associated	Common association

Signs and Symptoms

Seizures

- Apnea
- Eye deviation
- Blinking
- Sucking

- Pedaling movements of the legs
- Tonic extension of the limbs
- Flexion of the arms and extension of the legs
- Clonic localized jerking
- Flexion jerks of the upper or lower extremities

Differential Diagnosis

Seizures

- Jitteriness
- Hypoxic ischemic encephalopathy
- Intracranial hemorrhage
- Intracranial infections (meningitis)
- Development defects
- Hypoglycemia
- Hypocalcemia
- Inborn errors of metabolism

Transport Management

Seizures

- Monitor vital signs and oxygen saturations.
- Intubate and provide ventilatory support if necessary to treat apnea.

Hypoxic Ischemic Encephalopathy

Hypoxic ischemic encephalopathy (HIE) is the neuronal injury caused by oxygen deprivation (hypoxia) or decreased perfusion (ischemia) to the brain. Redistribution of blood occurs during prolonged asphyxia, with more of the cardiac output being delivered to the vital organs (eg, brain, heart, adrenal glands) at the expense of less vital organs (eg, lungs, gut, kidneys, skin). Multiple organ dysfunction is common after HIE and may influence the eventual outcome for the infant. HIE is the single most common cause of seizures in both term and preterm infants. The time of onset for organ dysfunction from HIE, hypoglycemia, and other metabolic disturbances is from birth to 3 days. As compared to HIE, the time of onset for all the other listed causes of seizures can be from birth to longer than 3 days. Infants with HIE often present with decreased tone, poor perfusion, and in more severe situations, hypotension and recurrent apnea.

Management of neonatal HIE includes maintaining adequate ventilation, perfusion, and blood pressure; maintaining adequate blood glucose levels; carefully monitoring fluid intake and output; and adjusting treatment based on renal output, oxygen saturation, and ventilation status. The infant's temperature should be maintained in the low normal range (97.5°F [36.4°C]) to avoid hyperthermia, which is associated with a worse outcome in HIE. If a newborn with HIE arrives at the tertiary care center within 6 hours of birth, more aggressive therapeutic hypothermia may be offered as a treatment modality under controlled conditions. It is therefore critical to expedite transport of these infants to a

center that can provide appropriate therapy.

Transport Management

Hypoxic Ischemic Encephalopathy (HIE)

- Maintain adequate ventilation, perfusion, and blood pressure.
- Maintain adequate blood glucose.
- Monitor fluid intake and output and adjust based on renal output.
- Monitor saturations and maintain oxygen levels in the normal range.
- Maintain the infant's temperature in the low normal range.

Lethargy

In infants, lethargy (a decreased level of consciousness during which you are unable to arouse the infant) can be caused by many conditions, but it almost always implies a serious, frequently life-threatening underlying condition. Some common causes of lethargy include sepsis, severe hypoxia, severe hypoglycemia, intracranial hemorrhage, and hypoxic ischemic encephalopathy.

Management includes attention to the ABCs, obtaining IV access, ensuring normoglycemia, providing oxygen as needed, initiating antibiotics in conjunction with the supervising physician, and expediting transfer to a tertiary center skilled in dealing with critically ill neonates.

Differential Diagnosis

Lethargy

- Sepsis
- Severe hypoxia
- Severe hypoglycemia
- Intracranial hemorrhage
- Hypoxic ischemic encephalopathy

Transport Management

Lethargy

- Monitor the ABCs.
- Ensure normoglycemia.
- Provide oxygen as needed.

Metabolic Conditions

Hypoglycemia

Hypoglycemia, or a blood glucose level of less than 40 mg/dL, is a medical emergency; in severe cases, it can lead to brain damage and even death. Hypoglycemia is more common in infants who are small or

large for gestational age, infants of diabetic mothers, and infants who are stressed. Signs of hypoglycemia include decreased activity, jitteriness, and seizures.

If you suspect hypoglycemia, a heel stick should be performed immediately to determine the blood glucose level. If hypoglycemia is present, IV dextrose (2 mL/kg $D_{10}W$) should be given, followed by an infusion of glucose. First administer 60 mL/kg/d $D_{10}W$ to maintain the neonate's blood glucose level as you contact the regional perinatal center for guidance on the rate and type of IV solution; this can vary based on gestational and chronological age. An inborn error of metabolism is another metabolic condition that is initially treated with a glucose infusion. Fortunately, these are relatively rare, but if a neonate is lethargic and experiences tachypnea (suggesting a metabolic acidosis), an infusion of glucose should be started, even if the neonate is normoglycemic. This infusion will help decrease the buildup of toxic byproducts the body produced when the neonate was unable to metabolize nutrients normally.

Signs and Symptoms

Hypoglycemia

- Decreased activity
- Jitteriness
- Seizures

Transport Management

Hypoglycemia

- Maintain the infant's blood glucose level.

Hypocalcemia

Hypocalcemia is most commonly seen in low birth weight infants and can occur after significant stress, as well as in infants of diabetic mothers. Significant hypocalcemia can lead to cardiac arrhythmias, seizures, and tetany. In general, calcium infusion is not indicated unless hypocalcemia is documented. There are two common timeframes when hypocalcemia occurs in the neonate. The first is at age 2 to 3 days. The second (late-onset hypocalcemia) is unusual in the United States but may be seen in infants who consume cow's milk or synthetic formulas that are high in phosphorus. In an infant with documented hypocalcemia and who is younger than 24 hours, administer an IV infusion of 2 to 3 mEq/kg/d of calcium in $D_{10}W$ during transport. If an infant has documented hypocalcemia, is older than 24 hours, and has normal renal function, administer 10% dextrose in 0.25 normal saline with 10 mEq/kg of potassium chloride in 500 mL during transport.

Signs and Symptoms

Hypocalcemia

- Cardiac arrhythmias
- Seizures

- Tetany

Transport Management

Hypocalcemia

- Administer an IV infusion of 2 to 3 mEq/kg/d of calcium in $D_{10}W$ during transport to an infant with documented hypocalcemia and who is younger than 24 hours.
- Administer 10% dextrose in 0.25 normal saline with 10 mEq/kg of potassium chloride to an infant who has documented hypocalcemia, is older than 24 hours, and has normal renal function.

Inborn Errors of Metabolism

An **inborn error of metabolism** is a class of genetic diseases mostly due to defects of single genes that code for enzymes that facilitate conversion of various substances into others leading to accumulation of substances which are toxic or interfere with normal function. There are many different inborn errors of metabolism, and they are relatively rare, so a complete review would be outside the scope of this chapter. Common presenting features include lethargy, acidosis, poor perfusion, and hypoglycemia. If an inborn error of metabolism is suspected, enteral feeding should be stopped and an IV glucose infusion begun before transfer by the critical care transport team.

Signs and Symptoms

Inborn Errors of Metabolism

- Lethargy
- Acidosis
- Poor perfusion
- Hypoglycemia

Congenital Adrenal Hyperplasia

- Dehydration
- Shock

Transport Management

Inborn Errors of Metabolism

- If suspected, stop enteral feeding and begin an IV glucose infusion before transfer.

Congenital Adrenal Hyperplasia

- Normalize electrolytes and fluid balance.
- Consider initiating hydrocortisone replacement; consult with supervising physician.

Congenital adrenal hyperplasia results from a defect in the synthesis of cortisol. An infant may have virilizing features and problems with salt loss, depending on which enzyme is deficient. If the infant experiences salt loss, he or she can present with dehydration and shock. Acute treatment includes normalizing electrolytes and fluid balance, and initiating hydrocortisone replacement after consultation with the supervising physician.

The Transfer Process for a Neonate

In the well-organized regional referral system, transport of a high-risk neonate proceeds through the following steps:

1. A physician at the referring hospital initiates a request for transport. A physician in the regional control center decides which facility can accommodate the patient and gives the referring physician advice on management of the neonate until the transport team arrives.
2. A mode of transportation is chosen—ground transportation, helicopter, or fixed-wing aircraft, depending on the patient's status, distance, availability of services, and weather conditions.
3. The transport team is mobilized, including communication with the CCTP regarding any relevant information known about the neonate to be transferred, and equipment is assembled. Optimally the team consists of a health care provider with special training in caring for critically ill neonates, a respiratory therapist with similar special training, and a paramedic who has spent a period of apprenticeship or received additional training in transporting an ill neonate. For particularly critical patients, a physician may also accompany the team. The equipment used by the CCTP is highly specialized, requiring appropriately designed ventilation and oxygenation units and an incubator meeting stringent criteria.
4. On arriving at the referring hospital, the transport team helps with further stabilization of the infant if needed, before embarking on transport. Conditions such as hypoxemia, acidosis, hypoglycemia, pneumothorax, shock, and hypovolemia must be treated before leaving the referring hospital. If the infant is suspected of having an infectious disease, appropriate cultures should be obtained prior to transport and early antibiotic therapy initiated. If a contagious disease is suspected, transport personnel should take adequate safety precautions.
5. While stabilizing the infant, the team collects information and materials, including a copy of the mother's and infant's medical records and any radiographs, labs, and medications for the infant. Often transport teams operate on the basis of implied consent, meaning that transport is considered a part of treatment of the life-threatening emergency. Some areas require signed consent before transport is initiated. Awareness of local and state guidelines is important.

Once the infant's condition is stabilized as much as possible at the referring hospital, transport the patient to the nearest facility that can provide the appropriate level of care. Ideally, someone will contact this facility to discuss the situation and obtain advice regarding care and disposition throughout the process. Complications can arise en route, such as a dislodged breathing tube, or development of a pneumothorax. It is critically important to keep vigilant to recognize changes in the status of the infant, such as desaturation or change in color. Quick intervention will help ensure an optimal outcome.

Using an Incubator

An incubator is a box with clear walls, a heat source, and portal access in which a neonate can be placed to maintain normothermia and minimize noise stress. An incubator is indicated for transport of infants

who weigh less than 10 lb or are younger than 30 days. When preparing to use an incubator, also ensure that the oxygen tank attached to the incubator is full, and that there are blankets and a bulb syringe inside the incubator.

Skill Drill 22-4 shows the steps for using an incubator, which are described below:

1. Plug in the AC power cord **Step 1**.
2. Set the main AC power switch **Step 2**. The charging indicator will be a constant light if internal batteries need charging. The light will flicker or go off when fully charged. Battery life is approximately 2 hours.
3. Depress the "controller" switch **Step 3**. The power failure alarm and AC/DC power mode should light up. The high temperature sensor, heater temperature, and low DC alarm should flash. A short beep will be heard at the end of the test. The set point indicator should light and the centigrade reading should indicate 91.4°F (33°C) ± 0.1 for 15 seconds.
4. Check and adjust the set point temperature to 100.4°F (38°C) **Step 4**. The maximum temperature is 104°F (40°C). It takes approximately 30 minutes to warm up.
5. Do not block or cover the end portions of the incubator.
6. Check the oxygen and air tanks **Step 5**. All tanks should read 2,000 psi.

Family Communication and Support

Throughout the process of transporting an ill neonate, ongoing communication with the family regarding what is being done for the infant and what care is planned will help allay fears. Do not be specific about the infant's chances of survival. Many factors play into morbidity and mortality and you do not want to be misleading. If family members have questions you cannot answer, be straightforward. Tell them that you do not have a definite answer, but you will help put them in touch with the people who do (most likely individuals at the center to which the infant is being transferred). Often, just explaining what you are doing and giving the family a realistic time frame in which they should be able to speak to someone at the receiving institution is very helpful. Families usually appreciate knowing the name of the receiving physician, if this is known at the time of transfer.

Skill Drill 22-4

Using an Incubator

1 Plug in the AC power cord.

2 Set the main AC power switch. The charging indicator will be a constant light if internal batteries need charging. The light will flicker or go off when fully charged.

3 Depress the "controller" switch. Check that the power failure alarm and AC/DC power mode light up; and that the high temperature sensor, heater temperature, and low DC alarm flash. A short beep will be heard at the end of the test. The set point indicator should light and the centigrade reading should indicate 91.4°F (33°C) ± 0.1 for 15 seconds.

4 Check and adjust the set point tempera-I ture to 100.4°F (38°C). Do not block or cover the end portions of the incubator.

5 Check the oxygen and air tanks. All tanks should read 2,000 psi.

Flight Considerations

Air transport, either fixed wing or helicopter, presents a new set of challenges and should be managed appropriately. Some of these are listed here:

- **Airway:** It is critical to establish a secure airway before takeoff, because it is extremely difficult to intubate and assess breath sounds during flight. If this step is necessary while in flight, special attention to signs of adequate ventilation and endotracheal tube position need to be considered, such as condensation on the ET tube, chest rise, and change in color of the $ETCO_2$ indicator.
- **Circulation:** It can also be difficult to hear a heartbeat while in the air, so alternative methods of assessment, such as palpating pulsation on a freshly clamped umbilical cord, or feeling the cardiac movement at the point of maximal intensity, can be helpful.
- **Oxygenation:** There is decreased partial pressure of oxygen at higher altitudes; additional oxygen may be needed to maintain oxygen saturation.
- **Temperature regulation:** Extra care should be taken to avoid hypothermia.

Finally, minimize the neonate's exposure to fuel vapor and noise.

Helicopter transport may be used for medium distance transfers of up to 150 miles. This mode may not be appropriate for transporting the neonate because of limited space and the relative inability to carry the necessary equipment such as an incubator with additional personnel. There is restricted patient access during flight and patient evaluation may be compromised because of noise and vibrations. The cabin is not pressurized, which may lead to further hypoxic and hypothermic stress.

Fixed-wing (airplane) transport is generally reserved for long distances (> 150 miles). As compared to helicopter transport, the space allocated for patient care is generally adequate. Transport will involve ambulance transportation to the airport, subsequent air transport, and further ambulance transportation to the hospital. It is important to not only arrange the helicopter or airplane, but also the ambulance to and from the airports and the respective hospitals.

Summary

Neonates have a unique anatomy, physiology, and pathology, making transfer of such patients challenging but rewarding. The CCTP must be familiar with the special needs of this age group to provide optimal care during transfer to reduce morbidity and improve survival.

Case Study

YOU AND YOUR NEW PARTNER HAVE STOPPED for a slice of pizza after a routine transport. While ordering, you receive a call to respond to a local emergency room for a 3-week-old with a fever. You and your partner understand that young children do not fare well in febrile states. The pizza will have to wait.

On arrival at the emergency room, you are met by the charge nurse and attending physician. They report to you that a 20-day-old African-American female was brought in by the mother, who said that the infant was sleepy, refusing to eat, and felt warm to the touch. Triage noted that the patient was not responding to external stimuli and presented with a weak cry and delayed capillary refill. The patient was delivered at the same hospital and records showed a normal, cephalic delivery with no complications. In addition, the mother had received prenatal care and only had a history of asthma.

Physical exam of the patient reveals diffuse congestion with mild substernal and intercostal

retractions, peripheral cyanosis and mottling, a capillary refill time of 4 seconds, and a rectal temperature of 102°F (38.8°C). The child is nonresponsive to verbal stimulation and grimaces when you perform tactile stimulation. Vital signs show a pulse of 180 beats/min, with atrial tachycardia on the monitor, a blood pressure of 100/40 mm Hg, and an oxygen saturation of 90%. An IV of normal saline 9% has been established in the left antecubital vein with a 24-gauge catheter, and it appears patent. You estimate the infant's weight to be 8 kg. A CBC was drawn with a WBC count of 26,000/mm^3. The serum blood glucose level was 80 mg/dL. All other blood values were normal. No arterial blood gas analysis, chest radiograph, or lumbar puncture has been performed. The attending physician advises you that he has been in contact with the neonatal intensive care unit (NICU) at University Hospital and he has the receiving physician's information.

1. What is your presumptive diagnosis?
2. What are some potential causes of this patient's altered mental status?
3. What are your primary concerns in managing this patient?

You note that the patient is receiving oxygen by a nonrebreathing mask at a flow rate of 8 L/min. You ask your partner to switch to bag-mask ventilation at a rate of 20 breaths/min as you prepare to contact the receiving facility.

Your partner advises you that the bag-mask ventilation is compliant and there is good chest rise. The patient's oxygen saturation has increased to 95% with other vital signs remaining unchanged. You contact University Hospital and speak with the receiving physician. After confirming the bed assignment, you confer your findings. It is agreed that bag-mask ventilation, while effective in the emergency department, may become a challenge to maintain during the 45-minute transport. You receive an order to perform rapid sequence intubation. Your protocol for intubation includes premedication with atropine, fentanyl, and succinylcholine. The patient is intubated. You are unable to obtain cultures, but due to the high suspicion of serious infection, the referring physician instructs you to administer ampicillin and gentamicin at weight-appropriate doses per the *Harriet Lane Handbook*, by RE Rau, JW Custer, and CK Lee, a pediatric reference tool.

After administration of 160 mL of normal saline, you note that the infant's blood pressure is 110/70 mm Hg, the pulse is 170 beats/min, and capillary refill is 2 seconds. During transport, your patient's oxygen saturation increases to 98%, the $_{ETCO2}$ is 43 mm Hg, and her color has improved. However, lung sounds are still diffusely congested. You arrive at the NICU and transfer your patient.

Analysis

Although you are somewhat convinced the infant has sepsis, your concern is about the patient's impending respiratory failure due to acute respiratory distress syndrome. In addition, the $\dot{V}/\dot{Q}$ mismatch is causing acidosis by failing to allow release of carbon dioxide.

In addition to hypoxia, you must also be aware of the other potential causes of neonatal altered mental status, namely hypoglycemia and seizures. Once the airway is secured, CCTPs must assess the infant's blood glucose level.

Overall, the most critical aspect of managing this difficult patient is the ability to secure the airway, control oxygenation, and stabilize hemodynamics.

Two weeks later, you and your partner follow up on this case during a QA meeting. On admission, the patient received a spinal tap, which was negative, but a blood culture revealed *Streptococcus pneumoniae*, a very common bacterium that causes illnesses ranging from ear infections to meningitis. The patient received 7 days of IV ampicillin and gentamicin and successfully fought off the infection,

remaining on a ventilator for 3 days and being discharged after 7 days. Credit was given to the community hospital for prompt recognition that this patient was in need of more specialized care and to the transport unit that made the decision to aggressively manage the patient's airway.

Prep Kit

Ready for Review

- A newborn refers to an infant within the first few months of life; a neonate refers to an infant within the first 28 days of life; term newborns are delivered between 37 and 42 weeks of gestation.
- Because short-term and long-term outcomes in infants have been linked to initial stabilization efforts, CCTPs must be knowledgeable about problems that may occur with neonates and how to treat them, they must have the appropriate equipment available to treat neonates, and they must carefully consider the neonate's transport plan.
- Newborns differ anatomically and physiologically from young children. CCTPs must be aware of those differences.
- Neonates become cold easily due to a variety of factors ranging from a high body surface area to body weight ratio to increased evaporative heat loss through skin. To avoid hypothermia, use prewarmed blankets and equipment, including a prewarmed transport incubator, place a cap on the infant's head, and minimize exposure to cold. While avoiding hypothermia, be careful to also avoid environmentally induced hyperthermia.
- The lungs of neonates are small relative to their body size, with little respiratory reserve. Their heads are large in relation to their bodies, resulting in neck flexion and obstruction in the supine position. Placing a rolled towel under a neonate's shoulders helps open up the airway.
- Neonates are obligate nose breathers and may require an oral airway during transport if they present with obstructive apnea. When positive-pressure ventilation in needed, attempts should be made to maintain physiologic tidal volume breaths.
- Neonates have a high oxygen requirement; they do not tolerate interruptions in oxygen delivery for any length of time, and they become hypoxic and bradycardic quickly.
- The neonate's heart rate is an important determinant of cardiac output. Neonates respond to careful volume loading with an increase in cardiac output, but they do not tolerate fluid overload. Changes in cardiovascular circulation that take place after the umbilical cord is clamped at birth include a sudden rise in the systemic vascular resistance, increased pulmonary blood flow, and physiologic closure of the fetal shunts. With extrauterine breathing and expansion of the lungs, tissue oxygenation increases, pulmonary vascular resistance decreases, and pulmonary blood flow increases.
- Pulmonary vascular resistance may increase in the neonate, causing persistent pulmonary hypertension. During transport, give special attention to maintaining normothermia, optimal oxygenation, fluid status, and systemic blood pressure, and minimize acidosis and noise to reduce the risk of the neonate developing severe persistent pulmonary hypertension.
- Patent ductus arteriosus (PDA) is seen in 50% of extremely premature infants. Minimizing fluid overload during transport can decrease the risk of persistent symptomatic PDA.
- Nephrogenesis is complete at 36 weeks' gestation. The glomerular filtration rate at term is low; creatinine levels at birth reflect the mother's level and falls to reflect the infant's renal function by 1 week of age. Infants tolerate fluid restriction poorly and become dehydrated quickly.

- Total body water represents 85% of body weight in premature infants, 75% of body weight in term infants, and 60% of body weight in adults. Contraction of the extracellular compartment and weight loss in the first few days after birth are normal. Fluids should be restricted until after the postnatal weight loss occurs.
- Liberal fluid regimens in the first few days of life have been associated with worse outcomes in premature infants. $D_{10}W$ is usually used for IV fluid maintenance during the new-born's first 24 hours. After 24 hours and the establishment of adequate urine output, 10% dextrose in 0.25 normal **I** saline is used. Neonates are considered to have pressure-passive cerebral circulation. An acceptable mean arterial pressure generally equates to the gestational age of the newborn. It is important to minimize stress and use appropriate sedation for procedures to minimize blood pressure peaks that can lead to intraventricular hemorrhage.
- Neonates, including premature neonates, show well-developed responses to painful stimuli. Use appropriate analgesic measures to treat neonates for pain.
- Neonates are prone to soft-tissue and bony injuries, such as rib fractures, due to incomplete ossification of bones and fragile tissues. The CCTP should be aware of this when administering CPR.
- The risks for complications in neonates increase as birth weight and gestational age decrease. Additional complications such as maternal infection, diabetes, premature labor, and the presence of meconium also increase risk. The CCTP should be familiar with antepartum and intrapartum risk factors.
- The APGAR score is a numeric expression of an infant's condition after birth. It measures the infant's appearance, pulse, grimace, activity, and respiration. The score is affected by the infant's physical maturity, maternal medication intake or narcotic use, and neuromuscular or cardiorespiratory conditions.
- The first steps in stabilizing a neonate with acute airway obstruction, ineffective respiration, or insufficient cardiovascular circulation are airway management and ventilation. To optimize oxygenation and ventilation, thoroughly suction the neonate's airway. Free-flow supplemental oxygen, ventilations, intubation, chest compressions, or volume expansion may be necessary. The CCTP should communicate information such as relevant history, vital signs, and physical exam to the accepting facility as soon as possible.
- The ABCs of neonatal resuscitation are the same as those applied to adults—airway, breathing, circulation. The most important and effective action in neonatal resuscitation is getting oxygen into the baby's lungs. Maintenance of normal body temperature is also important.
- Familiarity with the length-based, color-coded resuscitation tape allows the CCTP to estimate body weight based on the child's crown-heel length. Most neonatal drug doses are based on body weight, and pediatric supplies are organized according to the color-coded, length-based classification scheme.
- Free-flow oxygen, oral airways, bag-mask ventilation, or intubation may be required to assist ventilation in a neonate. The CCTP should be familiar with the equipment and procedures associated with each of these interventions.
- Chest compressions and resuscitation may be necessary if the infant's heart rate remains below 60 beats/min despite other interventions. Chest compressions can be performed using the thumb technique or the two-finger technique with one or two rescuers. Medications are rarely used to resuscitate neonates, but the CCTP should be familiar with medications that may be used, and should be able to estimate the infant's weight because medication doses for neonates are based on weight.
- Fluid resuscitation may be required if the infant is hypovolemic. A low umbilical vein is used in a

newborn; a peripheral IV or intraosseous line is placed in an infant more than a few days old.

- Neonatal transport incubators are typically fitted with a ventilator that provides synchronized intermittent mandatory ventilation. In some areas high-frequency ventilators are available, as well as the ability to initiate and transport infants on a nitric oxide or extracorporeal membrane oxygenation circuit. CCTPs should be familiar with the technology available in their area.
- There are two types of apnea: primary and secondary. Primary apnea responds to stimulation and oxygen supply; secondary apnea responds to ventilation and supplemental oxygen.
- If the infant inhales meconium-stained amniotic fluid *in utero* or at delivery, the airways can become plugged and hypoxia can follow. Meconium aspiration syndrome (MAS) is the respiratory distress caused by meconium aspiration.
- Tracheal intubation and aspiration with a meconium aspirator are performed before other interventions when meconium is present in the amniotic fluid of a depressed newborn at birth. MAS may occur despite tracheal suctioning. During transport, the CCTP should monitor the infant for complications of MAS and should be prepared to perform a needle thoracentesis for pneumothorax decompression if necessary.
- Neonates are at increased risk for pneumonia. Premature infants, especially those less than 32 weeks' gestation, are at high risk for development of respiratory distress syndrome (RDS). Male infants and infants of diabetic mothers are at increased risk of RDS. A sepsis workup should be performed prior to transport because pneumonia is indistinguishable from RDS in a newborn.
- Exogenous surfactant may be administered to the infant with RDS in a controlled clinical setting by providers experienced in its delivery. During transport following administration of exogenous surfactant, the CCTP needs to monitor the infant for improving compliance and the potential need to decrease respiratory support.
- In mild RDS, continuous positive airway pressure can be helpful to minimize alveolar collapse during transport.
- Simple pneumothorax causes lung collapse; tension pneumothorax is a life-threatening condition. Management of pneumothorax includes transillumination, thoracentesis, and possible intubation. If a symptomatic pneumothorax is suspected, a needle thoracentesis can be life saving.
- General assessment of the neonate includes assessment of appearance, work of breathing, or abnormal breath sounds. Evaluation of circulation includes abnormal skin color or bleeding. Determine if the condition is life threatening.
- Primary assessment includes airway, breathing, and circulation. Assess the airway to determine if it is patent. Assess cardiovascular and end organ function. Respiratory problems are a major cause of cardiac arrest in newborns and infants. Prompt recognition and effective management are fundamental to pediatric life support.
- Cyanosis is caused by decreased arterial oxygenation. Cyanosis has cardiac and noncardiac causes. Acrocyanosis is a benign, common condition.
- Cyanotic congenital heart disease (CHD) is caused by developmental anomalies of the heart or major vessels leading to abnormal blood flow. The CCTP may perform tests ranging from a CBC to a hyperoxia test to monitor the infant's condition and determine the appropriate course of action.
- Sinus tachycardia is a heart rate faster than the normal for the child's age. Common pathologic causes for tachycardia include tissue hypoxia, hypovolemia, fever, metabolic stress, pain, anxiety, drugs, and anemia.

- Supraventricular tachycardia is an abnormally fast cardiac rhythm. Emergency interventions such as vagal maneuvers, cardioversion, and pharmacologic interventions should be performed under appropriate supervision.
- Bradycardia in a neonate is usually a result of tissue hypoxia and resolves when tissue oxygenation is restored. After delivery, cardiac pacing may need to be initiated, but is usually nonurgent.
- Cardiac arrest is uncommon in children. It is more often caused by progression of respiratory distress, respiratory failure, or shock typically associated with hypoxemia and acidosis, than by cardiac arrhythmias.
- Persistent pulmonary hypertension (PPHN) is persistence of elevated pressures in pulmonary vasculature after birth. Meconium aspiration is the most common cause of PPHN.
- Care for an infant with PPHN includes optimizing oxygenation, minimizing stress, maintaining a normal blood pressure, and avoiding or correcting acidosis. Critically ill neonates often have a mix of metabolic and respiratory acidosis. Respiratory acidosis often responds to assisted ventilation. Management of metabolic acidosis includes managing ABCs, providing adequate oxygenation, ventilation, hydration, and treating the underlying cause. Treatment may include administering bicarbonate or IV dextrose.
- Shock is a serious condition that can affect all organ systems If emergent access is necessary to provide fluids, an umbilical vein line can be placed in a newborn, or an intraosseous line can be placed in an older infant (older than 1 week).
- Anemia is a hematocrit value of less than 38% in preterm neonates and less than 42% in term neonates. A neonate with anemia may be asymptomatic or present with signs and symptoms, including, but not limited to, tachycardia, jaundice, heart failure, and shock.
- Management of anemia includes managing the ABCs and obtaining IV access (optimally two sites). Treatment may include volume resuscitation with normal saline bolus, followed by blood if the infant is in shock, or packed red blood cells and fresh-frozen plasma if coagulopathy is suspected.
- Any part of the gastrointestinal tract may have congenital anomalies. Bilious vomiting is considered obstruction until proven otherwise; passage of meconium does not rule out an obstructed bowel. An obstructed bowel can lead to perforation and peritonitis.
- Gastroschisis is a centrally located, full-thickness abdominal wall defect. Omphalocele is a herniation of abdominal contents into the umbilical cord. Gastroschisis is usually an isolated defect; omphalocele is often associated with congenital defects, CHD, and other anomalies.
- If an infant with gastroschisis or omphalocele is born at a remote facility, transport must be expedited to a facility with a neonatal intensive care unit and pediatric surgery. Precautions must be taken to cover and protect the exposed abdominal contents and to prevent infection.
- The presence of bile in vomit must be treated as a surgical emergency until obstruction, especially malrotation, has been ruled out. The CCTP should know the signs and symptoms of esophageal atresia, proximal intestinal obstruction, distal intestinal obstruction, imperforate anus, and Hirschsprung's disease. During transport of neonates with any of these conditions, thermoregulation of the neonate should be maintained and vital signs should be monitored closely.
- Obstructed bowel, necrotizing enterocolitis, and iatrogenic perforation can cause perforation of the bowel. Infants with acute intestinal perforation are at high risk of shock and severe metabolic acidosis secondary to inflammation and vascular leak. Hematemesis can occur with several conditions, including volvulus, which can be fatal. Intestinal volvulus is a surgical emergency.

- Intussusception is the most common cause of intestinal obstruction in children between 3 months and 6 years of age. Intussusception may be reduced by enema, but frequently needs open reduction.
- Diarrhea in a neonate is unusual. Maintain fluids during transport, monitor vital signs, and maintain normothermia.
- Neonates are prone to becoming compromised by serious infectious diseases because their immune systems are not fully developed. The infant's condition can deteriorate rapidly; monitor vital signs and maintain normothermia while transferring to a center that can manage a critically ill neonate.
- Hyperthermia is usually caused by overbundling or exposure to excessive heat. It is also seen with herpes simplex infection.
- Hypothermia is more common during the winter months. It can lead to thermal shock, disseminated intravascular coagulopathy, and death in serious cases. Once stabilized, the infant should be placed in a prewarmed incubator or covered with warm blankets for transport.
- The most important principle of managing toxic exposure/ingestion is stabilization of the neonate. Be sure to obtain a history from the sending facility. Contact the local poison control center for expert guidance in managing exposure to toxins.
- Most birth traumas are self-limiting and have a favorable outcome. Birth injuries can be caused by prolonged labor, breech presentation, and extreme prematurity among other causes and risk factors.
- Head and neck injuries include vacuum caput and caput succedaneum, which are benign, and serious head injuries such as subgaleal hematoma and cephalohematoma. Nerve injuries often occur because of hyperextension or overstretching during delivery, especially with breech presentation. Recurrent laryngeal nerve injury requires immediate attention and transfer to a facility that can provide a high level of care.
- Vaginal breech delivery is the greatest risk factor for spinal cord injury.
- The clavicle is the most common fracture seen in newborns. A fractured clavicle will heal itself within 7 to 10 days with limited motion of the arm. Pneumothorax is a rare complication of fractured clavicle. Soft-tissue injuries mostly need routine care.
- Shaken baby syndrome is the most common cause of death in cases of child abuse. Other maltreatment includes blunt trauma to the abdomen, burns, bruising, and skeletal injuries. It is important to be able to distinguish between seizures and jitteriness in the neonate. Seizures are usually a sign of a serious underlying abnormality; jitteriness is characteristically a disorder of the newborn and is rarely seen at a later age.
- Hypoxic ischemic encephalopathy (HIE), caused by oxygen deprivation or decreased perfusion to the brain, is the most common cause of seizures in term and preterm infants. Multiple organ dysfunction is common after HIE and may affect the outcome for the infant. Hyperthermia has been associated with a worse outcome for the infant with HIE.
- Lethargy is almost always a sign of a serious, frequently life-threatening underlying condition. Hypoglycemia is a medical emergency that can lead to brain damage and even death in severe cases. Signs of hypoglycemia include decreased activity, jitteriness, and seizures.
- Hypocalcemia is most commonly seen in low birth weight infants and can occur after significant stress, as well as in infants of diabetic mothers. Significant hypocalcemia can lead to cardiac arrhythmias, seizures, and tetany.
- An infant with congenital adrenal hyperplasia may present with dehydration and shock. Acute treatment includes normalizing electrolytes and fluid balance, and hydrocortisone replacement after consultation

with the supervising physician.

- Transport of a high-risk neonate proceeds through a series of well-organized, established steps. Awareness of local and state guidelines is important, as is communication among the members of the transport team, the referring facility, and the receiving facility. Communicating with family members regarding what is being done for the infant and a time frame in which they can expect to speak to someone at the receiving facility is helpful in allaying their fears.
- The CCTP must be prepared to manage the challenges presented by different modes of air transport. Airway, circulation, oxygenation, and temperature regulation are among those challenges.

Vital Vocabulary

acrocyanosis A decrease in the amount of oxygen delivered to the extremities. The hands and feet turn blue because of the lack of oxygen.

APGAR score A scoring system for assessing the status of a newborn that assigns a number value to each of five areas of assessment (the total possible score is 0 to 10).

apnea Respiratory pause greater than or equal to 20 seconds.

atresia Complete absence of lumen.

caput succedaneum In the newborn, a subcutaneous collection of fluid with poorly defined margins within the scalp, resulting from pressure on the head during delivery.

central cyanosis Bluish coloration of the skin due to the presence of deoxygenated hemoglobin in blood vessels near the skin surface.

cephalohematoma A subperiosteal collection of blood; may be accompanied by a linear skull fracture.

choanal atresia A congenital narrowing or blockage of the nasal airway by membranous or bony tissue.

congenital adrenal hyperplasia A disease group in which the adrenal glands do not function properly, and as a result do not produce a sufficient amount of the hormone cortisol.

Coombs' test A test used to identify antibodies that may bind to the red blood cells and cause hemolysis.

diaphragmatic hernia Passage of loops of bowel with or without other abdominal organs, through the diaphragm muscle; occurs as the bowel from the abdomen "herniates" upward through the diaphragm into the chest (thoracic) cavity.

distal intestinal obstruction Partial or complete obstruction of the distal small bowel (ileum) or large intestine.

esophageal atresia A congenital defect in which the esophagus ends in a blind pouch, often associated with other anomalies such as tracheoesophageal fistula.

gastroschisis A centrally located, full-thickness abdominal wall defect.

gestation Period of time from conception to birth. For humans the full period is normally 9 months (or 40 weeks).

Hirschsprung's disease A congenital lack of ganglion nerve cells in a portion of the distal intestines leading to poor intestinal peristalsis, constipation, and, if not diagnosed and treated, potentially death.

hypotonia Low or poor (floppy) muscle tone.

hypoxic ischemic encephalopathy (HIE) Damage to cells in the central nervous system (the brain and spinal cord) from inadequate oxygen.

imperforate anus A congenital condition where the anal opening is either absent or displaced.

inborn error of metabolism A class of genetic diseases mostly due to defects of single genes that code for enzymes that facilitate conversion of various substances into others leading to accumulation of substances which are toxic or interfere with normal function.

incubator An enclosed, clear plastic, heated bed that keeps the infant warm.

intussusception An event where one part of the intestine folds into another part of the intestines, leading to a blockage.

malrotation A congenital anomaly of rotation of the midgut, in which the small bowel is found predominantly on the right side of the abdomen.

meconium A dark green fecal material that accumulates in the fetal intestines and is discharged around the time of birth.

neonate An infant during the first 28 days after birth.

newborn An infant within the first few hours after birth.

oligohydramnios Decreased volume of amniotic fluid during a pregnancy; a risk factor associated with abnormalities of the urinary tract, postmaturity (birth after a prolonged pregnancy), and intrauterine growth retardation.

omphalocele Herniation of abdominal contents into the base of the umbilical cord.

oxygen hood A tent placed over the head of an infant for the purpose of delivering supplemental oxygen.

patent ductus arteriosus A situation in which the ductus arteriosus, which assists in fetal circulation, does not transition as it should after birth to become the ligamentum arteriosum; the result is that the connection between the pulmonary artery and the aorta remains, allowing some oxygenated blood to move back into the heart rather than all of it moving out of the aorta and into the systemic circulation.

peripheral blood smear A glass slide containing a drop of blood; analysis allows the lab to microscopically examine the structure of the blood cells.

persistent pulmonary hypertension (PPHN) The persistence of elevated pressures in pulmonary vasculature after birth, associated with failure to transition from fetal circulation to postpartum or normal newborn circulation.

Pierre Robin syndrome A condition present at birth marked by a very small lower jaw (micrognathia). The tongue tends to fall back and downward (glossoptosis) and there is a cleft soft palate.

polyhydramnios An excessive amount of amniotic fluid. May cause preterm labor.

primary apnea The initial response to asphyxia at birth, characterized by increased respiratory rate and a slight decrease in heart rate with increased blood pressure; responds to stimulation and oxygen therapy.

proximal intestinal obstruction Obstruction at or above the level of the jejunum.

repogle A double-lumen tube used for gastric decompression in infants with gastrointestinal issues; for an infant, usually an 8F size is used and is connected to low intermittent wall suction to collect gastric secretions.

secondary apnea The response if asphyxia at birth continues, characterized by a period of gasping respirations, falling heart rate, and falling blood pressure; does not respond to stimulation unless resuscitation is initiated immediately.

surfactant A substance formed in the lungs that helps keep the small air sacs or alveoli from collapsing

and sticking together; a low level in a premature baby contributes to respiratory distress syndrome.

term newborn Used to describe an infant born between 37 and 42 weeks' gestation.

TORCH screen A set of lab values taken to detect congenital infections, including toxoplasma IgG and IgM (1:1,024), rubella titers, urine cytomegalovirus titer, viral culture (for herpes), and culture of scrapings from any vesicular lesions.

transient tachypnea of the newborn Transient respiratory distress, lasting a few hours, in the newborn and which is secondary to retained fetal lung fluid.

vacuum caput In a newborn, an accumulation of fluid at the site where a vacuum extractor was used to assist in delivering the newborn.

volvulus Twisting of the stomach or intestine, which often has the effect of cutting off its blood supply.

Bibliography

American Academy of Pediatrics and the American Heart Association. *Textbook of Neonatal Resuscitation*, 5th ed. Dallas, TX: American Heart Association; 2006:1-26–1-27.

The Apgar Score. American Academy of Pediatrics, Committee on Fetus and Newborn, American College of Obstetricians and Gynecologists and Committee on Obstetric Practice. Policy Statement. *Pediatrics*. 2006;117:1444–1447.

Crain EF, Gershel JC. *Clinical Manual of Emergency Pediatrics*, 4th ed. Available at: http://books.mcgraw-hill.com/getbook.php?isbn=0071377506&template=. Accessed April 4, 2009.

Jaimovich DG, Vidyasagar D. *Handbook of Pediatric & Neonatal Transport Medicine*, 2nd ed. Philadelphia, PA: Hanley and Belfus, Inc; 2002.

Kattwinkel J, ed. *Textbook of Neonatal Resuscitation.* Elk Grove Village, IL: American Academy of Pediatrics; 2006.

Laroia N. Birth Trauma. In: Itani O, Konop R, Clark DA, Wagner C, Finer N, eds. emedicine. Continuing Education. Available at: http://www.emedicine.com/ped/topic2836.htm. Updated April 16, 2008. Accessed April 28, 2009.

MacDonald MG, Seshi MMK, Mullett MD, eds. *Avery's Neonatology: Pathophysiology & Management of the Newborn*, 6th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; 2005.

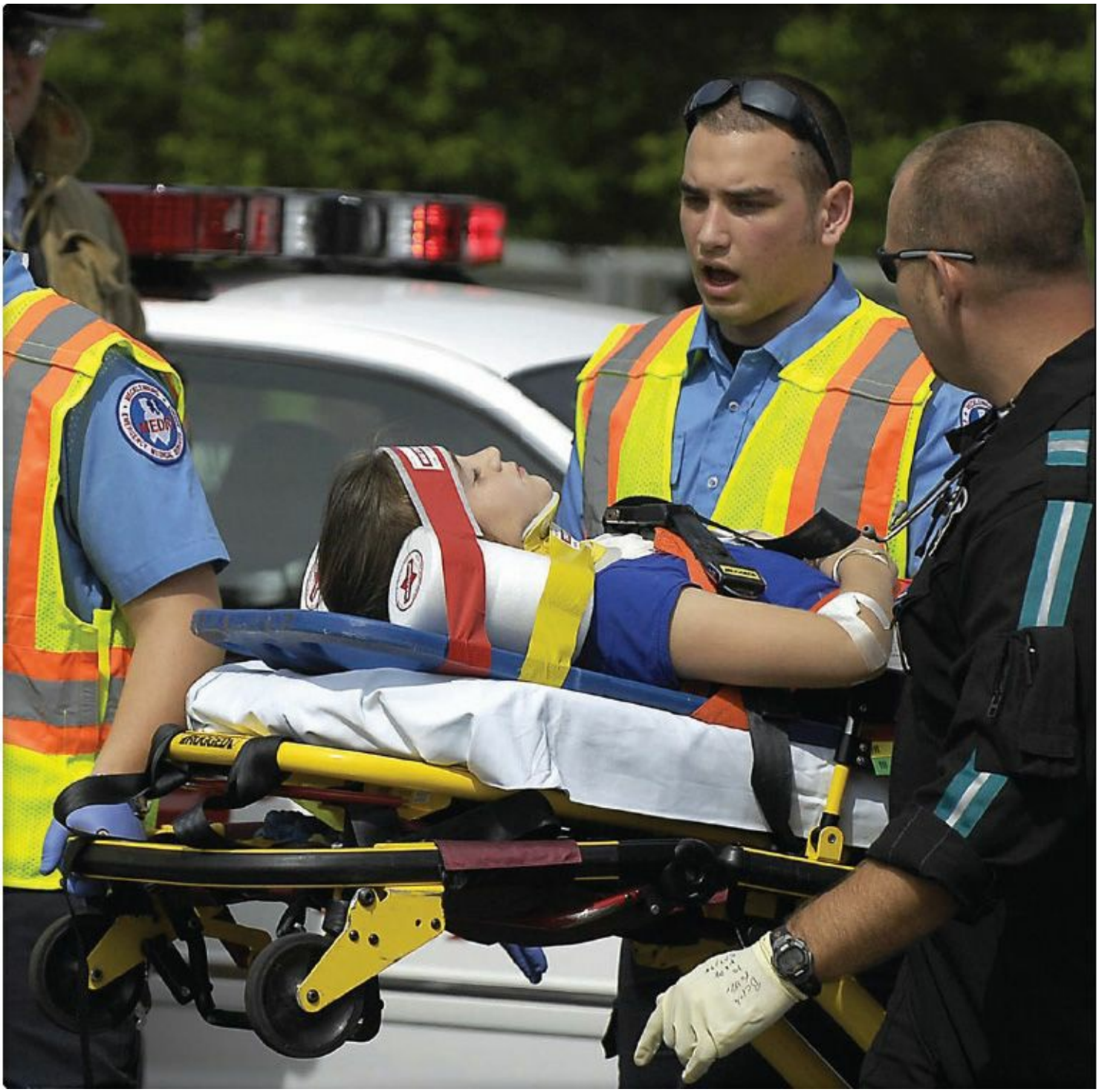
Martin R, Fanaroff A, Walsh M. *Fanaroff and Martin's Neonatal Perinatal Medicine: Diseases of the Fetus and Infant*, 8th ed. St Louis, MO: Mosby; 2005.

Pediatric Advanced Life Support. American Academy of Medicine. American Heart Association. Dallas, TX: 2006.

Saugstad OD, Ramji S, Soll RF, Vento M. Resuscitation of newborn infants with 21% or 100% oxygen: an updated systematic review and meta-analysis. *Neonatology*. 2008;94(3):176–182.

Taeusch WH, Ballard RA, Gleason CA. *Avery's Diseases of the Newborn*, 8th ed. Philadelphia, PA: WB Saunders Co; 2004.

Volpe JJ. *Neurology of the Newborn*, 5th ed. Philadelphia, PA: WB Saunders Co; 2008.



Pediatric Emergencies

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Objectives

1. Review the anatomic and physiologic differences between adult and pediatric patients (p 924–927).
2. Outline the differences in the general approach to critical care transport patient assessment between adult and pediatric patients (p 927–939).
3. Discuss the indications, dosage, and route of administration for medication administration in pediatric patients in the critical care transport environment (p 931–934).
4. Describe what special pediatric equipment may be needed in a critical care transport (p 939).
5. Discuss interfacility transport considerations relating to pediatric critical care transport (p 939–941).
6. Explain considerations when parents and caregivers accompany during a transport (p 941).
7. Discuss common medical emergencies in the pediatric population, including respiratory conditions, cardiac conditions, renal disorders, sepsis, and meningococcal infections, and their assessment and management in the critical care transport environment (p 943–961).
8. Examine common pediatric trauma emergencies, including head injuries, spinal cord injuries, related breathing and circulation abnormalities, fractures, and burns, and their assessment and management in the critical care transport environment (p 961–969).
9. Describe critical care transport management of a pediatric patient with suspected abuse or neglect (p 969–970).
10. Describe critical care transport management of a pediatric patient with hypothermia (p 970).
11. Describe critical care transport management of a pediatric patient with heat stroke (p 970–971).
12. Describe critical care transport management of a pediatric drowning patient (p 971).
13. Discuss how to transport a pediatric critical care patient with special health care needs (p 971–972).

Introduction

Most CCTPs experience some discomfort when caring for children who are in distress, primarily because of the limited experience that they may have with this population. Depending on the structure and the experience level of the critical care team (adult vs pediatric), these types of transports may be of high risk and low frequency. CCTPs must always remember that the pediatric patient is not a “small adult” and should be treated according to the parameters discussed within this chapter and the protocols set forth by your own agency medical director.

Pediatric patients differ anatomically, physiologically, and emotionally from adults. In addition, the

types of illnesses and injuries children sustain and their responses to them vary across the pediatric age span. When dealing with the often limited communication abilities of children, it is more difficult to rapidly and accurately assess them. For these reasons, CCTPs must tailor their approach to accommodate the developmental and social issues unique to pediatrics. Pediatric critical care transports also usually involve one or more caregivers who may be stressed or frightened themselves.

This chapter addresses some of the special considerations that will enhance the CCTP's effectiveness in caring for ill or injured children in the critical care transport environment. It discusses the special needs of children, the most common reasons for pediatric critical care transport, and critical care transport management for pediatric emergencies.

Anatomy and Physiology

Cardiovascular System

In the absence of an anomaly, the basic anatomy of the circulatory system in children is the same as it is for adults. However, there are some physiologic differences, such as different normal ranges for pulse rate and blood pressure. In addition, children have a limited duration of compensatory mechanisms. Once these mechanisms are diminished, the condition of the patient quickly deteriorates.

Respiratory System

Lung development begins in utero and continues through adolescence. Central regulation of respirations is immature in infants; therefore, the respiratory pattern of an infant is often irregular. It remains this way until the infant is older than 1 year, when the rate and depth of respiratory patterns become regular.

The lower airways of children are smaller and, therefore, more prone to obstruction and collapse **Figure 23-1**. Smaller airways cause greater resistance to airflow than do adult airways. Airways grow as the child grows, so it is logical that any disease causing narrowing of the airways, such as bronchiolitis, will be more detrimental in younger children than in adolescents or adults. Mucosa is less adherent to the airways and more prone to the development of edema, which contributes to increased airway resistance.

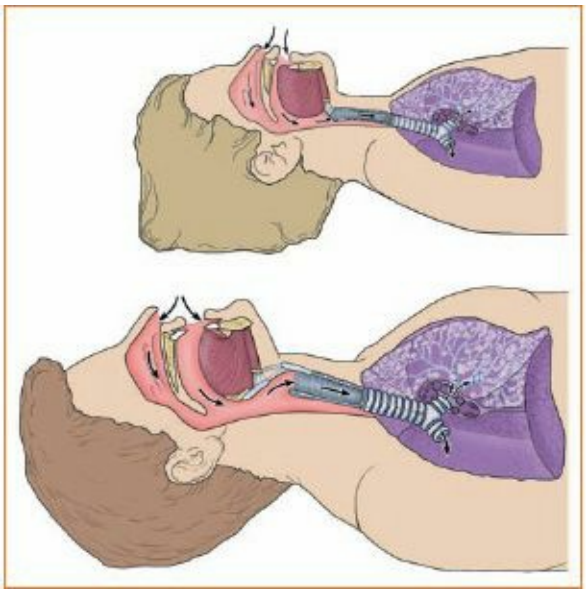

Figure 23-1 The anatomy of a child's airway differs from that of an adult's airway in several ways. The trachea is smaller in diameter and more flexible. The airway itself, including the larynx and vocal cords, is lower and narrower. The back of the head is larger, and head positioning requires more care. Finally, the mandible is proportionately smaller and the tongue is proportionately larger than in an adult.

At birth, the number of peripheral airways is limited. These airways then increase in number until about 7 years of age. The airways then increase in size through adolescence. Therefore, children up to 3 to 4 years of age are at a relatively higher risk of severe symptoms related to lower airway disease than are older children or adults.

During the first few years of life, alveoli mature from a single cell capillary bed to a double cell bed, followed by an increase in the communication between these alveoli, called collateral ventilation. Children may not be able to ventilate airways distal to an obstruction and may be at a higher risk of atelectasis.

Until adolescence, children have a relatively large tongue in relation to the size of their mouth. Another critical difference in the airway of young children is that the narrowest part of the trachea is the cricoid ring. In infants and toddlers, the epiglottis is long, floppy, and narrow compared with an adult **Figure 23-2**. The pediatric epiglottis tends to extend over the glottic opening more than in adults, and the vocal cords of children are thin and angled and have a lower attachment anteriorly. Later, we will discuss how these airway differences affect airway management.

Finally, infants have highly reactive airways, making them more prone to responses from environmental allergens, viral respiratory diseases, and hereditary factors.

Neurologic System

The basic brain anatomy and perfusion requirements in children are similar to those in adults; however, there are a few key anatomic differences. Children do not have a brain that is well compartmentalized, which means there is more room for the brain to move within the skull. This leads to an increased susceptibility to brain injuries, such as diffuse axonal injuries.

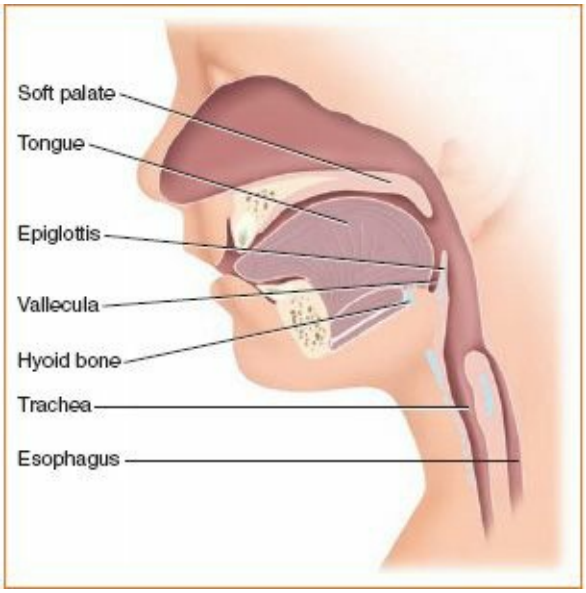

Figure 23-2 The child's epiglottis and surrounding structures.

Key anatomic and physiologic differences in the neurologic system exist between children and adults. The anterior or diamond-shaped fontanelle closes at 16 to 18 months of age, and the posterior fontanelle closes at 2 months of age **Figure 23-3**. A normal fontanelle is soft and flat but with a feeling of fullness. These differences are important to know when assessing children and infants. For example, a newborn may be born with a cephalohematoma—a collection of blood between the bone and subperiosteal space. The incidence of this condition is infrequently seen by the CCTP, but it occurs in 1% to 2% of all live births that involve mechanical trauma (forceps or vacuum-assisted delivery). This type of hematoma can occur anywhere on the skull but is found more predominantly in the parietal region of the

skull. The hematoma usually resolves in 6 to 8 weeks, and can be associated with linear skull fractures (10% to 25% of infants affected). The CCTP should note the size of the hematoma during the initial assessment and during the transport. Complications are rare, but include infection, hyperbilirubinemia, meningitis, and osteomyelitis. The blood mass does not cross the suture line and may increase in size after birth. Blood loss is minimal, but the size and location should be noted on the transport assessment documentation. Conversely, caput succedaneum is merely soft-tissue swelling and usually resolves within 24 hours.

Musculoskeletal System

Several differences exist between the musculoskeletal system of a child and an adult. First, children have fewer calcified bones. The immature bones of children are more porous than those of adults and tend to respond to kinetic forces by buckling rather than fracturing. If the forces are great enough to cause a fracture, an investigation of underlying organ injury is warranted. Epiphyseal-metaphyseal growth plate injuries account for 10% to 15% of fractures in children; however, with reduction and immobilization, most of these injuries have excellent outcomes.

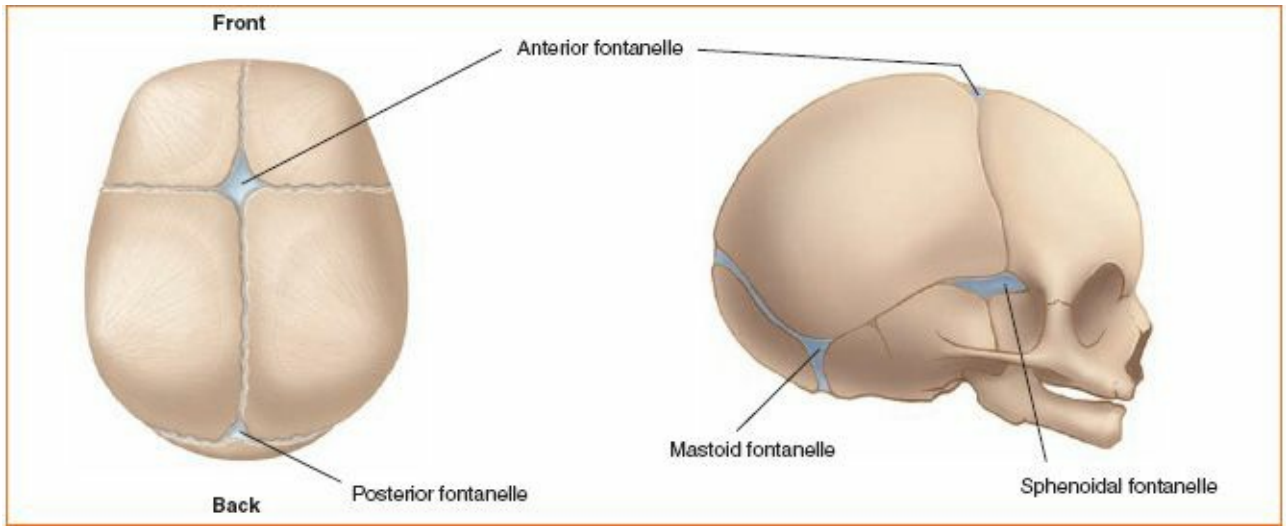

Figure 23-3 The fontanelles.

The child's thorax is pliable, meaning that it can withstand greater kinetic forces without fracturing. The chest is surprisingly resilient and can absorb a significant amount of energy compared to the child's size. However, rib fractures are not common. If a small rib fracture is detected or suspected, this suggests a large force of energy as its cause, possibly indicating a severe pulmonary contusion. In this situation, the pulmonary contusion is more of a life threat to the child than the rib fracture.

The ligaments in children are stronger and more resilient to tensile forces, thereby providing strength to the joints. As a result, dislocations are rarely found in pediatric populations compared with adult populations.

The pediatric spine has incomplete ossification, epiphyseal growth plates, and hypermobility. As a result, the cervical spine in children is highly flexible and young children tend to have avulsions rather than fractures. However, this hypermobility does not always protect the child's cervical spine. Compounding factors are that children have relatively large, heavy heads with weak neck muscles, causing the head to lean forward with a fall. This makes this age population more prone to head injuries. The saying "big heads go to ground" applies here. Rapid deceleration injuries resulting in hyperflexion and torsion forces are most commonly seen in children.

Gastrointestinal System

Several anatomic and physiologic differences increase the risk of aspiration in children. For example, the cardiac sphincter, which is the valve between the esophagus and the stomach, is located above the diaphragm until 6 months of age and does not close completely, leading to frequent regurgitation. Also, children have a smaller stomach capacity, which leads to more frequent meals. Therefore, the chances of encountering a child with a full stomach in an emergency situation are high. The child should be considered at risk for aspiration if the time of the last meal is unknown or if he or she has eaten within the past 4 hours.

Another key difference between adults and children is that the abdominal muscles are weaker in the younger child, providing less protection to the internal organs. The liver and spleen can be palpated below the costal margin; therefore, this makes these organs more susceptible to blunt abdominal trauma.

Liver function is also immature in young children, resulting in fewer glucose stores, a prolonged clotting time, and decreased clearance of drugs by the liver. For this reason, children who are critically ill or injured are susceptible to hypoglycemia. Therefore, an essential component of pediatric resuscitation is checking blood glucose levels.

Renal System

The pediatric renal system has some significant physiologic differences from the adult renal system, including that children have a higher percentage of body water than adults, making them more prone to dehydration. In addition, children are unable to concentrate urine as effectively as adults. They may be more prone to electrolyte loss because they have a higher clearance for blood urea nitrogen (BUN), creatinine, and electrolytes. The minimum urine outputs expected in pediatrics are as follows:

- Infants: 2 mL/kg/h
- Child: 1 mL/kg/h
- Adult: 0.5 mL/kg/h

Thermoregulation

Temperature regulation in infants is not fully developed, making them highly susceptible to hypothermia. Infants have thinner skin and lack a subcutaneous layer of fat. They are unable to shiver and lose heat through the head, which is proportionally larger in relationship to the body.

Children have a large body surface area to mass ratio relative to that of the adult population. This ratio can be advantageous to children when exposed to mildly warm environments and during times of intense exercise in mildly cool weather, because they have the increased ability to dissipate heat. In contrast, when this population is exposed to temperature extremes of cold and heat, their ability to dissipate heat can be a disadvantage that leads to environmental emergencies.

Metabolism

The physiology of pediatric metabolism, like other systems in this patient population, is at a higher rate compared with that of the adult population. The basal metabolic rate is accelerated in the pediatric population, which means the consumption of medications and oxygen is a concern for the transport team members. For example, an infant's consumption of oxygen is twice that of the adult patient. This known variable is important to consider when providing supplemental oxygen and when looking at the mode of transportation (for air transports, the higher the altitude, the more oxygen consumption a patient requires). The CCTP should also take into account the nature of the illness (medical vs trauma) and the likelihood of the patient decompensating during the transport. As with the rate of metabolism, the rate of onset of

hypercapnia and hypoxemia is also accelerated in this patient population, warranting close monitoring during transport.

Glucose Requirements

The pediatric population is predisposed to developing episodes of hypoglycemia secondary to several factors, such as an increased metabolic rate that ultimately depletes available serum glucose levels in the bloodstream. Infants and small children have decreased glycogen reserves and an immature liver that is not capable of stimulating glycogen stores when required. The additional stress on the body from an illness or after a traumatic event creates another opportunity for these hypoglycemic events to occur. The need to assess and reassess is paramount while caring for the pediatric patient during a transport. As with the adult patient, hypoglycemia can manifest at a rapid onset with either an asymptomatic or a classic presentation. Newborns and young infants may be asymptomatic or may have obvious nonspecific symptoms ranging from irritability to lethargy, pallor to cyanosis, and tremors to seizures. On the other hand, the older child will have a more classic presentation that mirrors adult symptoms (anxiety, diaphoresis, tachypnea, and weakness). The ideal treatment regimen for hypoglycemia varies depending on the weight and age of the patient involved. The typical hypoglycemic pediatric patient would be administered 25% dextrose at a dose of 2 to 4 mL/kg. For a neonate or preterm infant, 10% dextrose would be administered at a dose of 5 to 10 mL/kg. For an older child or adolescent, 50% dextrose would be administered at a dose of 1 to 2 mL/kg. If the hypoglycemic episode has been corrected prior to the arrival of the CCTP, then a glucose maintenance infusion should be initiated at a rate of 6 to 8 mg/kg/min or per standing protocol. Although the administration of 50% dextrose is ideal in the adult population, it is contraindicated in the pediatric population. 50% dextrose increases the plasma osmolarity in the blood, which subsequently leads to hypernatremia and cerebral edema in the pediatric patient.

Growth and Development

Physical

One of the important aspects of pediatric care is an understanding of normal growth and development. It is common for children to regress during an illness or when recovering from a serious injury. Failure to meet normal developmental milestones (such as the ability to walk or talk) may be a sign of an underlying illness, a family crisis, or a neurologic injury (ie, a lack of oxygen or blood flow to the brain that may permanently affect the child's ability to reach normal developmental milestones) **Table 23-1**. Knowledge of the child's developmental level also helps to guide safety considerations.

It is optimal to have an understanding of the child's developmental level prior to assessment so that appropriate comparisons can be made. Parents and other caregivers are the best source for information about how the child will react under normal circumstances. The parent or primary caregiver may be able to tell you the developmental age of the child, which will guide the manner in which you communicate with the child and explain procedures. Communication techniques for children who do not have language skills include using sign language, observing facial expressions, and having the child move his or her eyes to the right or left or blink a specific number of times to indicate yes or no.

TABLE 23-1 Developmental Milestones

Age, mo	Milestone
2	Smiling
5	Rolling over

9	Crawling
10	Grasping small objects
12	Walking
24	Talking

Psychosocial

Psychosocial concerns for pediatric patients vary tremendously based on the patient's age. An infant's primary fear is often separation from his or her parents, so efforts should be made to minimize the time of separation. Toddlers also fear separation from parents or caregivers, and they fear loss of control; allowing the toddler to help dictate the order in which you do different portions of an exam may help it go more smoothly. Preschoolers also fear loss of control, in addition to bodily injury, the dark, and the unknown; explain your actions clearly and provide plenty of reassurance. As with preschoolers, school-aged children fear body injury; simple and concise explanations should be used to describe procedures as ways to help the child. Adolescents and teenagers may have a fear of loss of control and alterations of body image; encourage the adolescent's involvement and respect the adolescent's privacy. Adolescents often have increased compliance if honest explanations are given and if they are allowed to participate in decision making regarding their own health care.

Basic care strategies for psychosocial concerns cross all age spectrums. Foster trust by speaking at eye level, using first names, and explaining medical procedures using age-appropriate words. Encourage a sense of control by offering choices whenever possible, while keeping in mind that in some situations, bargaining and negotiating may not be possible or appropriate. At all times, a calm and confident demeanor can help reassure both the parent and child.

Approach to Parents and Caregivers

CCTPs should do a self-assessment to ensure that they are prepared—academically, technically, and emotionally—to care for pediatric patients. Children of any age may be seriously ill or injured. You may be distracted with the emotional response of a child or parent. CCTPs should watch for signs of increased stress in their transport partners.

Be sensitive to the fact that the child is not the only patient; the needs of the parents, guardians, or caregivers must also be addressed. Common responses may include guilt for not preventing or recognizing the illness or injury, frustration related to not being the primary caregiver to the child during hospitalization (loss of parenting role), or helplessness from a perceived inability to protect the child. Parents often feel fearful, out of control, and uninformed, so an effort should be made to keep parents or other caregivers informed and actively involved in health care decisions. This often requires that information be repeated, sometimes on numerous occasions. Whenever possible, provide parents or caregivers with written information and involve them in the plan of care.

Approach to Pediatric Assessment

All pediatric assessment should begin with a general impression that can be accomplished using the **pediatric assessment triangle (PAT)** Figure 23-4. The PAT is an assessment tool that uses the child's appearance, work of breathing, and circulation. Appearance can be assessed using the TICLS (tickles) mnemonic: Tone, Interactiveness, Consolability, Look or gaze, and Speech or cry Table 23-2. The nature of the child's airway sounds and signs of increased breathing effort characterize work of breathing.

Circulation is assessed by observing the child for pallor, mottling, or cyanosis. By combining the three parameters of the PAT, you get a quick idea of the physiologic stability of a child and, in conjunction with the chief complaint, make decisions regarding the need for life support.

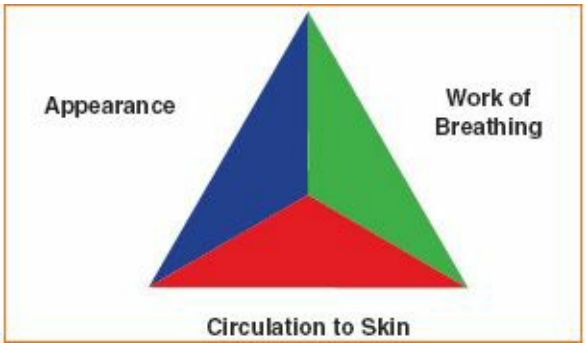

Figure 23-4 The pediatric assessment triangle. Used with permission of the American Academy of Pediatrics, *Pediatric Education for Prehospital Professionals*, © American Academy of Pediatrics, 2006.

After using the PAT to form a general impression of the patient, perform a primary assessment evaluating, in order:

- Airway patency
- Respiratory rate and quality
- Pulse rate
- Skin temperature and capillary refill time
- Blood pressure
- Neurologic status

A child's neurologic status can be obtained by forming a general impression and assessing the level of consciousness and pupillary reaction. Any abnormal finding indicates a need for immediate treatment and may require deferral of the next step (ie, if the airway is not patent as the result of an airway obstruction, intervention must be immediate). A secondary assessment should be performed on medical and trauma patients.

All pediatric patients, even the youngest children, should be assessed for pain. The quality of pain can be assessed using a numeric scale for older children and adolescents. Younger children and infants may be assessed using pain scores, such as the **FLACC scale** **Table 23-3** or the FACES scale **Figure 23-5**.

Each of the five categories that make up the FLACC scale—face, legs, activity, cry, and consolability—is scored from 0 to 2, which results in a total score between 0 and 10.

Although physiologic abnormalities are more likely to be identified by the PAT and primary assessment, the secondary assessment typically helps identify anatomic abnormalities. Sometimes this exam is not possible to perform because the patient is in an unstable condition and needs to be transported immediately. In this case, the exam should be conducted en route to the receiving facility. The secondary assessment focuses on the specific body part or body system involved.

To fully assess a child, a hands-on approach is needed in addition to the use of technical equipment. Touch can provide many clues to a child's condition, which might otherwise go unnoticed. As you assess a child's vital signs, assess skin color, temperature, and turgor. Auscultate the child's chest with a stethoscope and note the rate and quality of respirations, as well as chest and abdominal movements.

TABLE 23-2 Characteristics of Appearance: The TICLS Mnemonic

Characteristic	Features
Tone	Is the child moving or resisting examination vigorously? Does the child have good muscle tone? Is the child limp, listless, or flaccid?
Interactiveness	How alert is the child? How readily does a person, object, or sound distract the child or draw the child's attention? Will the child reach for, grasp, and play with a toy or exam instrument, such as a penlight or tongue blade? Is the child uninterested in playing or interacting with the caregiver or CCTP?
Consolability	Can the child be consoled or comforted by the caregiver or the CCTP? Is the child's crying or agitation unrelieved by gentle reassurance?
Look or gaze	Does the child fix his or her gaze on a face, or is there a "nobody home" glassy-eyed stare?
Speech or cry	Is the child's cry strong and spontaneous or weak and high pitched? Is the content of speech age appropriate or confused or garbled?

TABLE 23-3 FLACC Scale

Category	Scoring		
	0	**1**	**2**
Face	No particular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid, or jerking
Cry	No cry (awake or asleep)	Moans or whimpers, occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging, or being talked to, distractible	Difficult to console or comfort

Figure 23-5 Pictures such as the Wong-Baker FACES pain rating scale allow for self-assessment of pain in young children. From: Hockenberry MJ, Wilson D, Wikelstein ML: *Wong's Essentials of*

Pediatric Nursing, ed. 7, St. Louis, 2005, p. 1259. Used with permission. Copyright, Mosby.

Figure 23-6 Use of a length-based resuscitation tape is one way to estimate a child's weight and to identify the correct size for pediatric equipment and medication doses.

Staff at the receiving facility will determine whether a child is weighed in triage or on arrival to the treatment area. At the facility, the actual (not estimated) weight is measured and reported in kilograms. In the critical care transport setting, you should obtain the child's weight from the parents or caregivers, and if that is not available, estimate the child's weight using a tool such as a length-based resuscitation tape (eg, Broselow tape) **Figure 23-6**.

History

A significant allergy in children who have neural tube defects is latex allergy. This can cause life-threatening reactions, and all latex products must be removed from the care areas.

Parents or caregivers can provide extensive information regarding the child's history. Pertinent questions should include those about what is the child's current behaviors, such as eating and sleeping patterns, input and output, and activity level. This information can then be compared with the child's usual behavior patterns in these areas. The history for infants and chronically ill children will also include the perinatal history, the delivery history, gestational age, and gestational weight.

Scene or situation information may be useful as well. For example, be sure to make inquiries to responders or determine from prehospital reports any information, such as heating systems, a dilapidated automobile, and other environmental factors, that may have contributed to the situation. The CCTP may discover additional information from family or other sources.

Pediatric Medication Delivery

Delivery of medications in pediatric emergencies can be quite challenging because many emergency medications are prepared using common adult doses (such as medications for cardiac arrest that come in prefilled syringes). Pediatric medication doses often require the CCTP to perform calculations, which increases the possibility of error. Doses of high-alert medications, such as narcotics, heparin, insulin, and potassium chloride, require an independent recheck before they are administered.

Several methods are available for delivery of medications. Medications may be delivered via IV push, syringe pump, or infusion pump. Because infusion pumps have been developed with safety guards, such as a flow stop, they are routinely used for the administration of fluids and medications. An additional safety tool worth mentioning is the buretrol, an inline container between the patient's IV catheter set and the bag of IV fluids. The maximum capacity of the buretrol is set to 150 mL of fluids to be infused. The rationale behind using a buretrol is to fill the buretrol to a designated level. Then, if the IV pump malfunctions, only that volume will flow into the patient, thus preventing overhydration (pulmonary edema). Buretrols are no longer commonly used in transport systems; however, they are still commonly used by many agencies. CCTPs should work with referral agencies and involve them in the discussion of best practices for pediatric infusions.

The best option for administering resuscitation medications is a vascular access device. A pediatric resource with a formulary may be helpful in determining the amount and type of diluent required for specific medications. The formulary will also list the time frame for administration. When using a syringe or infusion pump, always follow the manufacturer's recommendations for flushing and maintenance.

Controversies

Pediatric medication errors are a major focus of concern in hospitals. Measuring devices used to calculate emergency drug doses have been found to contribute to errors for hospitalized children. Although use of the measuring devices for estimation is appropriate for initial prehospital and emergency department encounters, an accurate height and weight must be obtained on admission and used to prepare an individualized dosing list of emergency medications for each child.

Figure 23-7 A nasal cannula may be the most effective way to provide oxygen to an infant in respiratory distress.

Medications used in pediatric critical care transport are summarized in **Table 23-4**.

Airway

Although adults cannot breathe and swallow at the same time, infants have this necessary skill so that they can breathe while nursing. All infants are obligatory nose breathers, meaning they only actively breathe through their mouths if they are crying. Therefore, nasal congestion to an infant can be life threatening. Normal saline spray and a bulb syringe is often all that is necessary to clear the nasal airways. Positioning the infant with the head up 30° to 45° also aids in keeping the nasal airways clear. Administration of oxygen via a nasal cannula may work well in infants with mild to moderate respiratory distress because a significant amount of tidal volume comes through their noses and they do not tolerate face masks **Figure 23-7**.

As mentioned earlier, children have a relatively large tongue in relation to the size of their mouth. Proper choice of blade size, proper positioning, and effective sweeping of the tongue are essential for successful intubation.

Numerous airway differences can interfere with airway patency. To accommodate these differences, a "twisting" technique is sometimes used to insert the endotracheal tube more easily past the vocal cords. This prevents the tube from getting caught on the angled cords. In addition, the trachea of most pediatric patients is anterior, found close to C2 to C3. As the result of the lack of cartilaginous support around the airway, the pediatric upper airway is more easily compressed. This is best managed by avoiding

hyperextension of the neck. Placing the noninjured child in the sniffing position with the aid of towel rolls provides optimal positioning **Figure 23-8**. It is also common to find large tonsils and often copious oral secretions in pediatric patients.

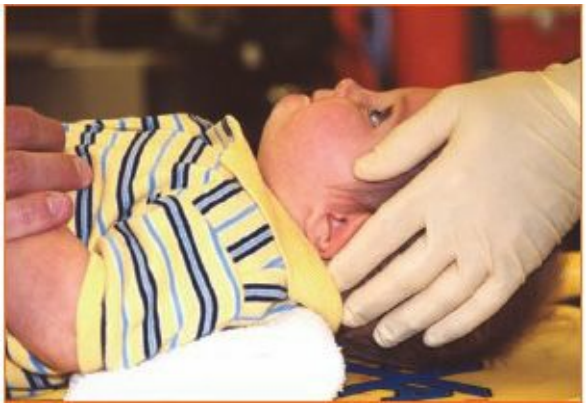

Figure 23-8 A shoulder roll should be used in an infant without trauma to position the airway in a neutral position.

Patency of the pediatric airway may be impaired for numerous reasons, including congenital anomaly, infections, trauma, edema, any reason that would diminish protective reflexes, or foreign body airway obstruction. Foreign body airway obstruction is of particular risk for children younger than 5 years. Diminished protective reflexes may include any condition causing a decreased level of consciousness and/or neuromuscular disorders.

Upper airway obstruction may be recognized by signs of nasal flaring and increased inspiratory effort with a prolonged inspiratory phase. Stridor, snoring, gurgling, or wheezing respirations are noted. Basic assessments include observing whether the child can open his or her mouth without difficulty, cry, and/or vocalize. The quality of these vocalizations and the quality of the air exchange should be noted. In most cases, the minutes taken in proper preparation make securing the airway faster and easier.

A child with a decreasing level of consciousness may be at risk for no longer being able to protect his or her airway and may require endotracheal intubation. Protective reflexes are lost in ascending order. The first reflex lost is swallowing, then coughing, then the gag reflex, and finally the corneal reflex. A useful intubation “pearl” is to assess the corneal reflex prior to inserting an oral airway or to assess for paralysis related to neuromuscular blocking agents. If the child does not blink when the open eyelids are stroked, the child no longer has a gag reflex. This prevents the risk of aspiration by testing the gag reflex with a tongue blade or a suction catheter, which may cause the child to vomit.

TABLE 23-4 Pediatric Medications for Critical Care Transport

Medication Class	Indications	Mechanism of Action	Commonly Used Medications	Dosage (Route of Administration Is IV, IO, or as Indicated)	Side Effects Common to This Class of Medications	Special Considerations
Airway medications						
Ultra-short-acting nonbarbiturate hypnotic agents: etomidate type	Induction agent to facilitate airway device placement	Depresses the reticular activating system by stimulating GABA receptors	Etomidate (Amidate)	0.2–0.4 mg/kg (maximum: 20 mg)	Adrenal suppression, apnea, hypotension, hypertension, tachycardia, bradycardia, myoclonus, nausea, vomiting	Multiple doses increase the likelihood of adrenal suppression. Avoid extravasation.
Ultra-short-acting nonbarbiturate hypnotic agents: ketamine type	Induction agent to facilitate airway device placement	Interrupts pathways within the cortex and limbic system; prompts release of endogenous catecholamines	Ketamine (Ketalar)	1–2 mg/kg	Hypertension, emergence reactions, increased ICP, vivid dreams, tremors	Consider premedication with benzodiazepines to prevent an emergence reaction. Ketamine may be the preferred induction agent in patients with respiratory failure.
Benzodiazepines	Sedative agents, often administered to facilitate airway device placement; maintenance sedative agents; anxiolytic agents; first-line anticonvulsant agents	Binds with benzodiazepine receptors on GABA neurons; enhances GABA inhibition of the CNS, resulting in CNS cellular depression	Diazepam (Valium) Lorazepam (Ativan) Midazolam (Versed)	0.5–1.0 mg/kg 0.1–0.4 mg/kg 0.2–0.3 mg/kg	Sedation, respiratory depression, apnea, hypotension, ataxia, vasodilation	Benzodiazepines may cause paradoxical reactions in children. They are not ideal for use as a sole agent for anesthesia induction. They may produce desirable amnesia effects. The antagonist flumazenil (Romazicon) is available for use in selected situations involving benzodiazepine overdose.
Depolarizing muscle relaxant agent	Short-term chemical paralytic to facilitate airway device placement	Activates, binds, and then occupies with acetylcholine receptor sites at the neuromuscular junction, preventing further activation, and leading to chemical paralysis	Succinylcholine (Anectine)	1–2 mg/kg	May cause or worsen hyperkalemia, causes a transient increase in ICP, may cause bradycardia (especially in children), muscle fasciculations, malignant hyperthermia	Consider pretreatment with atropine in patients prone to bradycardia. Do not administer succinylcholine in repeat doses or as an IV drip. Avoid or use with caution in patients with acetylcholinesterase poisoning. Use with caution in patients with neuromuscular disease. Provides no analgesia or sedation; must be accompanied by analgesic and sedative medications.
Nondepolarizing muscle relaxant agents	Short-term chemical paralytic to facilitate airway device placement; chemical paralytic agents for patients who require ongoing chemical paralysis for transport and certain modes of mechanical ventilation	Competitively antagonizes acetylcholine at receptor sites of the neuromuscular junction, causing extended chemical paralysis	Rocuronium (Zemuron) Vecuronium (Norcuron) Pancuronium (Pavulon) Cisatracurium (Nimbex) Atracurium (Tracrium)	0.6–1.0 mg/kg 0.1–0.3 mg/kg 0.08–0.1 mg/kg 0.15–0.2 mg/kg 0.4–0.5 mg/kg	Apnea in unventilated patients, hypotension, hypertension, tachycardia, or bradycardia; atracurium may cause bronchospasm or excessive secretions	Use cautiously in patients with neuromuscular disease. These medications provide no analgesia or sedation and must be accompanied by analgesic and sedative medications.

Respiratory medications						
Beta-agonist medications	Reactive airway disease, bronchospasm	Activate beta-2 receptors in the respiratory tract, causing bronchodilation	Albuterol (Ventolin)	Neb: 0.1–0.2 mg/kg/dose Max: 4 mg/dose in 5 mL NS every 15–20 min	Tachycardia, hypotension, hypertension, chest pain, CNS stimulation, irritability, nervousness, headache, palpitations	Prolonged use of albuterol may cause metabolic acidosis. Use with caution in patients with cardiovascular disease.
			Terbutaline (Brethire)	Neb: 0.1–0.3 mg/kg/dose Max: 3 mg/dose in 2.5 mL NS every 15–20 min		
			Epinephrine (Adrenaline)	SC: 0.01 mL/kg/dose of 1:1,000 to maximum of 0.3 mL/dose every 20 min up to 2 doses		
Anticholinergic bronchodilator medications	Reactive airway disease, bronchospasm	Block acetylcholine at bronchial smooth muscle receptor sites	Ipratropium (Atrovent)	Metered-dose inhaler only, two inhalations; repeat in 4–6 h	Palpitations, bronchitis	Administered only every 6–8 h.
Theophylline derivatives	Reactive airway disease, bronchospasm	Block phosphodiesterase and stimulate the release of endogenous catecholamines	Theophylline or aminophylline	6 mg/kg over 20 min, then continuous drip	Tachycardia, nervousness, restlessness, nausea, vomiting	Numerous food and medication interactions may occur.
Corticosteroids	Inflammatory disorders, airway edema, cerebral edema, high-altitude pulmonary edema	Suppress immune response; restore normal capillary permeability; limit neutrophil migration	Methylprednisolone (Solumedrol)	Neb: 2 mg/kg (load); 1–2 mg/kg/dose every 6 h	Insomnia, nervousness, immunosuppression, hyperglycemia	Use with extreme caution or avoid in patients with many active infections. Numerous side effects may occur, especially with long-term administration. Numerous medication interactions may occur.
			Prednisone	PO: 0.1–2 mg/kg/d in 1–4 divided doses		
			Dexamethasone (Decadron)	0.03–0.15 mg/kg/d PO, IV, IM divided twice a day to four times a day		
Cardiovascular medications						
Alpha-adrenergic antagonists, ADHD treatment	Hypertension, unwanted vasoconstriction from exogenous or endogenous catecholamines, pheochromocytoma	Block alpha receptors in vascular smooth muscle, preventing vasoconstriction from catecholamines	Clonidine	PO: 0.0005–0.010 mg/kg/d in divided doses every 6–12 h	Hypotension, tachycardia, dizziness, weakness, dry mouth, flushing	Phentolamine is infiltrated into subcutaneous tissues affected by extravasation of vasoconstrictor medications to prevent tissue necrosis.
Antiarrhythmic class I medications	Treatment of various atrial or ventricular arrhythmias (different class I agents are appropriate for different types of arrhythmias)	Block fast sodium channels, inhibit depolarization of neuronal cells, and decrease myocardial conduction velocity and automaticity	Lidocaine	1 mg/kg per dose	Hypotension, nausea, vomiting, unwanted conduction disturbances, cardiovascular collapse (numerous others)	Correct any electrolyte disturbances. Avoid use in patients with any significant cardiac conduction abnormality without an appropriately functioning pacemaker.
			Lidocaine infusion	20–50 µg/kg/min		

Antiarrhythmic class III medications	Various atrial or ventricular arrhythmias	Block potassium channels, which delay phase 3 repolarization and increase the effective refractory period	Amiodarone	5 mg/kg	QT interval prolongation, hypotension, bradycardia, other conduction abnormalities, flushing, edema, numerous cardiac arrhythmias, cardiovascular collapse (numerous additional side effects)	Amiodarone affects sodium, potassium, and calcium channels along with alpha- and beta-blocking properties. Amiodarone has an exhaustive list of side effects and medication interactions.
Antiarrhythmic medications: adenosine	Paroxysmal supraventricular tachycardia with or without Wolff-Parkinson-White syndrome	Replicate endogenous nucleoside, which slows or interrupts reentry pathways through the AV node	Adenosine (Adenocard)	0.1–0.2 mg/kg (maximum strength dose, 12 mg)	Flushing, headache, dyspnea, hypotension, ECG changes, hyperventilation; prolonged asystole is possible	Administer rapidly through the largest proximal vein possible. Expect a significant sinus pause immediately following IV administration. Warn patient, family, or other bystanders of this event. Several significant medication interactions occur with adenosine.
Anticholinergic medications	Bradycardia, asystole, acetylcholinesterase inhibitor toxicity may be given to prevent bradycardia in patients undergoing rapid-sequence intubation; may also be used as a mucolytic agent in certain circumstances	Block acetylcholine receptors in the parasympathetic nervous system; counteracts excessive vagal stimulation	Atropine	0.02 mg/kg per dose Minimum dose: 0.1 mg Maximum single dose: 0.5 mg in child; 1.0 mg in adolescents	Arrhythmias, flushing, tachycardia, dry mucous membranes (may be beneficial), various CNS changes	Extreme quantities may be required for severe acetylcholinesterase inhibitor toxicity. Tachycardia is not an indication to withhold atropine in patients with acetylcholinesterase inhibitor toxicity.
Catecholamines and sympathomimetic agents	Bronchospasm, airway edema, cardiac arrest, allergic or anaphylactic reactions, profound hypotension, profound bradycardia; certain medications may also be used when inotropic support or renal perfusion is needed	Mimic various effects and functions of the sympathetic nervous system through stimulation of alpha, beta-1, beta-2, and dopamine receptors in blood vessels, tissues, and organs	Dobutamine (Dobutrex) Dopamine (Inotropin) Epinephrine (Adrenaline)	2.5–20 µg/kg/min 2.0–20 µg/kg/min Bradycardia/asystolic/pulseless arrest: 0.01 mg/kg (1:10,000) ET: 0.1 mg/kg (1:1,000)	Hypertension, tachycardia, worsening CHF, increased myocardial oxygen consumption, cardiovascular collapse, peripheral vasoconstriction/ischemia, anxiety, nervousness, restlessness (numerous others)	Continuous hemodynamic monitoring is essential for any of these medications. Administer via central venous access whenever possible; peripheral administration may cause tissue necrosis if the IV line infiltrates. Many vasoactive medications will worsen myocardial performance in compromised patients. Avoid using sodium bicarbonate in the same IV lines.

Phosphodiesterase inhibitors	Indicated in situations in which both vasodilation and inotropic support are required	Inhibits cAMP in myocardial and vascular tissues	Milrinone (Primacor)	50–75 µg/kg over 10 to 60 min followed by 0.5 to 0.75 µg/kg/min	Hypotension, arrhythmias, nausea, vomiting	Do not use during or immediately after an acute myocardial infarction.
Neurologic medications						
Opioid antagonist medications	Reversal of opioid intoxication following accidental, intentional, or therapeutic overdose	Displace opioids and bind with opioid receptor sites in the CNS	Naloxone (Narcan)	< 20 kg use 0.1 mg/kg IV or 0.2–0.5 mg/kg ET; > 20 kg use 2.0 mg IV or 5 mg ET	May precipitate withdrawal symptoms in patients addicted to opioids	Carefully titrate naloxone intervals and dosing to reverse opioid-induced respiratory depression and sedation without precipitating withdrawal symptoms. Many opioids have a longer duration of action than naloxone, risking undetected return of toxic symptoms.
Miscellaneous medications						
Alprostadil (prostaglandin E)	Maintains patent ductus arteriosis in neonates with certain ductal-dependent congenital heart defects	Prostaglandin, which causes direct relaxation of ductus arteriosis and vascular smooth muscle		0.05–0.1 µg/kg/min IV infusion	Apnea, flushing, fever, bradycardia, tachycardia, hypotension, hypertension, cardiac arrest	Anticipate more than 10% chance of apnea. Consider early rapid-sequence intubation or airway management. Consult appropriate specialist when managing neonatal patients who require alprostadil.
Calcium chloride, calcium gluconate	Documented or presumed hypocalcemia, hyperkalemia with ECG changes, magnesium toxicity, hydrofluoric acid exposure	Replacement of lost or sequestered body calcium; membrane stabilizer that promotes normal cell function		Calcium chloride: 10–20 mg/kg (0.2 mL/kg) Calcium gluconate: 30–100 mg/kg (0.6–1 mL/kg)	Hypercalcemia, tissue necrosis following extravasation, arrhythmias, hypotension, bradycardia	Avoid IV calcium in patients with cardiac glycoside toxicity. Closely monitor peripheral IV sites for signs of infiltration.
Magnesium sulfate	Hypomagnesemia, ventricular arrhythmias	Stabilizes excitable cell membranes, slows SA node conduction, decreases acetylcholine in motor nerve terminals, electrolyte essential for ion movement across cell membranes		25–50 mg/kg (maximum dose: 2 g) over 10 to 20 min; faster for torsade de pointes	Flushing, hypotension, respiratory depression/apnea, vasodilation, cardiac conduction abnormalities	Have IV calcium available when administering magnesium sulfate. Monitor respiratory status, blood pressure, and deep tendon reflexes in patients receiving a continuous magnesium infusion.
Sodium bicarbonate	Certain types of metabolic acidosis, for enhanced elimination of certain toxic exposures, severe hyperkalemia, rhabdomyolysis	Powerful alkalinizing agent that stabilizes various cell membranes, increases serum and urinary pH, promotes renal excretion of weak acids and myoglobin		1 mEq/kg per dose	Cerebral hemorrhage, edema, worsening of congestive heart failure, hypernatremia, metabolic alkalosis, hypocalcemia, hypokalemia, altered hemoglobin oxygen affinity	Drug is not indicated for routine administration during cardiac arrest unless hyperkalemia or metabolic acidosis is documented. Monitor serum potassium during and after administration.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; AV, atrioventricular; cAMP, cyclic adenosine monophosphate; CHF, congestive heart failure; CNS, central nervous system; ECG, electrocardiogram; ET, endotracheally; GABA, gamma-aminobutyric acid; ICP, intracranial pressure; IM, intramuscularly; IO, intraosseous; Max, maximum; Neb, nebulizer; NS, normal saline; PO, orally; SA sinoatrial; SC, subcutaneous.

Breathing

Children with a lower airway obstruction may experience end-expiratory grunting. Grunting occurs when the child is trying to exhale against a partially closed glottis. This is a physiologic positive end-expiratory pressure (PEEP) when they are attempting to "pop open" their alveoli.

Another significant respiratory consideration related to children is that the diaphragm is the chief muscle for ventilation. If the diaphragm fails, the child may develop paradoxical breathing. The breathing pattern is described as a seesaw type motion, because the abdomen and chest are moving in opposite directions. Intercostal and accessory muscles are not fully developed until the child is school aged.

The chest wall of infants and children is very compliant and, therefore, is more prone to retractions when conditions increase airway resistance, such as when disease is present. This ineffective breathing pattern is evidenced by increased attempts to use the accessory muscles. These measures are typically

unsuccessful and lead to greatly increased work of breathing and eventual fatigue. Children who are having difficulty breathing usually cannot hide it, which makes assessing the work of breathing easier. Tools used to qualify the work of breathing may include an assessment of stridor, snoring, retractions, head bobbing, accessory muscle use, tripoding, nasal flaring, wheezing, and grunting.

Children having respiratory difficulties often place themselves in a tripod position **Figure 23-9**, which allows for better gas exchange because the lungs are located more anteriorly in the chest. Respirations and ventilations rarely improve in a supine position. In addition, as the stomach fills up with air (aerophasia), secondary to the increased respiratory rate, the child may be at a higher risk of aspiration.

The respiratory rate and depth can indicate the child's impending respiratory status. The most effective way of increasing a child's minute ventilation is to increase the depth of respirations. This is seen in diabetic ketoacidosis when a child presents with Kussmaul respirations. However, when disease states are primarily pulmonary related, the increased airway resistance and lower chest wall compliance leads to rapid, shallow respirations (tachypnea).

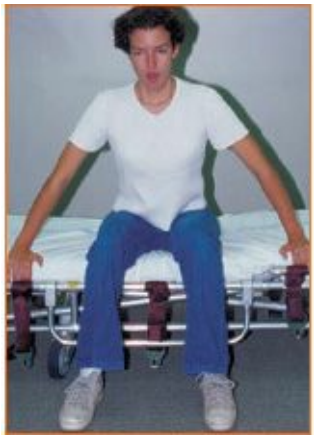

Figure 23-9 A child in a tripod position is maximizing his or her accessory muscles of respiration.

The same factors that can increase the heart rate can increase the respiratory rate, including pain, fear, and fever. For every 1°C rise over 37°C, a child's respiratory rate can increase by 10 breaths per minute. As respiratory distress progresses, respiratory rate may become slower and more irregular. Bradypnea (a slow respiratory rate) is a late sign of respiratory distress and predicts impending respiratory failure. Immediate interventions are required to prevent complete respiratory and/or cardiac collapse. Children have a high metabolic rate and, consequently, a high oxygen demand. Oxygen demand for infants is 6 to 8 mL/kg/min compared to adults (3 to 4 mL/kg/min). Therefore, the pediatric populations are at risk of developing hypoxia sooner than adults. Parameters for pediatric vital signs are presented in **Table 23-5**.

As with every patient population, it is imperative to assess the patient and not the numbers or monitors. A decreasing respiratory rate may indicate improvement if accompanied by improved mental status or a decrease in air hunger; however, a decreased rate with worsening mental status may be an ominous clinical sign. Assessment of the child's level of consciousness during the primary/initial assessment, with particular emphasis on appearance, work of breathing, and circulation (the pediatric assessment triangle), provides critical clues to the child's oxygenation status. Brain tissue has a high level of oxygen consumption, and hypoxia can result in an altered mental status. A child with mild to moderate hypoxia may appear agitated and anxious. As the hypoxia progresses, the child may become quiet and sleepy. Do not be falsely reassured that the child is better. Key assessment points should be focused on ascertaining whether the child can maintain and protect his or her airway. In addition, determine whether the child can maintain the current respiratory effort and determine the likelihood of deterioration en route

to the treatment facility. It is imperative for the CCTP to monitor the patient's respiratory rate, pattern, oxygen saturations, and electrocardiographic (ECG) waveform for any changes in heart rate (tachycardia or bradycardia) or increased cardiac ectopic foci (premature atrial contraction, premature junctional contraction, or premature ventricular contraction).

TABLE 23-5 Normal Pediatric Vital Signs

Stage	Respiratory Rate (breaths/min)	Pulse Rate (beats/min)	Minimal Systolic Blood Pressire (mm Hg)
Infant	25–50	70–160	> 70
Toddler	20–30	90–150	> 80
Preschool-aged child	20-25	80–140	> 80
School-aged child	15–20	70–120	> 80
Adolescent	12–16	60–100	> 90

Palpation of the chest is useful when assessing chest excursion. Excursion should be equal from left to right and sufficient in quality. This means there is a noted rise and fall of the chest that suggests adequate ventilations. However, chest wall movement does not necessarily equal adequate air movement. The chest wall may be working, but there may be inadequate gas exchange related to upper and/or lower airway impedance.

Auscultation is another tool used to assess breathing adequacy. Children may exhibit the same adventitious breath sounds as adults. This may include fine rales or crackles, coarse crackles, rhonchi, wheezing, or a silent chest. Children have thin chest walls that are conductive to referred sounds. Care must be taken to compare lung sounds from both lungs. Lung sounds may be heard over a partially collapsed lung (pneumothorax), but they are referred sounds coming from the intact lung. Noting the pitch is the important factor. The collapsed lung may have a deeper and less loud pitch than the lung that is inflated. Consider if the absence of lung sounds is from consolidation, no air movement, or a pneumothorax. High suspicions can lead to very different prehospital treatment modalities, depending on findings of the history and physical exam.

Measurements commonly used in the field to help determine breathing adequacy include oxygen saturation as measured by pulse oximetry (SpO_2) and end-tidal carbon dioxide ($ETCO_2$) values. SpO_2 readings have been found to be a reliable, useful tool in the prehospital setting. SpO_2 only measures the percentage of hemoglobin molecules that are bound with oxygen, which does not provide a direct correlation with respiration (the exchange of oxygen and carbon dioxide). A child with profound anemia may have an SpO_2 of 100% because all the hemoglobin molecules are saturated. However, if there are insufficient hemoglobin molecules available, the child's end-organ tissue needs for oxygen are not being met. In carbon monoxide (CO) poisoning, CO has a higher affinity to hemoglobin than oxygen, so it readily binds to the hemoglobin molecule. The SpO_2 is 100%, but it is not measuring oxygen bound to hemoglobin, but rather oxygen bound to CO. Again, the child's end-organ tissue needs for oxygen are not being met. Other factors that can interfere with the reliability of SpO_2 measurements include poor perfusion and whether the measured extremity is cold.

Measurement of $ETCO_2$ is the standard of care for assessing adequate respiration in intubated patients. CO_2 is the byproduct of respiration and is more reliable for measuring adequate gas exchange. $ETCO_2$ monitors are available for tracheal tubes and nasal cannulas. Normal values for $ETCO_2$ are 35 to 45 mm Hg, which are the same values found in arterial blood gas (ABG) samples.

Finally, ABG determinations can provide information not only on gas exchange but also on compensatory mechanisms. ABG determinations may be most useful for children when the need for intubation is not clear-cut. Those children who present in obvious respiratory failure or impending failure need to have immediate interventions and should not wait for laboratory confirmation. ABG determinations are also useful when assessing the adequacy of mechanical ventilators. Be careful to weigh delays at the bedside with information obtained from an ABG determination (it usually takes about 20 minutes to receive ABG results, which may be too long to wait).

Circulation

Physical assessment of a child's cardiovascular system begins with observing the child's general appearance and level of consciousness. A child who is well perfused will be watching the CCTP and other activity in the room. Observation of the chest may reveal the child's heart beating through the skin (precordial activity); this is a normal finding in pediatric patients. Assessment of the fingers and toes should be conducted to look for evidence of clubbing. Clubbing is the broadening of a child's fingers and toes in response to chronically low oxygen levels for longer than 3 months.

Poor cerebral perfusion can be manifested with agitation, confusion, or a progressive decrease in response to external stimuli. The brain is the last vital organ to be affected by hypoperfusion. A change in level of consciousness, therefore, is significant.

In septic shock or traumatic brain injury, the mean arterial pressure (MAP) may be more important in guiding therapy. MAP is a better measure of end-organ perfusion. The MAP may be measured by an arterial line or a noninvasive blood pressure monitor. MAP may also be calculated with the following formula,

$$MAP = DBP + 1/3\,(SBP - DBP)$$

where DBP indicates diastolic blood pressure and SBP indicates systolic blood pressure.

Vital Signs

After obtaining a general impression of the child's circulatory status, it is appropriate to obtain vital signs. The position of a child's heart in the thoracic cage is identical to that of an adult. When you are auscultating the pulse rate of an infant, it may be useful to place the stethoscope over the second intercostal space at the midclavicular line. For older children, auscultation may be easiest over the fourth intercostal space at the midclavicular line. Cardiac sounds should be absent of rubs, murmurs, gallops, or secondary sounds. Keep in mind that heart sounds heard over the apex of the heart represent precordial activity and not necessarily a perfusing pulse.

Again, remember to assess the patient and not the numbers because an increase in the heart rate may have an expected etiology, such as crying, fever, or anxiety. For every 1°C above 37°C, the child's heart rate may increase by as much as 20 beats/min. Tachycardia is also the first sign of hypovolemia. Assess the quality of the pulses to determine if they are absent, thready/weak, normal, full, or bounding.

Doppler ultrasound may be useful if a pulse cannot be palpated; however, if the pulse cannot be palpated, cardiac activity and/or perfusion is considered inadequate. In addition to assessing the presence and quality of pulses, compare the pulses bilaterally and in the upper and lower limbs. In an older child (> 1 year), the pulse rate should be assessed in the carotid, radial, femoral, and dorsalis pedis arteries.

To assess a pulse in an infant younger than 1 year, use the brachial and femoral arteries. Palpating carotid pulses in infants is difficult because of their thick, short necks. The carotid pulse is only palpated by hyperextending the neck.

Stroke Volume

Typically, the younger the child is, the higher the pulse rate. Stroke volume is smaller in children than in adults. An increase in heart rate is the chief compensatory mechanism in children to increase end-organ perfusion and to maintain blood pressure. It is logical that bradycardia in children results in hypoperfusion and hypotension.

Skin Condition

An assessment of the child's skin is a good indicator of circulation and whether hypoperfusion exists. Early compensatory mechanisms shunt blood from the skin to the vital organs. Skin that is well perfused should be warm, dry, and the appropriate color for race. As a child becomes hypoperfused, the skin becomes cool, pale, mottled, or cyanotic. As perfusion deteriorates, the child's hands and feet are typically affected first. Clammy or diaphoretic skin may also be present in the child with poor perfusion. Capillary refill is also an excellent indicator of peripheral perfusion. Normal capillary refill in pediatric patients is usually immediate; however, up to 3 seconds is considered an acceptable limit. Capillary refill of greater than 4 to 5 seconds represents a significant delay. Keep in mind that ambient temperatures may affect capillary refill time, as seen with hypothermia.

Blood Pressure

After the immediate assessment of the child's pulse, level of consciousness, and skin, more specific values may be obtained, such as blood pressure. Children, at least initially, compensate well during hypoperfusion. They respond by releasing catecholamines that cause vasoconstriction, an increased pulse rate, and increased contractility. In addition to tachycardia, a normal blood pressure may be maintained until compensatory mechanisms are depleted. For example, in hypovolemia, a child can lose up to 25% to 30% of total volume before hypotension is noted.

A common problem when measuring blood pressure in a child is ill-fitting cuffs. A cuff that is too large and exceeds two thirds the length of the child's arm will give a reading that is falsely low; a cuff that is too small or is less than half the length of the upper arm will give a falsely elevated reading. Often, providers choose to measure blood pressure in a lower extremity, because it is well tolerated by children. Blood pressure values obtained from the lower extremities have been shown clinically to correlate well with values obtained from upper extremities; however, it is advised that both upper and lower extremity blood pressure values be documented to ensure correlation. Always consider the child's normal range and clinical condition. Pain, fear, and anxiety often increase a child's blood pressure.

For some disease processes, such as traumatic brain injury, the MAP may be a more important measurement in guiding treatment than blood pressure. The MAP may be a better measure of end-organ perfusion and may be measured by either an arterial line or a noninvasive blood pressure monitor.

Fluid Volume and Access

Understanding circulating blood volumes for children is useful for quantifying volume loss and calculating fluid replacements. Estimate 80 mL/kg of body weight for a child's circulating volume. Any trauma or medical condition that causes fluid losses, such as blood, emesis, urine, or insensible losses (those that cannot be quantified), requires IV access for volume replacement. IV access can be obtained via a peripheral or central route. The use of intraosseous (IO) access has become common in infants or children requiring urgent interventions with fluids, blood, or medications, in whom rapid venous access cannot be obtained. The most common site for IO insertion is the anterior tibia **Figure 23-10**. Alternative

sites include the distal femur, medial malleolus, and anterior superior iliac spine. IO insertion is recommended over a venous cutdown because success and procedure time are superior; venous cutdown was used in the past, but is no longer practiced given the advancements in IO devices. There are contraindications to this procedure, which include, but are not limited to, osteogenesis imperfecta (brittle bone disease or Lobstein's syndrome), osteopetrosis, and fractures of the ipsilateral (same side) extremity.

Additional IO devices include the EZ-IO and the Bone Injection Gun (BIG). The EZ-IO features a hand-held battery-powered driver, to which a special IO needle is attached **Figure 23-11**. This device is used to insert an IO needle into the proximal tibia of adults and children when IV access is difficult or impossible to obtain. The battery-powered driver of the EZ-IO is universal, but different sizes of needles are available for adults and children.

The BIG is a spring-loaded device that is used to insert an IO needle into the proximal tibia of adult and pediatric patients. It comes in an adult and a pediatric size **Figure 23-12**, although both versions offer the same operational features.

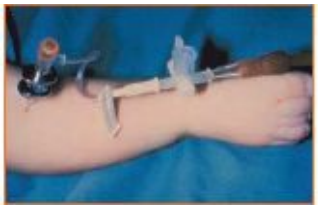

Figure 23-10 Standard pediatric intraosseous needle.

Figure 23-11 EZ-IO intraosseous infusion system.

Figure 23-12 Pediatric Bone Injection Gun.

For any sign of hypovolemia, a 20-mL/kg bolus of normal saline should be given, and repeated as necessary, up to 60 mL/kg during transport. If, during transport, a 60-mL/kg bolus fails to improve the patient's physiologic status, blood replacement and or inotropes should be considered depending on the underlying condition. Blood replacement is calculated as 10 mL/kg of body weight.

Urine output can be an objective guide in measuring pediatric circulatory status and the effectiveness

of volume replacement. Signs of poor kidney perfusion include a low urine output. In children wearing diapers, this means fewer than six wet diapers in a 24-hour period. In children with a urine catheter in place, this means less than 0.5 to 1 mL/kg/h. An adequate urine output is typically 1 to 2 mL/kg/h.

Neurologic Assessment

During the primary assessment, evaluate the child's general appearance. How is this child responding to the environment—is the child responsive or unresponsive, awake or lethargic? Assessment of the fontanelles is important. A sunken fontanelle may indicate dehydration, and a tense or bulging fontanelle may indicate increased intracranial pressure (as the intracranial pressure rises above normal limits, the fontanelle allows for expansion).

The Glasgow Coma Scale (GCS) is the most sensitive indicator of mental status. The score can be used to communicate the child's status to other caregivers and to guide treatment. For example, a child with a GCS of 8 or less from a head injury requires assisted ventilations. The GCS is modified when examining infants and children **Table 23-6**.

Another important component of the neurologic assessment is checking the child's pupil size and response to light. As with adults, very constricted or pinpoint pupils are consistent with narcotic overdose. A single dilated pupil is consistent with brain injury.

A good indicator of motor function is muscle tone. Children normally have flexed elbows and knees. Completely flaccid extremities are an abnormal finding. Another measure of motor function is the presence of normal reflexes, such as the **Moro reflex**, which occurs when the infant jumps or is startled in response to a loud noise. The **stepping reflex** is when an infant is held up in the air and moves his or her legs up and down as if marching. Finally, the **Babinski reflex** is different in infants and adults. In adults, the toes in a normal Babinski reflex point downward; flaring toes indicate brain injury. In children who are not yet walking, a normal Babinski reflex is just the opposite; flaring toes are normal and toes pointing downward indicate brain injury.

In a child with a decreased level of consciousness, the presence of protective reflexes can determine whether a child needs endotracheal intubation for airway protection. Protective reflexes are lost in ascending order—the swallow reflex, the cough reflex, the gag reflex, and, finally, the corneal reflexes. The cough and gag reflexes can be assessed using a suction catheter to elicit a response.

Renal Assessment

Assessment of the renal system typically begins with evaluation of hydration status. This assessment is often initially made by history. Parents or caregivers can offer information about the amount of intake delivered and information about the number of wet diapers, voids, stools, and frequency of emesis.

Physical assessment of the child may include assessment of the fontanelles (if the patient is an infant), assessment of skin turgor, the presence or absence of tears when crying, and sunken eyes. The measurement of wet diapers may be somewhat subjective because parents may change diapers that are not completely saturated. A good question to ask caregivers is whether the diapers are *different* than they normally are. A more objective measurement is weighing diapers, because 1 mL of urine equals 1 g of weight. Daily weights are also an objective measurement because they may be an early indication of fluid loss. Acute weight loss may be an early indication of fluid loss.

TABLE 23-6 Glasgow Coma Scale

Activity	Score	Description	Score	Description
Eye opening	4	Opens spontaneously	4	Opens spontaneously
	3	Opens to speech or sound	3	Opens to speech
	2	Opens to painful stimuli	2	Opens to pain
	1	No response	1	No response
Verbal	5	Coos, babbles	5	Oriented to person, place, and time
	4	Irritable cry	4	Confused
	3	Cries to pain	3	Inappropriate words
	2	Moans to pain	2	Incomprehensible words/sounds
	1	No response	1	No response
Motor	6	Normal spontaneous movement	6	Obeys verbal commands
	5	Withdraws to touch	5	Localizes pain
	4	Withdraws to pain	4	Withdraws to pain/nonpurposeful movement
	3	Abnormal flexion (decorticate)	3	Abnormal flexion (decorticate)
	2	Abnormal extension (decerebrate)	2	Abnormal extension (decerebrate)
	1	No response (flaccid)	1	No response (flaccid)

Fluid overload is much less common in children, although it may occur in patients with congenital heart defects or renal insufficiency. Signs of fluid overload in children may be vague and may initially have a more subtle presentation. For example, in pulmonary edema, hypoxia and grunting are two early signs. Other early signs, such as tachypnea, a cough, and increased oxygen needs, also may be discounted as other etiologies because of their vague presentation. Later signs are more obvious as they may include rales and pink frothy sputum associated with pulmonary edema. Pulmonary edema is later confirmed by a chest x-ray. Additional signs of fluid overload include weight gain, edema, and history of intake greater than output.

Exposure Considerations

Temperature regulation needs attention in all patients, especially in children. Environmental concerns regarding hypothermia or hyperthermia need to be addressed. Because of an infant's physiologic differences and inability to shiver, resuscitation of infants and children must include measures to maintain body temperature, being careful not to overwarm the patient.

Hyperthermia is related to increased oxygen consumption. Children who may be at risk for hyperthermia include those left in cars for extended periods of time, athletes, and those who are intoxicated with drugs and alcohol, which impair heat dissipation.

Heat loss can be minimized by replacing blankets on areas of the child that have been already assessed and by increasing the temperature in the transport vehicle. Be sure to leave heating/cooling measures in place in transport units while the patient is being prepared for transport.

Transport Considerations

Dedicated pediatric specialty teams are ideal for transporting the ill or injured child, but they may not be available or fiscally feasible. Essential knowledge includes understanding differences in pediatric physical assessment, airway management, ventilator management, vascular access, fluid and medication administration, and specific illnesses and injuries that are common in children. Essential skills include pediatric hemodynamic monitoring, intracranial pressure monitoring, mechanical ventilation, and the use of thermoregulation devices. It is helpful to practice new skills using the equipment you will find on the vehicle in which you will be working. In addition, although didactic education is required, it cannot substitute for hands-on pediatric experience including, but not limited to, routine pediatric care, critical pediatric care, and experience with pediatric intubations. Professionals who anticipate caring for critically ill or injured children should consider partnering with professional colleagues who care for

pediatric patients on a regular basis.

Equipment

It is essential to ensure that appropriate equipment is available when transporting pediatric patients. ECG monitoring with SpO_2 and $ETCO_2$ is the standard of care for all intubated pediatric patients. It is important to carry various sizes of ECG leads, pulse oximetry probes, and blood pressure cuffs to ensure accurate vital sign values. A transport ultrasound device may be helpful in verifying the presence of peripheral pulses in poorly perfused children **Figure 23-13**. The transport environment should also be *latex free*, including barriers such as gloves and monitoring and resuscitation equipment. Allergic reactions to latex are extremely dangerous, and it is especially important to remember the extremely high likelihood of latex allergies among special needs children. It is estimated that 73% of pediatric spina bifida patients have a latex sensitivity or a known history of an allergic reaction to latex, so the CCTP must be aware of this issue and make sure that non-latex equipment is available to care for this special population during the transport.

IV fluid administration in pediatric patients should be done with an infusion pump that delivers medication infusions calibrated to at least one decimal place. Fluid boluses and medications for resuscitation should be delivered with a syringe and stopcock technique. Fluid and medication formulas should be standardized and calculated prior to transport. It is also important to carry equipment for thermoregulation, such as a disposable gel-heated mattress or an incubator. Finally, know the capabilities of the transport ventilator for your service. Common requirements for ventilating children include pressure modes of ventilation, capacity for pediatric ventilator settings, and pediatric circuits.

All equipment and supplies in the vehicle should be secured prior to transport to reduce the possibility of causing injury from flying debris in the event of a crash.

Interfacility Considerations

When you are conducting an interfacility transport, there are several important pieces of information that will help to determine the mode of transport, the team configuration, and any specialized equipment that is needed. This information includes the age and weight of the patient, diagnosis, reason for transfer, physical exam findings, lab and other diagnostic tests results, IV access, and consent. It is helpful to develop a standardized intake form that will help the transfer team ask questions to determine the need for airway management or other procedures to stabilize the patient for transport. For example, for patients with an altered mental status, ask the sending facility for the patient's GCS score and the presence of a cough and gag reflex to determine the need for emergent airway management. Any treatment recommendations must be recorded on the transfer documentation.

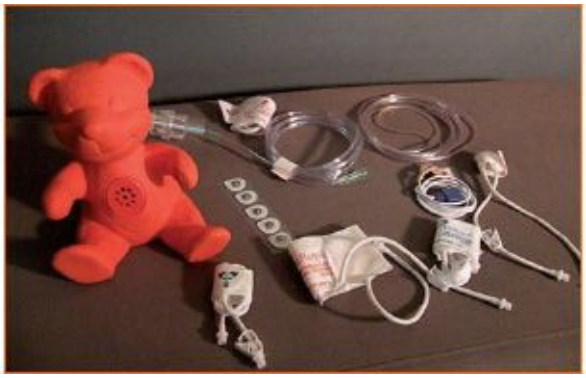

Figure 23-13 Appropriately sized equipment should always be accessible for transport of pediatric patients.

Once the CCTPs arrive at the sending facility, it is important to assess the patient immediately to determine the need for additional procedures prior to preparing the patient for transfer. Although it is important to ensure that all lines and tubes have been secured and to double-check all fluid and medication rates, a standardized team approach to the patient care hand-off is best. Using communication tools such as SBAR, which stands for situation, background, assessment, and recommendation, helps to standardize the exchange of information so that each provider has an expectation for what is to be discussed, even if the transport is time critical. This method can also be used to frame the conversation with other providers.

Baseline vital signs should be obtained, including temperature measurement and a plan to maintain body temperature. Trauma patients should have their immobilization status evaluated and enhanced as necessary until the cervical spine has been radiographically and clinically cleared at the receiving trauma center.

The sending facility must prepare copies of the medical records and films for transfer. Most facilities will not accept reports of radiographs because the films will need to be examined by a radiologist at the receiving facility.

Briefing for a contingency plan for resuscitation of the child if there is deterioration in the child's condition during transport is also important. The contingency plan may be covered in transport protocols or standing orders or may require consultation with a physician at the receiving hospital in the event of a specialized condition. For children with special needs, ask the parent or receiving physician for an explanation of the disease and any required modifications of resuscitation or transport procedures.

As in all transports, it is important to have a cohesive approach to transport. The driver should maintain a constant awareness of location, nearby hospitals, and, in the case of flight transport, potential landing zones in case of a sudden change in status of the patient or if weather issues prevent the completion of the transport.

Mode of Transport

To ensure the safe transport of pediatric patients, it is important to remember that children must be adequately restrained in the event of a motor vehicle (or flight) crash. Particular attention must be paid to the positioning and placement of child passenger safety restraints. Methods for restraining infants and young children include an isolette or similar device to maintain body temperature with attached straps, a child safety seat or a similar device that can be secured to the ambulance stretcher, and a pediatric immobilization device or backboard, although pediatric immobilizers are not approved by the Federal Aviation Administration. For children older than 4 years, the standard stretcher straps may be used. Generally, if the child requires resuscitation, he or she should be positioned supine and secured to the stretcher. If there is no likely threat to physiologic stability, the child should be transported in an upright, secured position **Figure 23-14**.

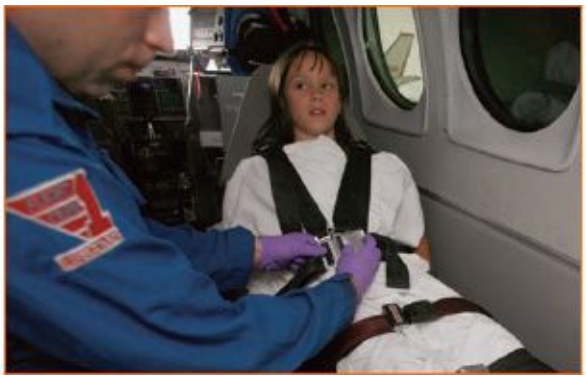

Figure 23-14 Transport the child in an upright, secured position if there is no likely threat to physiologic

stability.

All health care providers should be secured in the vehicle, and protective gear that is appropriate to the transport environment should be employed. For safety reasons, it is best to minimize the use of lights and sirens during transport. It is also best to perform as many anticipated interventions as possible that are needed to stabilize the patient prior to initiating the transport.

In patients with diseases or conditions that may be worsened by the effects of altitude, air medical transport may be limited to a specific altitude or may be contraindicated **Table 23-7**. Also, during air medical transport, always remember to take the following precautions to address the potential effects of altitude:

- Assess the patient for evidence of a pneumothorax and decompress any pneumothorax greater than 15% prior to the flight.
- Always vent gastric tubes during the flight and never clamp gastric tubes or indwelling urinary catheters when traveling above 8,000′.
- Be sure to monitor endotracheal (ET) cuff pressures with a pressure gauge and, if needed, fill the ET cuff with fluid to reduce the risk of cuff rupture or tracheal necrosis from increased pressure.
- Be sure to vent the mattress cover in an incubator to avoid an unexpected rupture.

Hypoxia worsens with higher altitudes and may require the administration of oxygen. It is good practice to give supplemental oxygen to all children being transported. In general, oxygen saturation levels should be greater than 90%. Exceptions include pediatric patients with cyanotic congenital heart disease and children with specific target saturations. Be prepared to provide assisted ventilations to children who require high doses of supplemental oxygen prior to flight.

TABLE 23-7 Altitude Restrictions

Altitude	Restriction
Sea level	Chest tube removal within 72 h Venous or arterial grafting performed less than 21 d ago Ventriculography or pneumoencephalography Decompression sickness Air emboli HAPE HACE
2,000″	Gas inclusion in body cavities Untreated perforated injuries of the eye Severe cardiac disease with cyanosis Retinal injuries Congestive heart failure Increased intracranial pressure or skull fracture Sickle cell crisis Ventricular arrhythmias GI disturbances
	Any two of the following:

4,000″	Clinical cyanosis Cor pulmonale Respiratory acidosis Anemia (acute or chronic) Sickle cell disease RBC < 25% Acute head injuries CVA Loss of consciousness Coma Severe cardiac disease with cyanosis Recent eye surgery Severe pulmonary emphysema
6,000″	Myocardial infarction Respiratory disease with increased vital capacity Space-occupying lesions of the lung Angina pectoris Sickle cell disease Alveolar block with cyanosis Clinical cyanosis Cor pulmonale Respiratory acidosis
8,000″	Marked ventilatory restriction from pregnancy, pleural effusion, or pleural fibrosis Alveolar exchange difficulties
10,000″	Any symptomatic or suspected cardiorespiratory disease

Abbreviations: CVA, cerebrovascular accident; GI, gastrointestinal; HACE, high-altitude cerebral edema; HAPE, high-altitude pulmonary edema; RBCs, red blood cells.

As discussed in Chapter 4, humidity also decreases with altitude. It is ideal to humidify supplemental oxygen and moisturize the eyes if the patient is unresponsive.

The condition of a patient with an air-filled cavity will worsen in higher altitudes because the cavity will expand. Examples include eye injuries with increased intraocular pressure and gastrointestinal conditions with a risk of intestinal perforation. Patients with abnormal arterial oxygen levels, such as children with cyanotic heart disease or chronic lung disease, are also adversely affected by altitude changes. CCTPs should be aware of local, regional, and national protocols and recommendations that address these issues.

Other factors to consider are related to additional stressors of flight, including barometric pressure changes, dehydration, noise, vibration, gravitational changes, cold, and tissue third spacing. Several measures can be taken to reduce these additional stressors. First, to help prevent ear pain from changes in barometric pressure, an alert infant or toddler can be given a pacifier to suck on during flight. To reduce the risk of dehydration, all children who are restricted to nothing by mouth should be started on maintenance IV fluids. To help decrease the stress of the noise and vibration of the aircraft, ear protection should be used with all patients. Forward or rear-facing positioning should be discussed prior to transport to minimize gravitational forces on landing; this is especially critical for children with closed

head injuries.

Children are at increased risk of hypothermia with air transport, so body temperature should be monitored during transport and warming measures should be initiated with all patients.

Accompanying Parents and Caregivers

The decision to transport parents or caregivers along with a child should be discussed, and guidelines should be put in place long before a child needs transport. In general, the presence of a parent or caregiver decreases a child's anxiety and emotional distress, which may also have a positive physiologic effect. For example, if a child in respiratory distress becomes more anxious and upset because of separation from a caregiver, the child's respiratory effort may increase and his or her physiologic status may deteriorate. The safe transport of the child has priority, however, and the situation must be assessed for the caregivers' comfort level and potential for disruption of care during transport. Consideration must be given to the seating of the parent or caregiver, and guidelines should be discussed with the caregiver prior to transport, particularly the need to remain seated and restrained in a vehicle restraint. No child should ever be held on a caregiver's lap for any reason while a vehicle is in motion.

Airway Management Devices

There are several oxygen delivery adjuncts used with pediatric patients in the critical care environment. These include devices commonly used in the prehospital environment, such as the nasal cannula, simple mask, partial rebreathing mask, non-rebreathing mask, Venturi mask, oxygen tent, and bag-mask device. However, flow-inflating bags, self-inflating bags, and oxygen hoods are not as common in the prehospital environment and will be discussed here for that reason. Shortened tracheal tubes and blade options for children will also be discussed.

Figure 23-15 A flow-inflating bag.

Figure 23-16 A self-inflating bag.

The American Academy of Pediatrics (AAP), via their Pediatric Advanced Life Support course, recommends that self-inflating bags be used for resuscitation. Most of this discussion will then involve the self-inflating bag; however, a brief review of flow-inflating bags will be discussed.

Flow-Inflating Bag

Flow-inflating bags have been routinely used for anesthesia. Features of flow-inflating bags are that they require an outside gas source to inflate and they have a pressure gauge port, a flow inlet dial, and an overflow port **Figure 23-15**. The volume of flow-inflating bags is 500 mL for infants and 600 to 1,000 mL for children.

Self-Inflating Bag

Safe and effective ventilations are possible with self-inflating bags **Figure 23-16**. However, they require more experience and more manipulation of the equipment to achieve adequate ventilations. For this reason, they are not recommended for resuscitation of infants and children outside of an environment where health care professionals have not received extensive training. Continued discussions will now focus on self-inflating resuscitation bags.

Figure 23-17 An oxygen hood.

Self-inflating bags come in different sizes to accommodate the various pediatric populations. A neonatal ventilation bag has a volume of 250 mL. Caution is advised because this small tidal volume may be inadequate for full-term newborns and infants. CCTPs may choose to use a pediatric ventilation bag that has a minimum volume of 450 to 500 mL and a maximum volume of 750 mL.

Features of self-inflating bags are that they do not require a compressed gas source, free flow may be delivered, and they have a pressure release valve (pop-off valve). The pop-off valve is a safety feature that releases when pressures exceed 40 cm H_2O. This helps to prevent excessive airway pressures while bagging children and minimizes the complication of a pneumothorax.

Oxygen Hood

An oxygen hood is an oxygen delivery system that is used in infant populations **Figure 23-17**. Children older than approximately 1 year are physically too big to fit in the oxygen hood. The hood allows for oxygen concentrations of 80% to 90% as well as humidified and warmed air. Oxygen delivery is reliable and allows for easy access of the child's body.

Shortened Tracheal Tube

A shortened tracheal tube may be used as an alternative nasal tube and may offer an advantage in that it is more rigid and less likely to be compressed; however, this rigidity may also be responsible for more

tissue trauma with insertion. Accidental advancement of the shortened tracheal tube is prevented by the 15-mm adapter cap at the end of the tracheal tube.

Blade Options

As you know from Chapter 6, the laryngeal blade is available in two shapes. The Miller blade is a straight blade and usually the choice for infants and younger children. A straight blade is preferred in these children because the epiglottis is cephalad in shape and the glottic opening is more anterior. Therefore, the straight blade allows better visualization for these anatomic considerations.

TABLE 23-8 Pediatric Blade Sizes

Age	Blade Size No.
Premature neonate	0
Term neonate	1
≤ 8 y	3
> 8 y	4

The MacIntyre blade is curved in shape and fits into the vallecula. This blade may be the choice for older children and adolescents because it better displaces the relatively large tongue, thereby allowing better visualization of the glottic opening.

Actual blade selection size is dependent on the child's size, as opposed to age. However, **Table 23-8** may guide the health care professional in size selection.

Respiratory Conditions

Respiratory illness is a common reason for pediatric transport. Respiratory compromise or failure can be caused by any airway, pulmonary, or neuromuscular disease that interferes with oxygen and carbon dioxide exchange. Four types of respiratory conditions that lead to respiratory failure include the upper airway conditions, lower airway conditions, parenchymal diseases, and abnormal control of ventilation, including neurologic causes.

Upper airway obstructions are most commonly caused by a foreign body aspiration or infection characterized primarily by inspiratory stridor. Partially obstructed airways cause turbulent airflow. The obstruction can be above or can include the glottis, pharynx, and trachea. The child's voice will be muffled or hoarse. If the obstruction is subglottic or subtracheal, stridor will be present but the child's voice will sound normal.

Lower airway disease may include the trachea and mainstem bronchi as well as the peripheral airways. Lower airway aspiration most commonly occurs in children younger than 3 years. A diagnosis of lower airway disease is often made after a day of aspiration and may range in presentation from mild coughing and wheezing to severe respiratory distress.

Peripheral airway diseases that are common indicators for transport include asthma, bronchiolitis, and bronchopulmonary dysplasia.

Respiratory distress is characterized by a noted increase in the work of breathing. The effort to breathe is labored and is recognized by an increased rate (tachypnea) and possibly depth (hyperpnea) of breathing. Nasal flaring, retractions, and accessory muscle use are usually noted. Although the amount of effort to breathe is dramatically increased, the child may still have adequate gas exchange of oxygen and

carbon dioxide. In addition, this change in the respiratory pattern may be for compensatory reasons. For example, in diabetic ketoacidosis, an increased respiratory rate and depth is necessary to exhale excessive amounts of CO_2.

Respiratory failure is characterized as inadequate oxygen or the inadequate exchange of oxygen and carbon dioxide. There currently are no strict guidelines for defining respiratory failure because it depends on the child's baseline respiratory function. A need for intervention to prevent respiratory or cardiac collapse is paramount regarding this patient population. If there are any known indications in this patient prior to departing the referring facility or if there are worsening signs and symptoms while en route, the CCTP should take all measures (per protocol) to maintain and preserve a patent airway and hemodynamic status. It is possible for a patient to experience respiratory failure without signs of respiratory distress. For example, a child who is unconscious and apneic after a traumatic head injury will not demonstrate increased work of breathing but rather inadequate or absent breathing.

Croup

Croup (laryngotracheobronchitis) is a common viral infection of the upper airway that affects the larynx but may also extend into the trachea and bronchi. Symptoms include a low-grade fever and hoarse "barking seal" cough that often is worse at night. Croup is most common in children younger than 3 years. In these children, the diameter of the cricoid area is smallest, so swelling may cause severe symptoms. Children most commonly present with mild distress but severe respiratory effects have been reported. The CCTP should be familiar with the hallmark sign of croup being the harsh "barking seal" cough.

Management of croup depends on the severity of the child's symptoms. A child in mild distress may be best managed by maintaining a calm environment. Humidified oxygen is the initial therapy. Allow the parents to sit next to the child to hold the humidified oxygen to the child's face using a mask or blow-by method. If airway obstruction (stridor) is more severe, racemic epinephrine (2.25% with 2.0 to 3.5 mL of normal saline) via a nebulizer usually improves airflow. It may be administered in the following dosages: 0.25 mL (age < 6 months), 0.5 mL (child), or 0.75 mL (adolescent). Racemic epinephrine is subtly different in chemical makeup from epinephrine, and it stimulates both alpha- and beta-adrenergic receptors, with a preference for beta-2-adrenergic receptors that causes bronchodilation. The vasoconstrictive effects of racemic epinephrine are temporary and remain only until the drug is metabolized, which should be considered with long transport times. Once the drug is metabolized, the swelling ("rebound worsening") may return to the same level as before or may worsen. In rare circumstances in which the obstruction is so severe that the child is no longer able to protect his or her own airway, oral intubation may be considered. When you suspect that the airway is edematous, the size of the ET tube chosen may be ½ to 1 size smaller than the tube typically used.

Signs and Symptoms

Croup

- Low-grade fever
- Hoarse, "barking seal" cough
- Mild distress
- Stridor

Differential Diagnosis

Croup

- Acute laryngeal fracture
- Angioneurotic edema
- Arnold-Chiari deformity
- Bacterial tracheitis
- Burns or thermal injury
- Dandy-Walker syndrome
- Diphtheria
- Epiglottitis
- Extrinsic obstruction by a vascular ring
- Foreign body
- Laryngeal papillomatosis
- Laryngomalacia
- Neoplasm or hemangioma
- Peritonsillar abscess
- Retropharyngeal abscess
- Smoke inhalation
- Subglottic stenosis
- Viral croup
- Vocal cord paralysis

Transport Management

Croup

- Administer humidified oxygen using a mask or blow-by method.
- For more severe obstruction, administer racemic epinephrine via a nebulizer.
- If the child is no longer able to protect the airway, consider oral intubation.

Epiglottitis

Epiglottitis (or supraglottitis) is a bacterial infection caused by *Haemophilus influenzae* that most commonly occurs in children aged 3 to 5 years. Symptoms include rapid onset of fever, stridor, and pronounced signs of toxicity. The child may appear anxious, have a muffled voice, and be in a tripod position with pronounced drooling because of supraglottic swelling and excess secretions. Remember the four Ds for epiglottitis: dysphagia, dysphonia, drooling, and distress. Children with epiglottitis are at a tremendous risk of acute and complete airway obstruction. Invasive procedures should be kept at a minimum so as not to aggravate and increase the child's work of breathing.

Management of suspected epiglottitis in the prehospital setting is controversial because this condition can worsen rapidly, leading to a complete airway obstruction. The use of racemic epinephrine with this condition is contraindicated because it can cause additional swelling to the affected inflamed tissues. There is consensus that a calm environment should be maintained so as not to precipitate complete obstruction. There is also agreement that a definitive airway should be secured prior to transport. However, these children are often difficult to intubate and may require specialized teams or

specialized tools, such as retrograde intubation, direct bronchoscopy, or possibly tracheostomy if intubation is unsuccessful. Therefore, the decision to secure definitive airway management in the prehospital setting is multi-factorial, including EMS protocols, skill, and experience of the transport teams, and the time and distance of the transport.

Signs and Symptoms

Epiglottitis

- Rapid onset of fever
- Stridor
- Pronounced signs of toxicity
- Distress
- Muffled voice
- Drooling
- Tripod position

Differential Diagnosis

Ep glotttis

- Bacterial tracheitis
- Foreign body ingestion
- Mononucleosis
- Anaphylaxis
- Croup or laryngotracheobronchitis
- Pertussis
- Pharyngitis
- Pneumonia
- Peritonsillar abscess
- Retropharyngeal abscess
- Caustic ingestion

Transport Management

Ep glotttis

- Maintain a calm environment.
- Secure a definitive airway prior to transport.

Foreign Body Airway Obstruction

Foreign body airway obstruction remains a significant source of morbidity and mortality in pediatric populations. More than 9% of deaths in children younger than 5 years are related to foreign body

aspiration. The onset may be dramatic, with an immediate life threat or with varying severity.

Removal of the foreign body should only be attempted in the prehospital setting by following the guidelines set forth by the American Heart Association and AAP to include the use of chest thrusts in infants and the Heimlich maneuver in older children, leading to direct laryngoscopy with Magill forceps to remove the foreign body. This includes specific algorithms, including only using back blows on infants younger than 1 year **Figure 23-18**. Direct visualization of the trachea and removal of the foreign body should only be considered in the event of impending respiratory failure.

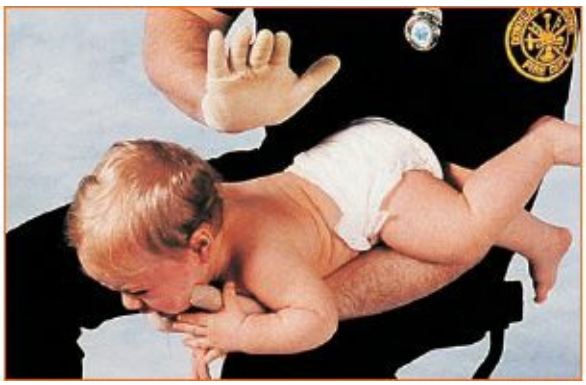

Figure 23-18 Back blows are only recommended on infants (children younger than 1 year).

Signs and Symptoms

Foreign Body Airway Obstruction

- Choking or gagging when the object is first inhaled or swallowed
- Sudden onset of coughing or cough that gets worse
- Stridor
- Wheezing
- Inability to speak
- Pain in the throat or chest
- Hoarse voice
- Blueness around the lips
- Not breathing
- Unconscious

Differential Diagnosis

Foreign Body Airway Obstruction

- Asthma
- Croup
- Myocardial infarction
- Anaphylaxis
- Acute epiglottitis

Transport Management

Foreign Body Airway Obstruction

- Removal of the foreign body is attempted only following AHA and AAP guidelines: chest thrusts in infants or Heimlich maneuver in older children.
- Direct laryngoscopy with Magill forceps.
- Back blows on infants younger than 1 year.
- Direct visualization of the trachea and removal of the foreign body only in the event of impending respiratory failure.

Inflammation

Generalized inflammation of the upper airway may be a result of inhalation burns or allergic and/or anaphylactic reactions. These airway insults are often precipitous in their presentation; early recognition and intervention are imperative to lessen morbidity and mortality. Therapeutic measures to treat all upper airway obstructions include the basics of a calm environment, supplemental humidified oxygen, providing a position of comfort, and preparing for deterioration of the airway status.

Asthma

Asthma, a chronic inflammatory disorder of the lower airways, results in significant inflammation, bronchospasm, and edema. Recurring episodes present with shortness of breath, wheezing, and chest tightness and occur more frequently at night or in the early morning. Increased hyperreactivity of airways may be noted with inflammation caused by a variety of stimuli, including allergens, exercise, emotions, infections, and cold air. The incidence of asthma in the pediatric population is 5% to 15%, making it a significant source of pediatric transports.

Reactive airway disease (RAD) is a term often used synonymously with asthma. Reactive airway disease occurs in children younger than 3 years; the conditions of 30% of children with RAD progress to asthma. Recall that all infants have highly reactive airways, making them more prone to responses from environmental allergens, viral respiratory diseases, and hereditary factors.

Parents of children who experience chronic exacerbations of asthma are usually successful at treating the symptoms or preventing rapid deterioration while at home. Typically, when CCTPs are needed to transport a child with an exacerbation of asthma, traditional treatments have failed and more advanced therapies are required. Obtain a history from the child and parents (caregivers) at the referring facility if they are available, and also obtain a detailed report from the transporting EMS crew (if available), the bedside nurse, and/or the referring physician. For example, if a child has a history of requiring numerous intubations for severe asthma, the child should receive all available treatment during rapid transport to the hospital.

Management of a child with an asthma exacerbation includes oxygen therapy because there is a significant ventilation/perfusion mismatch. A bronchodilator, most commonly a beta-2 agonist, is administered (albuterol, 2.5 to 5.0 mg via hand-held nebulizer). Beta-2 agonists work to relieve the bronchospasm by relaxing and dilating the bronchioles, allowing more unobstructed airflow. Corticosteroids are the cornerstone for anti-inflammatory therapy. They are used to manage both acute and chronic episodes of asthma. The effects of corticosteroids are often not seen for approximately 4 to 6 hours; therefore, they should be administered early in the course of an acute exacerbation of asthma. Corticosteroid use should not take the place of aggressive beta-2 agonist therapy, but should be

administered in conjunction with it.

Signs and Symptoms

Asthma

- Shortness of breath
- Wheezing
- Chest tightness

Differential Diagnosis

Asthma

- Airway obstruction with a foreign body
- Bronchitis
- Pneumonia/bronchiolitis
- Cystic fibrosis
- Bronchopulmonary dysplasia (in premature infants)
- Dysmotile cilia syndrome
- Alpha-1 antitrypsin deficiency
- Immunodeficiencies (National Heart, Lung, and Blood Institute 1997; National Heart, Lung, and Blood Institute 2003)

Transport Management

Asthma

- Administer a bronchodilator, commonly a beta-2 agonist.
- Administer corticosteroids early in an acute exacerbation.

Bronchiolitis

Bronchiolitis is a viral infection most commonly caused by respiratory syncytial virus (RSV) but may also be caused by a host of other viruses. Bronchiolitis primarily affects infants and young children. Infants and young children are at risk for inflammation, edema, and bronchospasms because of immature lower airways. The infection then causes airway obstruction and wheezing. The presentation for children may vary; some may merely have mild wheezing or they may have severe respiratory distress recognized by tachypnea, nasal flaring, and retractions.

Treatments for bronchiolitis are similar to asthma in that both require the administration of supplemental oxygen and IV fluids (avoiding overhydration, which may lead to pulmonary edema), but bronchodilators should be used with caution. Patients with bronchiolitis require the administration of ribavirin (child), an antiviral drug that has been shown to reduce the need for mechanical ventilation in children with lower respiratory tract infections caused by RSV. Diligent assessment must be made regarding the child's work of breathing prior to transport and frequently en route. Infants and young

children have limited energy stores and may fatigue easily with labored breathing.

Signs and Symptoms

Bronchiolitis

- Mild wheezing
- Tachypnea
- Nasal flaring
- Retractions

Differential Diagnosis

Bronchiolitis

- Asthma
- Gastroesophageal reflux
- Foreign body aspiration
- Vascular ring
- Enlarged adenoids
- Congestive heart disease
- Chronic lung disease

Transport Management

Bronchiolitis

- Administer supplemental oxygen and fluids.
- Administer ribavirin.
- Assess the child's work of breathing before and frequently during transport.

Bronchopulmonary Dysplasia

Bronchopulmonary dysplasia (BPD) is a chronic lung disease that develops in preterm neonates who have been treated with oxygen and positive-pressure ventilation. Neonates at risk for developing BPD are those born before 30 weeks of gestation and those weighing less than 1,200 g at birth. This is a complicated disease in which the pathologic progression of the disease and the healing process are not well understood. The relevance of this topic to this chapter is that infants who survive BPD have pulmonary hypertension and abnormal pulmonary vascular development. Children with BPD are at tremendous risk for severe symptoms during lower respiratory tract infections, and they are at very high risk of pulmonary morbidity and mortality during the first 2 years of life.

Management of the child with BPD can be challenging because the child is usually already receiving oxygen therapy and has a chronic underlying pulmonary dysfunction. Therapy often consists of bronchodilators, corticosteroids, and antibiotics. Dosage depends on the assertiveness of the referring facility. Children with BPD have decreased lung compliance and may require higher peak inspiratory

pressures if oxygenated via bag-mask device or mechanical ventilator. The specifics of ventilation for pediatric patients are discussed next.

Signs and Symptoms

Bronchopulmonary Dysplasia

- Pulmonary hypertension
- Abnormal pulmonary vascular development

Differential Diagnosis

Bronchopulmonary Dysplasia

- Aspiration pneumonia
- Bronchiectasis
- Bronchogenic cyst
- Congenital lobar emphysema
- Cystic fibrosis
- Emphysema
- Esophageal atresia/tracheoesophageal fistula
- Hyaline membrane disease
- Patent ductus arteriosus
- Neonatal pneumonia
- Viral pneumonia

Transport Management

Bronchopulmonary Dysplasia

- Administer bronchodilators, corticosteroids, and antibiotics.
- Oxygenate via bag-mask device or mechanical ventilator.

Mechanical Ventilation

Pressure-cycled ventilation evaluates the inspiratory tidal volume for assessing lung compliance. A target tidal volume of 7 to 10 mL/kg is recommended. Tidal volumes of less than 7 mL/kg may result in hypoventilation and atelectasis. Tidal volumes of greater than 10 mL/kg may result in overdistention of alveoli, compressing the pulmonary capillary bed and causing barotrauma.

Volume-cycled ventilation evaluates the peak inspiratory pressures (PIP) to assess lung compliance. The initial PIP setting should be the lowest possible value that results in adequate chest excursion. Most children should be able to be adequately ventilated with a PIP of 20 to 30 cm. Children with lung disease may require higher PIPs in the 30s. PIPs of greater than 40 cm increase the risk of barotrauma and spontaneous pneumothorax. Many pediatric critical care practitioners will consider an alternative type of mechanical ventilator if the patient requires a PIP of over 40 cm. **Table 23-9** lists age-appropriate

ventilator settings.

TABLE 23-9 Age-Appropriate Ventilator Settings

Setting	Infant	Child
FIO_2	0.21–1.0	0.21–1.0
I time, s	0.5–0.8	0.6–0.8
I:E ratio	1:1 to 1:2	1:1 to 1:2
Tidal volume, mL/kg	7–10	7–10
PIP, cm	15–18	16–24
PEEP, cm	3–5	3–5
PS, cm	5–15	5–20
Rate, breaths/min	25–30 (neonates: 30–50)	20

Abbreviations: PEEP, positive end-expiratory pressure; PIP, peak inspiratory pressure; PS, pressure support.

Controversies

Practitioners accustomed to managing complex mechanical ventilation of adults will find much dissimilarity in the pediatric population. One important consideration is positioning. In adults, sideways rotation to elevate a difficult-to-ventilate lung often improves oxygenation. For reasons poorly understood, the opposite effect is often seen in children.

Physiologic PEEP for infants and children is 3 to 5 cm. Higher levels of PEEP may help with alveolar recruitment and may improve oxygenation. Higher levels of PEEP also increase intrathoracic pressure, resulting in decreased venous return to the heart. The decreased venous return may cause hypotension in children with decreased intravascular volume status or cardiovascular instability. PEEP levels of 8 to 12 cm increase the risk of barotraumas and spontaneous pneumothorax. Many pediatric critical care practitioners will consider an alternative type of mechanical ventilator if the patient requires PEEP values of greater than 12 cm.

The use of pressure support is most helpful in patients who ventilate best with a spontaneous respiratory pattern and in patients who are being weaned from mechanical ventilation. Increasing pressure support increases the tidal volume of the patient's spontaneous breaths. This may be helpful in patients who have an adequate spontaneous rate but an inadequate tidal volume. A patient with status asthmaticus, for example, generally is more effectively ventilated with a spontaneous pattern such as pressure support. The initial rate is set based on age-appropriate values and then titrated based on blood-gas findings and exhaled carbon dioxide values.

In infants and children with severe lung disease, an alternative type of ventilator is an oscillator. An oscillator delivers smaller tidal volumes with very rapid respiratory rates (60 to 1,000 breaths/min) that reduce the risk of barotrauma and pneumothorax. The availability of an oscillator may be the reason for

transporting the child to another facility.

Ventilator Setting Changes

Another difference in ventilator management in infants and children is the manipulation of ventilator settings to improve oxygenation or ventilation. In both pressure- and volume-cycled ventilation, increasing FIO_2, inspiratory time, and PEEP will all improve oxygenation. Increasing PIP and tidal volume within the target range will also improve oxygenation. The strategies to improve ventilation depend on the patient's initial rate. In patients with a rate of 20 breaths/min or less, increasing the rate will improve ventilation and lower the carbon dioxide value. In patients with a higher respiratory rate, increasing the rate may actually worsen ventilation because the expiratory time will be decreased.

For example, consider a patient with an inspiratory time of 0.8 seconds and a rate of 12 breaths/min. The total time for each breath cycle is 5 seconds. This time frame is determined by dividing 60 seconds by the rate of 12. The expiratory time is calculated by subtracting the inspiratory time from the total time for each breath cycle. In this case, 5 seconds – 0.8 seconds = 4.2 seconds. If the rate is increased to 15, then the total time for the breath cycle is 4 seconds. This means that the expiratory time is now 3.2 seconds. This leaves the patient plenty of time for adequate expiration.

Now let's look at a patient with an inspiratory time of 0.8 seconds and a rate of 30 breaths/min. In this case, the total time for each breath cycle is 2 seconds. The expiratory time is 2 seconds – 0.8 seconds = 1.2 seconds. This gives the patient an I:E ratio of 1:2. If the rate is increased to 33, then the total time for the breath cycle is 1.8 seconds. This changes the expiratory time to 1 second, and the I:E ratio changes to 1:1.25. In the second case, increasing the respiratory rate would worsen ventilation because it decreases the time available for exhaling carbon dioxide. In this case, increasing PIP or tidal volume is a better strategy. The CCTP might also consider decreasing the rate or the inspiratory time to lengthen the time available for expiration. This strategy is particularly helpful in patients with bronchospasm, in whom elevated carbon dioxide levels might be a result of air trapping.

Pneumonia

Pneumonia is a type of parenchymal lung disease that occurs in the lung itself. Pneumonia in infants and young children may not be tolerated as well as in older children and adults. Transport treatment includes a vigilant assessment of work of breathing and hydration status. Antibiotic therapy is required and may have begun at the initial facility prior to transport.

Signs and Symptoms

Pneumonia

- Fever
- Grunting
- Cough
- Congestion
- Irritability
- Decreased feeding
- Shortness of breath
- Sweating
- Shaking and chills
- Pleurisy

- Headache
- Muscle pain
- Fatigue

Differential Diagnosis

Pneumonia

- Airway foreign body
- Asthma
- Bronchiolitis
- Bronchioectasis
- Bronchitis
- Cystic fibrosis
- Gastroesophageal reflux
- Congestive heart failure
- Human immunodeficiency virus
- Pertussis

Transport Management

Pneumonia

- Be vigilant in assessing the child's work of breathing and hydration status.

Acute Respiratory Distress Syndrome

The presentation of acute respiratory distress syndrome, also called adult respiratory distress syndrome, is similar in children and adults. Regardless of the underlying cause, the cardinal determinant of acute respiratory distress syndrome is intrapulmonary shunting that has minimal or no response to oxygen therapy. In addition to reversing the underlying etiology, management is primarily supportive, including mechanical ventilation with the use of judicious PEEP and cardiovascular support.

Signs and Symptoms

Acute Respiratory Distress Syndrome

- Shortness of breath
- Rapid breathing
- Cough
- Fever
- Low blood pressure
- Confusion
- Extreme tiredness

Differential Diagnosis

Acute Respiratory Distress Syndrome

- Goodpasture's syndrome
- Hypersensitivity pneumonitis
- Multisystem organ failure from sepsis
- Pneumonia
- Respiratory failure
- Bacterial sepsis
- Septic shock
- Hemorrhagic shock
- Toxic shock syndrome

Transport Management

Acute Respiratory Distress Syndrome

- Provide support, including mechanical ventilation.

Near–Sudden Infant Death Syndrome vs Sudden Infant Death Syndrome

A **near–SIDS** (near–sudden infant death syndrome) event or an apparent life-threatening event is an acute episode of apnea accompanied by choking or gagging, skin color change (erythema, pallor, or cyanosis), and a noted change in muscle tone (limp or increased muscle tone) by the observer of the event (eg, a parent or a caregiver). Recent studies have indicated that several etiologies associated with this diagnosis include, but are not limited to, gastroesophageal reflux disease, viral lower respiratory tract infections, seizures, sepsis, and meningitis. Even with a comprehensive hospital evaluation, the differential diagnosis may conclude to be idiopathic in nature, which should heighten the CCTP's need for a detailed report from the receiving hospital caregivers and/or the parent(s) present at the time of the event. Laboratory results (complete blood cell count, to include a urinalysis to rule out a urinary tract infection) and any related radiographic studies will also be needed to rule out pneumonia. In most cases, near-SIDS patients only require supportive care and immediate transport to a higher level of care for further studies (electroencephalography, polysomnography/sleep studies) or consultation (cardiologist for ECG or electrophysiology studies to rule out a long QT syndrome or preexcitation arrhythmias).

Sudden infant death syndrome is the sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history. The incidence of SIDS is highest during the first 4 months of life and becomes more uncommon after the age of 6 months. Based on the CDC and the National Center for Health Statistics reports, the incidence of SIDS has declined by 50% since 1983. This decrease in death rates has been contributed to the adoption of such campaigns as "Back to Bed" and "Back to Sleep," which instruct that to prevent SIDS, infants should be placed on their backs when being laid down to sleep. This approach is supported by many organizations, such as the American College of Pediatrics. Unfortunately, there are still 2,500 cases of SIDS each year in the United States, which are attributed to risk factors such as low-birth-weight infants, prematurity, gender (61% males), lack of prenatal care or nutrition, and exposure to secondhand smoke.

CCTPs must be aware of the impact of care, and must be sensitive when communicating with parents (caregivers) of infants who experienced SIDS. If the infant arrests while en route to the receiving facility, follow standing orders per your agency protocol or contact medical control to request orders to continue or discontinue resuscitative measures. In most cases, all necessary medical interventions will be initiated in this scenario, especially if one of the patient's parents is being transported with the patient.

If your patient dies during the transport, this parent may become the patient. The CCTPs may have to shift attention to the parent's reaction, which could include denial or anger. As in all situations, the crew's safety must be paramount regardless of the mode of transport. Parents having to deal with such a tragic event may require emotional support from the transport team members or the receiving hospital's support staff on arrival.

Signs and Symptoms

Near SIDS

- Choking or gagging
- Skin color change (erythema, pallor, or cyanosis)
- Noted change of muscle tone (limp or increased muscle tone)

Transport Management

Near-SIDS

- Provide supportive care.
- Transport immediately to a higher level of care.

Transport Management

SIDS

- Follow standing orders or contact medical control to request orders to continue or discontinue resuscitative measures.
- Care for the parent if a parent accompanies the child during transport.

Cardiac Conditions

Pediatric cardiac conditions that have an effect on the circulatory system are common indications for transport. Conditions range from those acquired, such as hypovolemia related to trauma, or congenital conditions, such as structural heart defects that are addressed later in this chapter. The CCTP will routinely be involved in four different classifications of heart disease or conditions, which include a known cardiac defect, an unknown or suspected cyanotic defect (based on patient presentation), a cardiac arrhythmia, and some form of shock (cardiogenic, septic, hemorrhagic, neurogenic, or anaphylactic).

Shock

As discussed in Chapter 9, shock may lead to rapid death or may be more progressive, as in the example

of multi-organ failure.

Shock is categorized as compensated or uncompensated. A child in early or even moderate stages of shock is able to maintain perfusion. Pediatric patients will maintain the compensatory mechanism until they suddenly crash without warning. Once the child's compensatory mechanisms are depleted, the blood pressure falls and the shock classification becomes uncompensated. At this stage, coronary and cerebral perfusions are grossly inadequate. Immediate interventions must be performed to mitigate dysfunction and prevent continued cellular death.

Signs and Symptoms

Shock

- Lightheadedness
- Tachycardia
- Decreasing blood pressure
- Confusion or loss of consciousness (coma)
- Chest pain
- Diarrhea
- Kidney failure
- Pale, clammy skin

Transport Management

Shock

- Assess airway, breathing, and circulation.
- Attempt to control obvious bleeding with direct pressure.
- Obtain a fingerstick blood glucose level to make sure hypoglycemia is not present.
- Administer oxygen.
- Administer IV medications per protocols.

Blood pressure is not the only reliable parameter on which the CCTP should base assessment findings. Other indicators, such as the child's level of consciousness, heart rate, skin temperature, respiratory rate and pattern, and urinary output, are also valid markers of hemodynamic status.

Hypovolemic Shock

Hypovolemic shock is the most common cause of shock in pediatric populations and is characterized by inadequate intravascular volume. Volume loss related to blood in children is most commonly associated with trauma. Fluids lost through vomiting and diarrhea are often underestimated; these losses are particularly significant in young children because they have minimal volume reserves. Another form of fluid loss is excessive urination, as is seen in children with diabetes. Finally, volume losses related to severe allergic reactions may be related to increased capillary permeability and fluid escaping into the interstitial spaces.

When a child's intravascular volume falls, there is inadequate venous return and decreased cardiac output. Cardiac output is a primary factor in end-organ perfusion. Inadequate volume may be related to a

deficit in the circulating fluid volume, resulting in not enough volume.

Hypovolemic shock is categorized into four stages. In most cases of stage 1 hypovolemic shock, the child appears asymptomatic. In stage II, the child attempts to accommodate for volume losses but compensatory mechanisms are being maximized. Blood pressure may still be compensated at this time, but the child is dangerously close to decompensating if interventions are not rapidly applied. In stage III, the child is no longer able to compensate and hypotension occurs. In addition to traditional signs of shock, the child demonstrates mental status changes and significantly decreased urine output. Many children now require blood transfusions for resuscitation to be successful. Finally, in stage IV, death is imminent if the volume loss is not eliminated and aggressive volume replacement, including crystalloids and blood products, is not initiated.

Regardless of the etiology of the volume loss, the principles of resuscitation are identical. Stop the source of volume loss: bleeding, vomiting and diarrhea, and vasodilation.

The next step is to replace the lost volume. Fluid resuscitation for children is 20 mL/kg of crystalloid solutions and 10 mL/kg of blood products. Fluid resuscitation for infants is 10 mL/kg. Fluid boluses of crystalloid solution may be repeated up to three times. If volume remains a deficit, blood products need to be administered. If a child has a cardiac history or concurrent cardiogenic shock, fluid boluses should be at 10 mL/kg. Ultimately, the child may receive up to three boluses of 20 mL/kg but in smaller increments. Successful resuscitation is measured by a return to a normal heart rate, respiratory rate, blood pressure, improved mental status, and improved skin condition.

Transport Management

Hypovolemic Shock

- Stop the source of volume loss (ie, bleeding, vomiting, diarrhea, and vasodilation).
- Replace the lost volume with fluid boluses and blood products.

Cardiogenic Shock

Some causes of cardiogenic shock include, but are not limited to, congenital cardiac defects, drug toxicity, metabolic causes, hypovolemia, myocarditis, and arrhythmias. Congenital defects are much more likely to be the cause of cardiogenic shock than other etiologies. The child with cardiogenic shock will present with traditional signs of shock, including tachycardia, tachypnea, hypoxia, mental status changes, and changes in skin condition. The child in cardiogenic shock often presents with pulmonary congestion resulting in rales heard on auscultation and jugular venous distention. A chest radiograph will also often reveal cardiomegaly.

Signs and Symptoms

Cardiogenic Shock

- Traditional signs of shock include tachycardia, tachypnea, hypoxia, mental status changes, and changes in skin condition
- Pulmonary congestion resulting in rales heard on auscultation
- Jugular venous distention

Transport Management

Cardiogenic Shock

- Resuscitate with fluids.
- Provide inotropic support, including dopamine, dobutamine, epinephrine, milrinone, and norepinephrine.
- Treat primary pathologies or impedances.
- Support cardiac function pharmacologically until myocardial function returns.

Treatment principles concentrate on improving cardiac function with fluid resuscitation and inotropic support to include dopamine, dobutamine, epinephrine, milrinone (dosages listed in Table 23-4), and norepinephrine (0.01 to 0.5 µg/kg/min [rarely used]). Primary pathologies or impedances are treated, and cardiac function may be supported pharmacologically until myocardial function returns.

Distributive Shock

The major treatment goals for distributive shock include stopping the vasodilation, returning volume to the intravascular space, and improving tissue perfusion. Types of distributive shock include neurogenic shock, anaphylactic shock, dissociative shock, and septic shock.

Neurogenic Shock

Treatment for neurogenic shock often necessitates vasoactive medications; however, volume replacement with crystalloids must occur first. The principle of "fill the tank first and then make the container smaller" is the cornerstone of treatment.

Transport Management

Neurogenic Shock

- Replace volume with crystalloids.
- Administer vasoactive medications.

Anaphylactic Shock

Children in anaphylactic shock often present with increased general body edema, hypotension, rash, urticaria, anxiety, and warm, flushed skin. Bronchospasm and laryngeal edema may also be life threatening.

Signs and Symptoms

Anaphylactic Shock

- General body edema
- Hypotension
- Rash
- Urticaria

- Anxiety
- Warm, flushed skin
- Bronchospasm
- Laryngeal edema

Transport Management

Anaphylactic Shock

- Remove the allergen.
- Replace volume.
- Administer epinephrine.

Management of anaphylactic shock involves removing the allergen, volume replacement, and epinephrine. Epinephrine dilates the bronchials; relaxes the smooth muscle, causing vasoconstriction; and decreases vascular permeability, thereby inhibiting more volume loss.

Dissociative Shock

Dissociative shock is a rare form of shock in the pediatric population and is often not included in traditional classifications of shock. In dissociative shock, the hemoglobin molecule is unable to give up oxygen to the tissues. Tissue perfusion is normal, however, and the release of oxygen is abnormal.

Some examples of dissociative shock are carbon monoxide poisoning, methemoglobinemia, dyshemoglobinemia, and cyanide poisoning. In each case, the agents causing the condition have a higher affinity to hemoglobin and oxygen is displaced.

Treatment is early recognition, removal of the cause, oxygen administration, and supportive therapy.

Transport Management

Dissociative Shock

- Remove the cause of shock.
- Administer oxygen.
- Provide supportive therapy.

Septic Shock

Another cause of abnormal distribution from massive vasodilation is related to septic shock. This form of shock is caused by an overwhelming release of proinflammatory mediators, which are initially released to help damaged cells repair. In sepsis, however, the response is so large that it causes more tissue and capillary injury. Management of septic shock includes treating the underlying infection and providing multisystem support—oxygen, volume replacement, and often vasoactive medications.

The treatment of septic shock in the pediatric population is focused on managing the elements of hypovolemia and cardiogenic shock. The inflammatory response in sepsis is a result of an infectious agent or its contained toxins. Most of these infectious agents are bacterial, but sepsis may also be caused by a virus, a fungus, or other microorganisms. The CCTP must be aware of the two phases of septic shock,

which consist of the warm phase and the cold phase. The patient in the warm phase presents with a period of vasodilation or the appearance of well-perfused organs. On assessment, the skin is warm and the pulses are bounding on palpation. The cardiac output is within normal limits or elevated, but kidney function is diminished, and metabolic acidosis and a change in mental status will be noted. If left untreated, the patient will present with the signs and symptoms of the cold phase of shock, which represents a continually failing cardiac output with decreased contractility, poor perfusion status, and extremities that are cool to the touch. The CCTP may use the following formula for calculating normal systolic blood pressure in this patient population in order to compare this with the patient's actual blood pressure:

$$\text{Systolic Pressure} = 80 + (\text{Age in Years} \times 2)$$

According to the 2007 update from the American College of Critical Care Medicine, the clinical diagnosis of septic shock is made in children who (1) have a suspected infection manifested by hypothermia or hyperthermia and (2) have clinical signs of inadequate tissue perfusion, including any of the following: decreased or altered mental status; prolonged capillary refill of greater than 2 seconds (cold shock), diminished pulses (cold shock), mottled cool extremities (cold shock), or flash capillary refill (warm shock); bounding peripheral pulses; wide pulse pressure (warm shock); or decreased urine output of less than 1 mL/kg/h. The goal of treatment by the CCTP as previously stated is to focus on the resolution of hypovolemia and to improve the perfusion status caused by cardiogenic shock. The resuscitation management of the septic pediatric patient is detailed in **Figure 23-19**, which depicts a time-sensitive algorithm. This algorithm focuses on the maintenance or restoration of the following: airway management to include oxygenation and ventilation support; circulatory/perfusion status to include blood pressure, vascular access, fluid volume resuscitation, and use of inotropic/vasoactive drug support; and the use of extracorporeal membrane oxygenation (ECMO) in refractory shock.

Signs and Symptoms

Septic Shock

Warm Phase

- Period of vasodilation or appearance of well-perfused organs
- Warm skin
- Bounding pulses on palpation
- Normal or elevated cardiac output
- Diminished kidney function
- Metabolic acidosis
- Changed mental status

Cold Phase

- Failing cardiac output with decreased contractility
- Poor perfusion
- Extremities cool to touch

Transport Management

Septic Shock

- Administer oxygen and provide ventilation support.
- Establish IV/IO access.
- Administer fluids.
- Use inotropic/vasoactive drug support.
- Use ECMO in refractory shock.

Cardiac Anomalies

Approximately 1 of every 100 infants has a congenital heart defect. Depending on the severity of the congenital heart disease, some patients may need immediate medical management or surgical intervention to repair the defect. Other patients may only need supportive care until later in life, when they are able to tolerate such surgical intervention.

The pediatric population most likely to have an undiagnosed cardiac anomaly is the neonate. Typically, older children have cardiac defects diagnosed and surgical repair or pharmacologic interventions initiated. Congenital cardiac anomalies may not be diagnosed until the neonate is 1 to 2 weeks of age because the ductus arteriosus remains patent and allows adequate mixing of oxygenated and deoxygenated blood. For a neonate who has a relatively small lesion and a more subtle presentation, diagnosis may not be made until 2 to 8 weeks of age when the characteristic murmur may be heard. The murmur may not be apparent until this age because of high pulmonary and arterial pressures present immediately after birth. In addition, the initial presentations of pallor, poor perfusion, lethargy, and cyanosis may mimic sepsis. This may confuse early diagnosis of the cardiac anomaly, which may be classified as either an acyanotic or a cyanotic defect.

Children with a known or new-onset cardiac condition may present with a defect in their cardiac pump. The defect may have many possible etiologies. Often the outflow or filling of the heart is impeded, which prohibits forward flow of blood and results in edema. The pediatric patient may present in a similar fashion as an adult, with pulmonary edema, jugular venous distention, and hepatic venous congestion. Peripheral edema may also be assessed in common places, such as around the eyes, the lower back, or the top of the foot.

Cardiac Defects That Do Not Cause Cyanosis

A cardiac lesion is a cardiac defect or anomaly present at birth. Certain cardiac lesions do not cause cyanosis. They may cause obstruction of the left side of the heart or cause a left-to-right shunt of blood **Figure 23-20**. Lesions that cause a left-to-right shunt of blood cause systemic circulation to flow into the pulmonary vasculature. The higher pressure in the left side of the heart forces blood into the right side of the heart that has lower pressure. The resulting increased blood volume in the right side of the heart causes high pulmonary arterial blood flow and pressure and symptoms of tachypnea, poor feeding, irritability, failure to thrive, and, if left untreated, congestive heart failure. Examples of cardiac lesions that can create a left-to-right shunting of blood include ventricular septal defects, atrioven-tricular canal defects, atrial septal defects, aortic stenosis, patent ductus arteriosus, and pulmonary stenosis.

Signs and Symptoms

Cardiac Anomalies

Congenital Cardiac Anomaly

- Heart murmur

- Pallor
- Poor perfusion
- Lethargy
- Cyanosis

Defect in the Cardiac Pump

- Pulmonary edema
- Jugular venous distention
- Hepatic venous congestion
- Peripheral edema around the eyes, lower back, or top of the foot

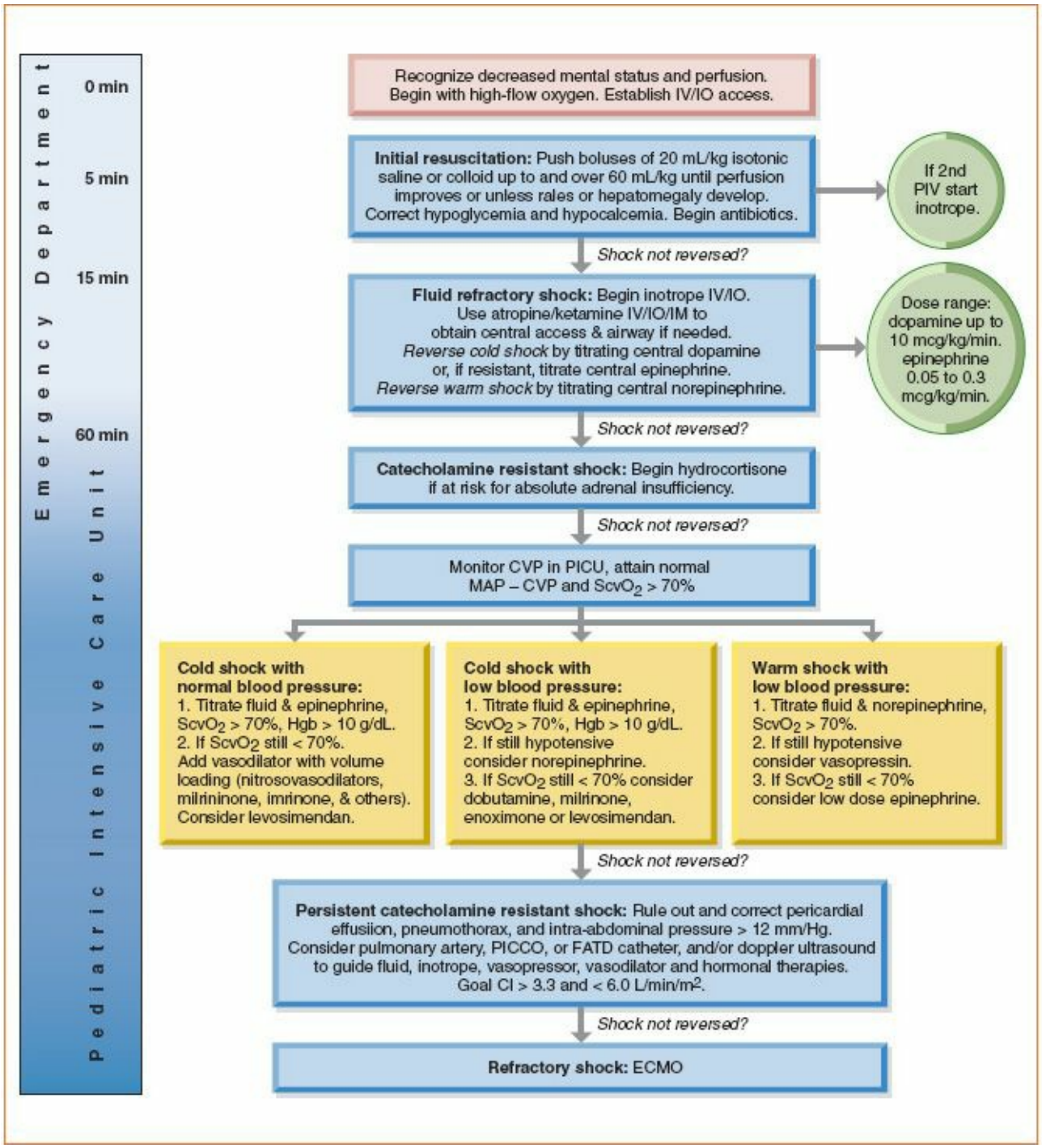

Figure 23-19 Algorithm for time-sensitive, goal-directed, stepwise management of hemodynamic support in infants and children. Proceed to next step if shock persists.

Abbreviations: CI, cardiac index; CRRT, continuous renal replacement therapy; CVP, central venous pressure; ECMO, extracorporeal membrane oxygenation; FATD, femoral arterial thermodilution; Hgb, hemoglobin; IM, intramuscular; IO, intraosseous; IV, intravenous; MAP, mean arterial pressure; PICCO, pulse contour cardiac output; PICU, pediatric intensive care unit; PIV, peripheral intravenous line; $ScvO_2$, mixed central venous oxygen saturation.
Source: Reproduced from Brierley J, et al. Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update. *Crit Care Med*. 2009 Feb;37(2):677. Used with permission of Wolters Kluwer Health.

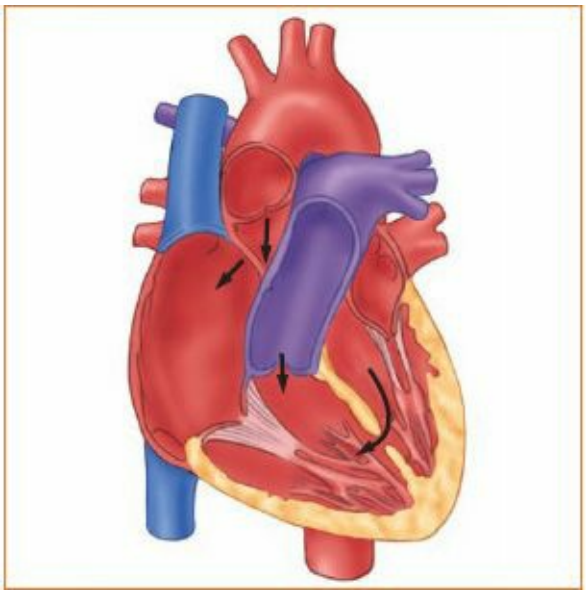

Figure 23-20 Left-to-right shunt of blood.

Locations of various cardiac lesions are shown in **Figure 23-21**. A **ventricular septal defect (VSD)** is a hole in the septum between the ventricles, in which blood from the left ventricle flows into the right ventricle. An **atrioventricular canal defect** is a hole in both the atrial and ventricular septal walls that allows blood to flow from the left side of the heart to the right side of the heart. It usually involves defects of both the mitral and tricuspid valves. **Atrial septal defects (ASDs)** occur as a hole in the atrial septal wall that allows blood to flow from the left atrium into the right atrium. **Aortic stenosis** is a narrowing of the aortic valve, and **pulmonary stenosis** is narrowing of the pulmonary valve. If the aortic and pulmonary stenosis is severe in these lesions, the patient may be cyanotic. Finally, **patent ductus arteriosus (PDA)** is a situation in which the ductus arteriosus, which assists in fetal circulation, does not transition as it should after birth to become the ligamentum arteriosum. The result is that the connection between the pulmonary artery and the aorta remains, allowing some oxygenated blood to move back into the heart rather than all of it moving out of the aorta and into the systemic circulation **Figure 23-22**.

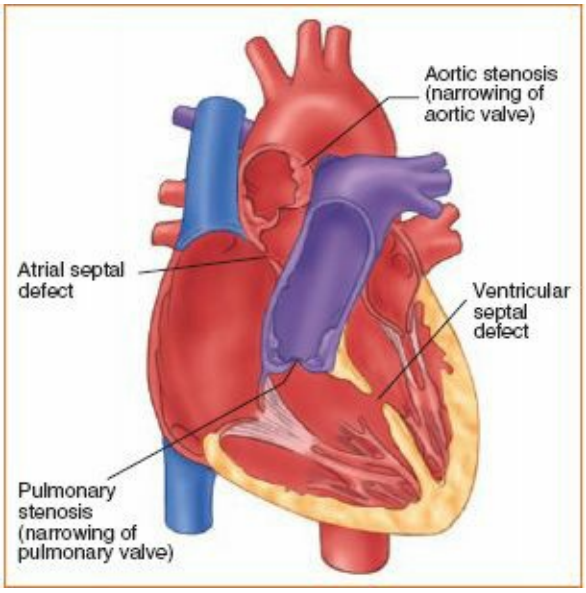

Figure 23-21 Locations of various cardiac lesions. Atrioventricular canal defect is not shown; it is a hole in both the atrial and ventricular septal walls.

Signs and Symptoms

Acyanotic Cardiac Anomalies

- Tachypnea
- Poor feeding
- Irritability
- Failure to thrive
- Congestive heart failure

Left Heart Obstruction Defects

Aortic stenosis, hypoplastic left heart syndrome, and coarctation of the aorta are all examples of obstructions that cause impaired left ventricular output that may be treated in the critical care transport setting. The neonate presents with symptoms of shock near the second week of life because this is when the PDA begins to close. These lesions are dependent on the PDA because it prevents pulmonary hypertension and congestion. As the PDA begins to close and pulmonary hypertension increases, symptoms of tachypnea, poor feeding, and congestive heart failure become evident. Untreated, this leads to cardiogenic shock.

Persistent pulmonary hypertension (PPHN) in the newborn is caused by an abnormally elevated pulmonary vascular resistance resulting in hypoxia. The typical presentation consists of low APGAR scores, hypoglycemia, or a congenital diaphragmatic hernia. In severe cases, these patients will present with marked cyanosis, decreased peripheral perfusion, and metabolic acidosis. The manifestation of congestive heart failure stems from the increased pressure overload on the right ventricle, which is typically a low-pressure system.

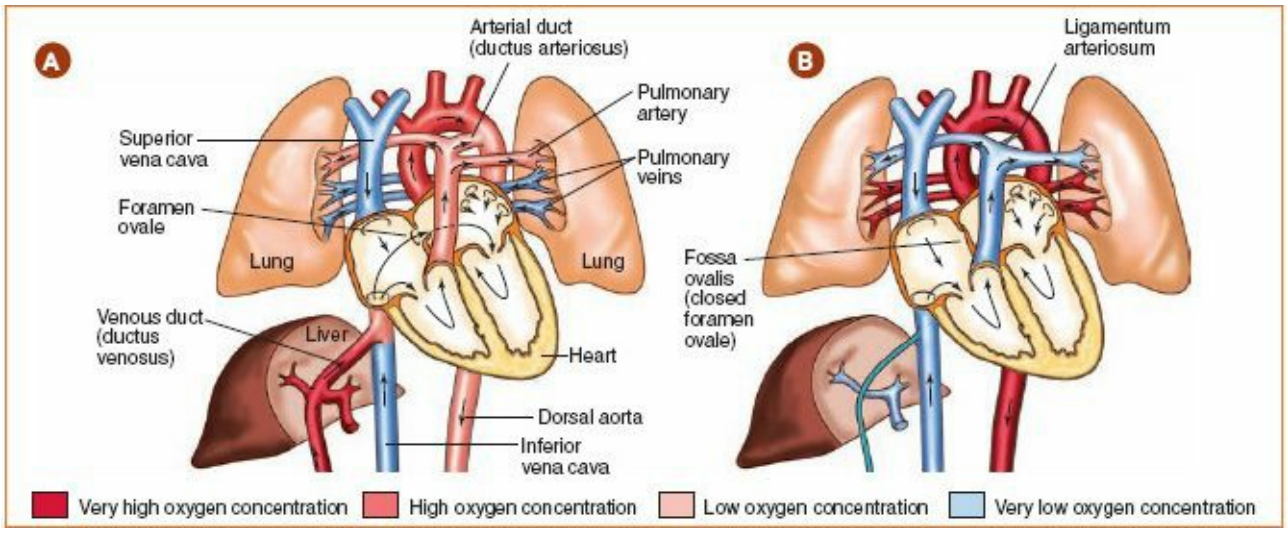

Figure 23-22 The ductus arteriosus plays a role in fetal circulation before birth **(A)** and normally transitions to become the ligamentum arteriosum after birth **(B)**.

Extracorporeal membrane oxygenation (ECMO) is a supportive intervention that is typically only used for short durations and is indicated for patients with PPHN and congenital diaphragmatic hernia. It provides oxygenation to a patient whose lungs and possibly heart may be failing, and does so by removing the patient's blood to a heart and lung machine that does the work normally done by the lungs and heart, then transfusing it back into the patient's circulation. The most common forms of ECMO in the transport setting include venoarterial and venovenous. Venovenous ECMO cannulates the venous circulation, whereas venoarterial ECMO cannulates the arterial circulation. Venovenous ECMO has a lower Pa_{O_2}, requires a higher perfusion rate, maintains pulmonary blood flow, and does not provide cardiac support to assist systemic circulation. Conversely, venoarterial ECMO has a higher Pa_{O_2}, requires a lower perfusion rate, bypasses the pulmonary circulation, and provides cardiac support to assist systemic circulation.

In the neonatal patient population, venoarterial ECMO is the form most predominately used. Exclusion criteria for the initiation of ECMO in the neonatal population consist of the following: birth weight of less than 2,000 g or younger than 35 weeks' gestation, evidence of grade II intraventricular hemorrhage or bleeding, mechanical ventilation with high F_{IO_2} and pressure settings for more than 7 to 10 days, and any signs of irreversible or fatal congenital anomalies. For pediatric and adult populations, there are no set inclusion or exclusion criteria for ECMO.

The decision to perform ECMO is usually based on the overall prognosis of the patient and the level of expertise of the institution involved. To date, there are only a few ECMO transport teams in the United States that have the capability of performing this type of transport. Patients undergoing ECMO are extremely fragile and require a multi-disciplinary transport team consisting of a physician, a neonatal/pediatric nurse, and a respiratory therapist who has specialty training and familiarity with these complex and intricate transports. Prior to the decision to transport, the referring physician and transport personnel need to ensure that the patient's parents have been educated on the severe instability of the child's health and the potential for deterioration or even cardiac arrest during transport to the receiving facility.

Hypoplastic left heart syndrome (HLHS) is the underdevelopment of the aorta, aortic valve, left ventricle, and mitral valve. This defect involves the entire left side of the heart. This cardiac lesion is dependent on a PDA, patent foramen ovale (PFO)/ASD for survival. **Coarctation of the aorta (CoA)** is a pinching or narrowing of the aorta that obstructs blood flow from the heart to the systemic circulation.

Signs and Symptoms

Acyanotic Cardiac Defects

- Tachypnea
- Poor feeding
- Irritability
- Failure to thrive
- Congestive heart failure, if untreated

Cardiac Defects That Cause Cyanosis

Some cardiac lesions are diagnosed early after birth because cyanosis is present. Most children with such lesions are identified early as neonates; the small percentage who are not identified early are due to the PDA sometimes allowing sufficient mixing of oxygenated and deoxygenated blood. An example of this may be **tetralogy of Fallot (TOF)**. This cardiac anomaly consists of four defects: ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and overriding aorta. When the PDA closes, these neonates develop tachycardia, tachypnea, and cyanosis. TOF is the most common heart defect seen beyond infancy. The CCTP should be aware of a hypercyanotic spell, also called a TET spell. TET spells present with marked cyanosis, irritability, tachypnea, pallor, and even a loss of consciousness. The cyanosis involved may be related to a transient increase in the obstruction of the right ventricular outflow tract. The CCTP should expect the following medications/interventions to be ordered, or to follow their agency protocols. Such orders may consist of supplemental oxygen, morphine, propranolol, and placing the patient in a knee-chest position.

Transposition of the great arteries (TGA) is a defect in which the great vessels are reversed **Figure 23-23**. The aorta is connected to the right ventricle, thereby perfusing the body with deoxygenated blood. In addition, the pulmonary artery is connected to the left ventricle, thereby carrying oxygenated blood back to the lungs. This creates a parallel circulation that is dependent on an ASD/PFO, VSD, or PDA to provide mixing of oxygenated and deoxygenated blood; if a defect allows oxygenated blood to eventually reach the systemic circulation, the body can still remain perfused to some extent. Because of a large VSD or ASD, a small percentage of these neonates, about 10%, will not develop cyanosis until the PDA closes. As the PDA closes, these neonates will present with classic signs of cyanosis, especially during episodes of crying or while feeding. Unsupported or uncorrected, this cyanosis will lead to hypoxia and metabolic acidosis.

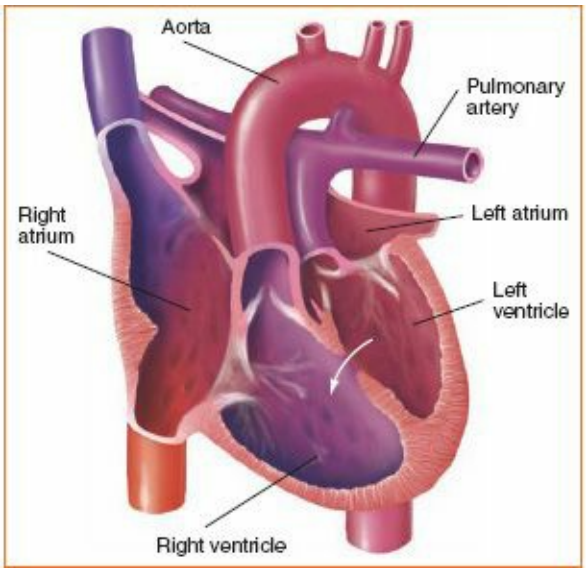

Figure 23-23 Transposition of the great arteries. Note that a ventricular septal defect is shown here as well.

Signs and Symptoms

Cyanotic Cardiac Defects

- Tachycardia
- Tachypnea
- Cyanosis
- TET spell (tetralogy of Fallot)

General Management Priorities

As with any critical care event, management priorities begin with patient assessment and support of the patient's airway, breathing, and circulation. All pediatric patients with congestive heart failure, regardless of age, require diuretics. Fluid boluses that may be required related to hypotension are given at 10 mL/kg. The CCTP must monitor for a situation in which fluid boluses may not be helpful and may increase pulmonary edema related to congestive heart failure. At that point, switching to a vasopressor may be a better option.

Any cardiac lesion that is dependent on the ductus arteriosus requires the administration of a prostaglandin E1 (PGE1 [Prostin VR]) infusion. PGE1 is usually initiated at the sending facility by the pediatric intensive care unit physician. The initial dose is 0.05 to 0.1 µg/kg/min, with a maintenance dose of 0.01 to 0.05 µg/kg/min. Pertinent side effects of prostaglandin are jitteriness, fever, hypotension, and apnea. Concerns for airway management must be considered before transport.

A child who shows persistent signs of hypoperfusion and/or cardiogenic shock may require the inotropic benefits of dopamine and dobutamine administration. These medications can increase peripheral vascular resistance, thus improving blood pressure, perfusion, and cardiac contractility. The neonatal dosage for dopamine (Intropin) is 5 to 10 µg/kg/min; the pediatric dosage is 2.0 to 20 µg/kg/min. The neonatal dosage for dobutamine (Dobutrex) is 2 to 10 µg/kg/min; the pediatric dosage is 2.5 to 20 µg/kg/min.

Transport Management

Cardiac Anomalies

- Assess and support the patient's ABCs.
- Administer diuretics.
- Monitor fluid bolus administration to determine if administration becomes unhelpful and increases pulmonary edema.
- Administer prostaglandin infusion, if required.
- Administer dopamine and dobutamine, if required.

Figure 23-24 Pediatric critical care transport patient with electrocardiographic (ECG) leads placed. A 12-lead ECG is not as common in children.

Pediatric ECG Interpretation

The interpretation of an ECG rhythm in children is different from that in an adult. To decrease the amount of respiratory artifact, it is helpful to avoid placing the ECG leads at the level of the diaphragm. The leads may be placed on the lower abdomen or on the patient's thighs. A 12-lead ECG is not as common in children but is obtained for children with abnormal findings on the rhythm strip, arrhythmias, congenital heart disease, acquired heart disease, chest trauma, or suspected cardiotoxic ingestions **Figure 23-24**.

The first aspect of rhythm strip interpretation is assessing whether the rate is appropriate for the child's age. The next step is evaluating the components of the rhythm, including the PR interval and the width of the QRS complex. Normal values for these components vary with age. A general guideline for children is a PR interval of 0.16 or less and a QRS complex of 0.08 or less. A PR interval of greater than 0.16 indicates a first-degree heart block. A QRS complex of greater than 0.08 indicates a wide complex arrhythmia. In tachyarrhythmia, it may be helpful to increase the ECG monitor sweep speed (typically 25 mm/s) to assess for the presence of a P wave. By increasing the ECG monitor sweep speed, the CCTP can identify subtle nuances in the components of the ECG (P, QRS, T wave).

Renal Disorders

Acute renal failure is defined as profound deterioration of renal function associated with increasing levels of solute (BUN or creatinine). Actual urine volume can vary from normal urine output to oliguria. Causes are generally divided into three categories—**prerenal, intrarenal**, and **postrenal**. Prerenal acute renal failure is caused by decreased perfusion of an intact nephron. Intrarenal acute renal failure is caused by damage of an actual nephron. Postrenal acute renal failure is caused by downstream obstruction with initially intact nephrons. **Table 23-10** includes common causes of acute renal failure.

Prerenal

Prerenal causes are the most common in children and are primarily related to dehydration and decreased renal perfusion. Dehydration results from fluid losses or acute blood loss. The patient may present with a range of signs and symptoms—nausea, vomiting, diarrhea, diabetic ketoacidosis, all forms of shock, and burns. A decreased circulating blood volume results in decreased renal perfusion.

Other causes of decreased renal perfusion may be related to any cardiac condition that results in decreased cardiac output, such as congestive heart failure. In patients with sepsis or burns, there may actually be sufficient or increased extracellular fluid volume as a result of increased vascular permeability, but the intravascular volume is greatly depleted.

Intrarenal

Intrarenal or parenchymal causes are diverse; all etiologies, however, lead to nephron damage and loss of glomerular function as a result of damage to the glomerular basement membrane. Damage to the arterioles and glomerular capillaries may be the result of coagulation, inflammation, barotraumas, or prolonged ischemia.

The proximal tubular cells of the kidneys have high metabolic demands that make them more susceptible to ischemic insults. Nephrotoxins cause sloughing and necrosis of cells in the proximal tubule and ascending loop of Henle, which result in obstruction of tubular blood flow and leakage of fluid into the peritubular space and plasma.

Occlusion of the renal arteries from emboli or thrombosis causes infarction as a result of decreased renal blood flow. Another example includes vascular congestion and hemorrhagic necrosis as a result of renal vein thrombosis.

Hemolytic uremic syndrome (HUS) is one of the most common causes of intrarenal failure seen in infants and children younger than 4 years. The classic triad identified in HUS is microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure (ARF).

Hemolytic Uremic Syndrome

Hemolytic uremic syndrome (HUS) is a serious condition in which the patient develops ATF. It is thought to be caused by several bacterial and viral infectious organisms; however, verotoxin-producing strains of *E. coli* have been associated with 75% of cases. The primary pathology is thought to be related to endothelial cell injury in the renal cortex that leads to localized vascular coagulation and fibrin deposits. As the red blood cells pass through the fibrin strands in the renal vasculature, they become fractured and cause microangiopathic anemia. Thrombocytopenia is the result of localized intravascular coagulation-consuming platelets.

Presentation typically follows a gastroenteritis-type illness, and symptoms include vomiting, abdominal pain, and bloody diarrhea. As the gastrointestinal symptoms subside, the child again becomes acutely ill, presenting with irritability, pallor, and signs of fluid overload, including peripheral and pulmonary edema, hypertension, and petechiae.

TABLE 23-10 Common Causes of Acute Renal Failure

Type of Failure	Causes	Examples
Prerenal (decreased perfusion of an intact nephron)	Hypovolemia	Hemorrhagic shock Dehydration (GI losses and DKA) Insensible losses (prematurity, burns) Sodium and water losses (renal, adrenal, endocrine) Hypoproteinemia
	Hypotension	CHF Decompensated shock Congenital heart disease
	Renal vessel injury with obstruction	N/A
Intrarenal (damage of the actual nephron)	Primary parenchymal disease	Hemolytic uremic syndrome Post-strep (acute) Glomerulonephritis Lupus erythematosus Henoch-Schonlein purpura Pyelonephritis Malignant hypertension
	Acute tubular necrosis	Ischemia (prolonged prerenal ARF) Nephrotoxins (heavy metals, uric acid, NSAIDs, ACE inhibitors, aminoglycoside antibiotics, beta-blockers, diuretics, radiocontrast, myoglobin, organic solvents)
	Large-vessel occlusion	Renal vein thrombosis Renal artery stenosis Thrombosis or emboli
	Congenital abnormalities	Renal agenesis Infantile polycystic kidney disease Bilateral multicystic kidneys
	Neoplasm	N/A
Postrenal (downstream obstruction with initially intact nephrons)	Obstructive uropathy	Posterior urethral valves Prune belly syndrome Neurogenic bladder UPJ syndrome Tumor Nephrolithiasis Renal calculi Trauma to the collecting system Ureterocele

Abbreviations: ACE, angiotensin-converting enzyme; ARF, acute renal failure; CHF, congestive heart failure; DKA, diabetic ketoacidosis; GI, gastrointestinal; N/A, not applicable; NSAID, nonsteroidal

anti-inflammatory drug; UPJ syndrome, ureteropelvic junction syndrome.

Laboratory studies show fragmented red blood cells (RBCs), thrombocytopenia, and evidence of ARF, including elevated BUN and creatinine levels, hyperkalemia, and metabolic acidosis. Urine studies show proteinuria and hematuria with or without anuria.

Treatment of HUS is primarily supportive and includes fluid and metabolic balance, control of hypertension (treatment of diastolic pressure above 120 mm Hg with labetalol, nifedipine, or hydralazine), transfusion of packed RBCs and platelets if required, and aggressive treatment of renal failure with possible dialysis (peritoneal) in the hospital. The prognosis is usually excellent when aggressive treatment is initiated early.

Signs and Symptoms

Hemolytic Uremic Syndrome

- Gastroenteritis-type symptoms: vomiting, abdominal pain, and bloody diarrhea
- After the Gl symptoms subside: irritability, pallor, signs of fluid overload, including peripheral and pulmonary edema, hypertension, and petechiae

Transport Management

Hemolytic Uremic Syndrome

- Support fluid and metabolic balance.
- Control hypertension.
- Transfuse packed RBCs and platelets if required.

Signs and Symptoms

Acute Poststreptococcal Glomerulonephritis

- Gross hematuria with urine the color of tea or cola
- Congestive heart failure
- Peripheral and pulmonary edema
- Hypertension

Transport Management

Acute Poststreptococcal Glomerulonephritis

- Restrict fluids.
- Administer loop diuretics.

Acute Glomerulonephritis

Acute glomerulonephritis (AGN) is a histopathologic diagnosis that is associated with edema, hypertension, and hematuria. AGN results from the deposits of circulating immune complexes in the kidney basement membrane, which ultimately causes reduced glomerular filtration. This syndrome can be induced by a variety of organisms, but the most common is group A beta-hemolytic streptococci. **Acute poststreptococcal glomerulonephritis (APSGN)**, or AGN following an infection by streptococci, can occur following a skin infection or a pharyngeal infection. APSGN occurs most commonly in school-aged children and more commonly in males than females.

The pathology of APSGN is thought to be an activation of the complement pathway leading to inflammation that causes glomerular damage. This occurs as a result of immune complex deposition in the basement membrane of the glomeruli.

Presentation typically manifests 1 to 2 weeks after the streptococcal infection, and a classic symptom is gross hematuria with urine the color of tea or cola (soda pop). The severity of presentation runs the spectrum from mild and not requiring hospitalization, to severe oliguria with symptoms of overload including congestive heart failure, peripheral and pulmonary edema, and hypertension.

Laboratory urinalysis values reveal RBCs and proteinuria. White blood cells represent glomerular inflammation and not pyelonephritis (inflammation of the kidney linings). In addition, expected evidence of renal insufficiency includes elevated BUN and creatinine levels, hyperkalemia, hyponatremia, and acidosis. Elevated antibody titers (or the presence and amount of antibodies in the blood) of streptococci confirm APSGN.

Treatment of severe APSGN is primarily supportive and includes fluid restriction of IV solution to avoid promoting peripheral and pulmonary edema. Loop diuretics are useful in treating mild hypertension, edema, and fluid overload. The prognosis is good, and full recovery is expected in more than 98% of all children.

Acute Tubular Necrosis

Acute tubular necrosis (ATN) is a condition that results in damage to the tissue of the kidneys' tubules. Like most renal pathologies, it can have numerous etiologies; however, the most common is renal ischemia precipitated by hypovolemia. Tubular damage can also occur following a toxic insult, such as heavy metal poisoning or as a result of myoglobin or hemoglobin accumulating in the tubules following a severe crush injury, burn, or hemolytic crisis.

With ATN, the renal tubular cells die when they do not get enough oxygen or if they are exposed to a toxic drug. Injury of the tubular cells is most prominent in the straight portion of the proximal tubules and in the loop of Henle. In addition to decreased filtration, there is also an increase of casts and debris obstructing the tubule lumen that causes a leak of filtrate through the damaged epithelium. In addition, decreased perfusion also leads to diminished production of vasodilators such as nitric oxide, leading to further vasoconstriction and hypoperfusion.

ATN is clinically defined in three phases. The first period is the oliguric phase, during which severe oliguria lasts approximately 10 days. Renal recovery of ATN is highly unlikely if the oliguria or anuria persists longer than 3 to 6 weeks. The second phase is the diuretic phase in which there is passage of large volumes of isothenuric urine containing sodium levels of 80 to 150 mEq/L. Finally, the third phase is the recovery phase in which signs and symptoms resolve rapidly as a result of regeneration of tubular epithelial cells. However, polyuria may be present for days to weeks, although the mechanism of the diuresis is not completely understood.

Transport treatment usually consists of fluid and electrolyte replacement. Ultimately dialysis is required to remove the toxins.

Signs and Symptoms

Acute Tubular Necrosis

- Severe oliguria lasting approximately 10 days
- Diuretic phase: passage of large volumes of isothenuric urine
- Recovery phase: signs and symptoms resolve rapidly
- Polyuria

Transport Management

Acute Tubular Necrosis

- Replace fluids and electrolytes.

Postrenal

Postrenal or obstructive failure has numerous etiologies. In most cases, unilateral obstructive abnormalities do not result in failure. Flank and/or abdominal pain is often present. Prolonged, unrelieved obstruction causes irreversible parenchymal damage because of infection and increased hydrostatic pressure. Obstruction of urine outflow in the renal system causes an increase in hydrostatic pressure at the proximal tubule and glomerulus, resulting in a decrease in glomerular filtration and renal function.

Complications

There are five potential life-threatening events that can occur in acute renal failure. The first event includes electrolyte imbalances such as hyponatremia, hypocalcemia, and hyperkalemia. **Hyponatremia** is most often dilutional (low sodium levels as a result of excessive fluid intake) secondary to volume overload, is asymptomatic, and is simply treated with fluid restrictions. Severe hyponatremia may cause seizures and is managed by infusing a hypertonic (3%) saline solution.

Hypocalcemia is caused by hyperphosphatemia and is not usually treated unless symptoms such as tetany, seizures, or decreased cardiac contractility are present. Rapid correction of hypocalcemia may result in the deposit of calcium salts in body tissues. Treatment includes oral dosing of calcium carbonate or, in emergent cases, cautious administration of IV calcium gluconate (10%) at a dose of 50 to 100 mg/kg).

Hyperkalemia has the potential to cause life-threatening arrhythmias by producing membrane excitability. ECG changes may include a peaked T wave, a widened QRS complex, and eventual bradycardia. An elevated serum potassium level is treated based on lab values and the presence of ECG abnormalities. Management may include careful monitoring and the removal of potassium chloride from IV fluids to pharmacologic treatments and, in rare circumstances, the use of dialysis. Possible medications include the following:

- **Kayexylate (sodium polystyrene sulfonate resin)**: An ion exchange resin that acts in the colon or ileum. Usual dosing is 1 g/kg every 2 to 4 hours and may be mixed with sorbitol.
- **Calcium gluconate:** Has no direct effect on the serum potassium but stabilizes the myocardial membrane and ventricular rhythm by changing the cell action potential. Monitor the heart rate and stop the infusion if the child's pulse rate drops by more than 20 beats/min.

- **Glucose and insulin:** Potassium can be moved intracellularly by administering a glucose infusion of 5% dextrose in water or 5% dextrose and 0.25 normal saline and a concurrent insulin infusion. The goal is to maintain the glucose level between 120 and 300 mg/dL. This has a rapid effect on lowering the serum potassium level; however, careful assessment of glucose blood levels is necessary to prevent hypoglycemia.
- **Sodium bicarbonate:** Sodium bicarbonate can be used even in the absence of acidosis. Calcium gluconate is not compatible with $NaHCO_3$ and IV lines must be flushed prior to administration.
- **Albuterol:** Nebulized albuterol causes a shift of extracellular potassium into the cell and can lower the serum potassium by 1.0 to 1.5 mEq/L. Albuterol also helps to protect against insulin-induced hypoglycemia.

Another life-threatening complication related to acute renal failure is related to volume losses or pulmonary edema and fluid overload. If it has been determined that there is prerenal ARF related to volume losses, immediate fluid replacement should occur to prevent possible progression to ATN. Initially, a fluid bolus of 20 mL/kg may be administered for a total of three infusions (3 doses) using a crystalloid solution. Once the source of the loss is treated and hypovolemia is corrected, maintenance fluids are initiated. The degree of fluid restriction depends on whether the patient is anuric, oliguric, or nonoliguric.

Volume overload may be best managed with the aid of a central venous pressure monitor, which adequately measures fluid resuscitation to avoid overhydration. The use of diuretics is controversial except in cases of ARF secondary to myoglobinuria or hemoglobinuria. In these cases, furosemide (1 mg/kg) or mannitol (0.5 to 0.75 mg/kg/dose) may be used. The use of furosemide in the presence of fluid overload may be therapeutic; in cases of euvolemia and oliguria, furosemide may stimulate urine production and filtration of waste products and improve renal function by increasing blood flow to this organ.

Hypertension that may progress to hypertensive encephalopathy is also life threatening. Hypertension is usually caused by sodium and water retention. Hypertensive encephalopathy is hypertension associated with neurologic symptoms such as nausea, vomiting, headache, visual changes, seizures, and mental status changes. As with any hypertensive crisis, it is important to aim for a MAP reduction of 15% to 25%. A more precipitous or pronounced drop in MAP that returns the child to normal blood pressure parameters may lead to hypoperfusion of the end organs. Septic shock may occur secondary to obstruction and/or infection.

Signs and Symptoms

Meningococcal Infections

- Fever
- Chills
- Nuchal rigidity
- Vomiting
- Photophobia
- Headache
- Back pain
- Seizures (in older children)

Infants and Young Children

- Poor feeding
- Marked irritability and agitation
- Characteristic high-pitched cry with bulging fontanelles
- Poor cry
- Decreased muscle tone
- Jaundice
- Weight loss
- Hypothermia
- Apnea
- Cyanosis
- Seizure activity

Transport Management

Meningococcal Infections

- Ensure that standard universal precautions are followed.

Meningococcal Infections

The presentation of the pediatric patient with a meningococcal infection depends largely on the age of the child, the type of organism, and the present state of health of the child. The onset of symptoms is typically abrupt, with hallmark signs that include fever, chills, nuchal rigidity, vomiting, photophobia, headache, back pain, and seizures in older children. Infants and young children may present with some of the same symptoms as the older child, but also may experience poor feeding, marked irritability and agitation, and a characteristic high-pitched cry with bulging fontanelles. The CCTP may test for or inquire whether the patient has a positive Brudzinski sign and Kernig sign on arrival to the bedside.

Kernig sign is the inability of the patient to straighten his or her leg with the hips flexed at a right angle. To evaluate for this condition, have the patient lie supine with the legs flexed at the hip and the knees flexed; the CCTP then tries to extend the knees. An abnormal response is present if there is pain and resistance to knee straightening (positive Kernig sign). If pain and resistance are present bilaterally, then meningeal irritation is likely present.

Brudzinski sign is flexion of the legs and thighs with forceful flexion of the neck onto the chest. Testing for this condition should not be performed if a cervical spine injury is suspected. Have the patient lie supine while the examiner flexes the head and neck in an attempt to have the chin touch the chest. An abnormal response includes flexion of the legs and hips during this process (positive Brudzinski sign).

The neonate patient may be extremely difficult to assess, and it is also difficult to make the diagnosis of meningococcal infection in a neonate because the symptoms are usually vague in nature and challenging to differentiate from other septic etiologies. The CCTP should inquire about the infant's feeding and sucking ability. The infant may also present with a poor cry, decreased muscle tone, jaundice, weight loss, hypothermia, apnea, cyanosis, and seizure activity.

Standard universal precautions should be taken with all pediatric patients with a suspected diagnosis of meningococcal infections, considering the close proximity of the patient/provider depending on the mode of transport.

Trauma

Trauma is the leading cause of death of children from 6 months of age through young adulthood. The most common pediatric injuries related to trauma are the result of motor vehicle (passenger), pedestrian, bicycle, submersion, burn, and firearm injuries. The systematic approach taken in the assessment and management of pediatric trauma patients is the same as in the adult population.

There are two different resources for CCTPs when assessing the severity of a pediatric patient. The most common is the GCS score that has been altered for pediatric patients, presented earlier in this chapter. The pediatric trauma score is another resource that accounts for weight, airway status, central nervous system (CNS) status, systolic blood pressure, fractures, and wounds.

Reprinted from *J Pediatr Surg*. 22(1), Tepas JJ III, Mollitt DL, Talbert JL, Bryant M. The pediatric trauma score as a predictor of injury severity in the injured child, 14–18, Copyright 1987, with permission from Elsevier (http://www.sciencedirect.com/science/journal/00223468).

A value is calculated for each of the components, as shown in **Table 23-11**. The values are then added to determine the total score. Scores are categorized as follows:

- A score of 9 to 12 indicates minor trauma. Follow local guidelines/protocols for treatment.
- A score of 6 to 8 indicates a potentially life-threatening condition. Consider transporting to a trauma center.
- A score of 0 to 5 indicates a life-threatening condition. Transport to a trauma center.
- A score of less than 0 indicates a condition that is usually fatal. This patient needs to be transported to the nearest trauma center equipped to care for this critically ill pediatric trauma patient.

Head Injuries

Head trauma is the leading cause of morbidity and mortality in children and accounts for 70% to 80% of all pediatric deaths from trauma. The death rate from head injury is five times higher than the next most common cause of pediatric mortality—leukemia. The main source of head trauma is motor vehicle accidents involving passengers, pedestrians, or bicyclists. Less common sources of head injury include sports-related injuries, falls, and abuse.

Injuries to the brain are traditionally evaluated as primary or secondary injuries. Primary injuries that result from penetrating, rapid deceleration or rotational injuries occur at the moment of impact and cause physical and mechanical destruction of brain tissue; they can include parenchymal injury, diffuse axonal injuries, and cerebral edema. In comparison with primary injuries, many factors may improve or worsen secondary brain injury, which involves a complicated cascade of cellular destruction. The primary brain injury causes a certain amount of tissue and cellular death. There may be injured areas of the brain (penumbra) surrounding the dead tissue that may remain viable. These injured areas are vulnerable to hypoxic, ischemic, and inflammatory responses. Aggressive management to prevent hypoxia, hypotension, and progressive edema is required to protect this unstable brain tissue. If

uncorrected, secondary brain injury leads to irreversible brain damage and death.

Increased Intracranial Pressure

Increased intracranial pressure (ICP) can be from numerous etiologies. The most common cause of increased ICP in pediatric populations is related to trauma or a failed cerebral spinal fluid shunt.

Possible signs and symptoms of increased ICP include headache, irritability, fever, dizziness, nausea, projectile vomiting, lethargy, visual changes, unsteady gait, high-pitched cry, bulging fontanelle, pupillary dilation, seizures, coma, and Cushing's triad. The symptoms become more progressive and severe as the ICP rises.

Treatment modalities may include inserting a monitor to measure ICP. This treatment is more pertinent for interfacility transports as opposed to prehospital transports. A ventricular catheter is placed in the ventricles connected to an external ventricular drain. This drain evacuates fluid within the ventricle and measures objective ICP. A subarachnoid bolt placed in the subarachnoid space can also measure ICP. This bolt cannot drain fluid and does not sound an alarm for increased pressure. Normal ICP is 0 to 10 cm H_2O. An acceptable ICP is usually below 15 to 20 cm H_2O. Cerebral perfusion pressure should be a minimum of 50 to 60 mm Hg for adequate brain perfusion.

Management goals include the following:

- Mild hyperventilation ($PaCO_2$ between 30 and 35 mm Hg)
- Maintain the cerebral perfusion pressure at greater than 50 to 60 mm Hg (increased MAP to maintain adequate cerebral perfusion pressure)
- Place the stretcher so that the patient's head is raised 15° to 30° to promote cerebral venous drainage
- Normothermia or mild hypothermia to decrease metabolic rate
- Administer mannitol (0.5 to 0.75 mg/kg/dose) (must administer with a filter)

Signs and Symptoms

Increased Intracranial Pressure

- Headache
- Irritability
- Fever
- Dizziness
- Nausea
- Projectile vomiting
- Lethargy
- Visual changes
- Unsteady gait
- High-pitched cry
- Bulging fontanelle
- Papillary dilation
- Seizures
- Coma
- Cushing's triad

Transport Management

Increased Intracranial Pressure

- Maintain cerebral perfusion pressure at greater than 50 to 60 mm Hg.
- Position the patient so the head is raised 15° to 30°.
- Maintain a normal or slightly elevated temperature.
- Administer mannitol.
- Manage pain.

- Pain management
- Possible coma from barbiturates (decreases the cerebral metabolic rate and oxygen consumption)

Concussion

A **concussion** is the most commonly seen head injury in the pediatric population. It is described as a transient interruption of normal neurologic function. There may be a brief loss of consciousness and posttraumatic amnesia. The CCTP's role in transporting a pediatric patient with a concussion is to provide supportive measures while en route to a receiving facility. As with any neurologic injury, it is necessary to frequently perform and document a neurologic assessment (baseline and continuous) during the transport. The patient's pain level and tolerance of the transport process should also be documented.

Transport Management

Concussion

- Provide supportive measures.
- Frequently perform and document a neurologic assessment (baseline and continuous).
- Document the patient's pain level and tolerance of transport.

Cerebral Contusion

A **cerebral contusion** most commonly occurs at the point of impact and/or on the side opposite the impact, such as with a coup-contracoup injury. A cerebral contusion is a focal hemorrhage in the brain that may be associated with a concurrent concussion.

Epidural Hematoma

Epidural hematomas typically include arterial bleeding from contusions or lacerations, most commonly of the middle meningeal artery. This blood vessel is located just outside the dura and may be torn when there is a parietal skull fracture. Another cause of epidural bleeding may be from damage to the diplotic veins in the posterior cranial fossa, although the most common cause is a lateral temporal fossa injury. Accumulation of blood in these injuries does not "touch" the brain tissue.

The classic pattern of an epidural hematoma includes a lucid period followed by rapid neurologic deterioration; however, this classic pattern only occurs in a minority of pediatric patients with an epidural hematoma. Children may never have a loss of consciousness or a lucid period.

Subdural Hematoma

A subdural hematoma is five to ten times more common than an epidural hematoma, especially in infants.

This injury is caused by shearing forces that slide the fragile brain tissue over the rough base of the skull. The hematoma occurs just below the dura, and as a result, blood comes into contact with brain tissue. Subdural hematomas are usually caused by a disruption of bridging veins or venous sinus beneath the dura. A subdural hematoma can be a result of child abuse such as shaken baby syndrome. Shaking an infant violently causes acceleration-deceleration forces that shear the bridging veins in the subdural space.

Risk Assessment and Management

Many children who experience closed head trauma may have only minor injuries. Children who have low-risk injuries are often asymptomatic, with normal neurologic findings on exam. Management at home with responsible adult supervision may be all that is necessary.

Children with moderate-risk injuries will present with an altered level of consciousness, progressive headache, or vomiting and may have an associated injury. They often require basic trauma care, including basic wound management and cervical spine immobilization with radiologic evaluation.

Children with high-risk injuries will present with a depressed level of consciousness and possible neurologic deficits or signs of increased ICP. These children often require immediate surgical interventions. Good patient outcome relies on early recognition and treatment, including an early consultation with a neurosurgeon for children with high-risk injuries.

As with any traumatic injury, initial management involves stabilization of the cervical spine, airway, breathing, and circulation. If clinical conditions warrant, ET intubation should be implemented. Proper sedation and analgesia should be administered to prevent spikes in ICPs.

With manual ventilation of the child, hyperventilation should be avoided. Only in impending herniation should short, intermittent episodes of hyperventilation be used. Although routine hyperventilation is not recommended for treatment of increased ICP, it may be used emergently to transiently decrease cerebral blood flow and blood volume. The benefit of creating “more room in the vault” is short lived because decreased blood flow causes hypoxia to already injured brain tissues.

Any source of bleeding in the head, including the brain, face, mandible, and scalp, can cause significant blood loss in the pediatric patient. Hypovolemia is of great concern, particularly in infants, who have a proportionally larger amount of blood volume in their heads. Even in older children, an open scalp laceration can be a significant source of blood loss that needs to be stopped while administering aggressive fluid resuscitation. The treatment goal is euvolemia because cerebral perfusion pressure depends on the MAP (CPP = ICP – MAP). Maintaining the patient’s head at 30° and midline also improves venous outflow from the head.

The child’s mental status may be altered for a number of reasons. The most life-threatening situation involves herniation syndromes in which increased pressure in the cranial vault causes the brain to shift. The brain may be shifted laterally or down through the foramen magnum. Possible treatments may include short-term hyperventilation or use of an osmotic diuretic (mannitol, 0.5 to 0.75 mg/kg/dose) or a loop diuretic (furosemide) to decrease the volume of water or cerebrospinal fluid (CSF) in the cranial vault.

Spinal Cord Injuries

Of all children who experience traumatic injuries, 3.8% have an injury to the spine and/or spinal cord. Although this is a relatively small incidence, mortality related to spinal cord injury in children is more than twice that in adult populations. Serious neurologic injuries are more common in older children and are often associated with sports injuries, diving injuries, or falls. The incidence of spinal cord injury dramatically increases in adolescents between 15 and 18 years of age. Of all sports-related injuries and reported spinal cord injuries, 30% to 50% involve the cervical spine and result in quadriplegia and/or severe disability.

Injuries in children younger than 8 years are most commonly found in the occiput (C1) to C3 area, and older children and adults tend to have injuries in the lower C-spine **Figure 23-25**. Because of the anatomic spine differences between adults and children, children are more prone to sustain cervical spine injuries of greater severity. By comparison, older children and adults incur injuries that are more evenly distributed throughout the cervical and thoracic spine.

Similar to brain injury, spinal cord injury may occur with a primary insult and then a secondary insult. The primary insult occurs from hyperflexion (most commonly), extension, rotational injury, or compression injuries. The secondary injuries occur from biochemical reactions, including oxygen-free radical production that damages endothelial cells within the brain, ion fluxes involving extracellular potassium, ischemia, or edema.

The most important intervention in the treatment of children with known or suspected spinal cord injury is proper immobilization. Initially, the child's cervical spine is secured manually while the airway is being assessed. Rigid infant and pediatric collars must be available in a range of sizes to ensure use of the proper size. A properly fitting cervical collar prevents flexion, extension, and head rolls or blocks prevent lateral head movement **Figure 23-26**. Ideally, pediatric backboards should be used because they allow for the relatively large size of the child's head by providing an occiput cut-out or head well. If a pediatric backboard is not available, the child may be placed on the standard backboard and neutral neck extension is provided by padding the child's torso to lift it relative to the head. The child should be placed on the backboard using a log roll technique **Figure 23-27**. One provider maintains manual stabilization of the head and neck while a minimum of two providers log roll the child. Care is taken to control movement of the torso completely.

When the child is confirmed to be in a neutral position, the child is secured to the backboard. Secure the child with straps over a minimum of three anatomic points, such as the thighs, pelvis, and shoulders. Care should be taken not to secure the child with straps over the diaphragm and abdomen because the straps could interfere with the muscles needed for breathing and may increase the potential for vomiting. Also, the shoulder straps need to be placed over the child's shoulder girdle and not the chest because this will also interfere with respirations. An infant would be secured with a pedi-mate or other type of restraining device.

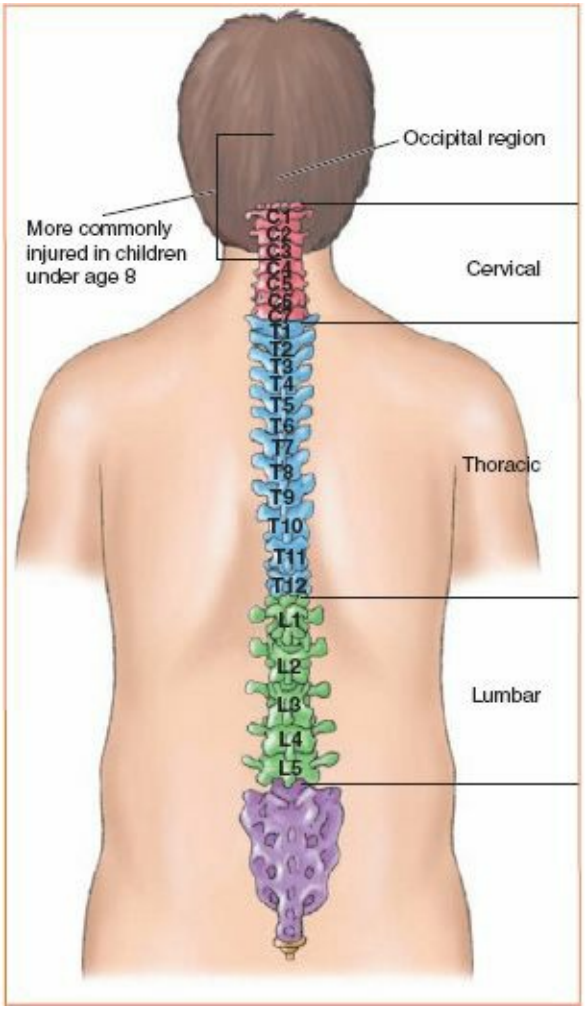

Figure 23-25 Injuries in children younger than 8 years are most commonly found in the occiput region to C3 area. Older children and adults tend to have injuries in the lower C-spine.

Figure 23-26 Use a properly fitting cervical collar and head rolls or blocks to stabilize the pediatric patient's spine.

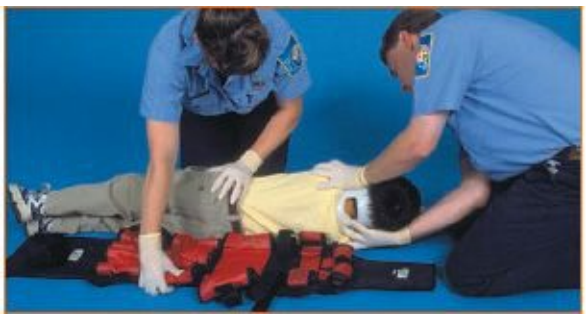

Figure 23-27 Place the child onto the backboard or immobilization device using the log roll technique.

After the child's body is secured, the head is secured with tape, straps, and head rolls. After the head is secured to the backboard, manual stabilization of the cervical spine may be released. Reassess the child's neurologic status, including gross motor and sensory function. Frequent assessment must be made during transport to detect the possibility of ascending lesions. For example, an older child may initially have a cervical spine injury at C6, thereby having no respiratory involvement. During transport, the C6 lesion could ascend up to the level of C3. This progression of the spinal cord injury would now affect the child's ability to maintain respiration.

Methylprednisolone has been used clinically to minimize the progression and treat the inflammation of spinal cord injuries. Limited studies demonstrate documented benefits; the treatment carries a low risk, however, and any benefit obtained is desired. The drug regimen includes 30 mg/kg IV over 15 minutes followed in 45 minutes by a continuous infusion of 5.4 mg/kg/h for 23 hours. Initiation of this treatment should be within the first 8 hours to improve the chance of neurologic recovery.

Because children have relatively weak spinal ligaments and increased mobility of the spine, they may sustain damage to the spinal cord and spinal ligaments without damage to the spinal vertebrae. This damage is called **SCIWORA**, which is an acronym that stands for spinal cord injury without radiographic abnormalities. SCIWORA should be suspected when there are clinical signs or symptoms of spinal cord injury in the presence of normal cervical spine radiographs. This condition is often associated with a poor neurologic outcome, a high proportion of complete neurologic injuries (those in which there is no sensory or motor function below the level of injury), and a high susceptibility of delayed neurologic deficit. If a child reports pain, has difficulty with range of motion, or is unresponsive, spinal immobilization should remain in place until additional studies can confirm no injury or until a skilled pediatric specialist clears the spine.

Transport Management

Spinal Cord Injuries

- Manually secure the child's cervical spine while assessing the airway.
- Immobilize the child on a pediatric or standard backboard; secure the child's body first, then the head.
- Release manual stabilization of the cervical spine.
- Reassess the child's neurologic status, and continue to assess frequently during transport.

Breathing Abnormalities

Trauma and the effects of trauma may also cause upper airway obstruction. Facial trauma and soft-tissue swelling can block the upper airway. There may be direct trauma to the upper airway in the example of hanging injuries or "clothes-line" injuries, or there may be secondary injury related to the trauma. Hanging injuries occur in the toddler population from crib accidents and window cords. Hanging injuries may also occur in adolescents as a method of suicide, or during a "choking game," in which an individual at the point of near strangulation escapes to enjoy an altered mental state. A clothes-line injury is most common in ATV (all-terrain vehicle) crashes, and occurs when the patient in motion is struck in the anterior neck by a stationary object (ie, a clothes-line wire).

Head injury may cause a decrease in the level of consciousness and result in the patient's tongue

obstructing the airway. Life threats that may be encountered include simple pneumothorax, tension pneumothorax, open pneumothorax, hemothorax, and flail chest. Flail chest is unusual in pediatric trauma because rib calcification is poor, except in adolescents.

Management of these injuries in the pediatric population is usually exactly the same as with adult patients. The following sections discuss only the differences in managing these injuries in pediatrics. For a complete discussion of these injuries, refer to Chapter 10.

Transport Management

Tension Pneumothorax

- For older children and adolescents, insert a 14- to 16-gauge angio-needle in the second to third intercostal space, midclavicular on the affected side.
- For infants, insert a 21- to 23-gauge butterfly needle anteriorly at the fourth intercostal space. Attach to a stopcock and a 20-mL syringe. Empty the chest of air or fluid until the infant's condition improves.

Tension Pneumothorax

Treatments for tension pneumothorax include a 14- to 16-gauge angio-needle decompression for older children and adolescents in the second to third intercostal space, midclavicular on the affected side **Figure 23-28**. For infants, treatment is a 21- to 23-gauge butterfly needle inserted anterior at the fourth intercostal space (nipple line). The needle is then attached to a stopcock and a 20-mL syringe. The chest is emptied of air or fluid until the infant's condition improves.

Hemothorax

Treatment for a hemothorax includes inserting an appropriately sized chest tube in the fifth intercostal space, anterior, and midaxillary. Ideally, the chest tube is attached to a fluid collection or suction system that allows for autotransfusion. Large amounts of blood can accumulate within the pleural cavity; as long as the blood does not become contaminated by an open wound, it can be returned to the child's circulation. This relieves the concern about draining too much blood from the pleural space. If autotransfusion collection is an option for the CCTP, then it should be used to reduce the risk of donor transfusions. If autotransfusion collection is not possible, there is controversy in the literature regarding how much blood should be accumulated before clamping the thoracostomy tube. Advanced Trauma Life Support® (ATLS) guidelines state that approximately 20% of total blood volume is suggested. Pediatric patients have a circulating blood volume of 80 mL/kg. Clamping the chest tube is a temporary measure until an open thoracotomy is performed. Adequate fluid resuscitation with blood or crystalloids, without overload, must also be considered to prevent and treat the impending hypovolemic shock.

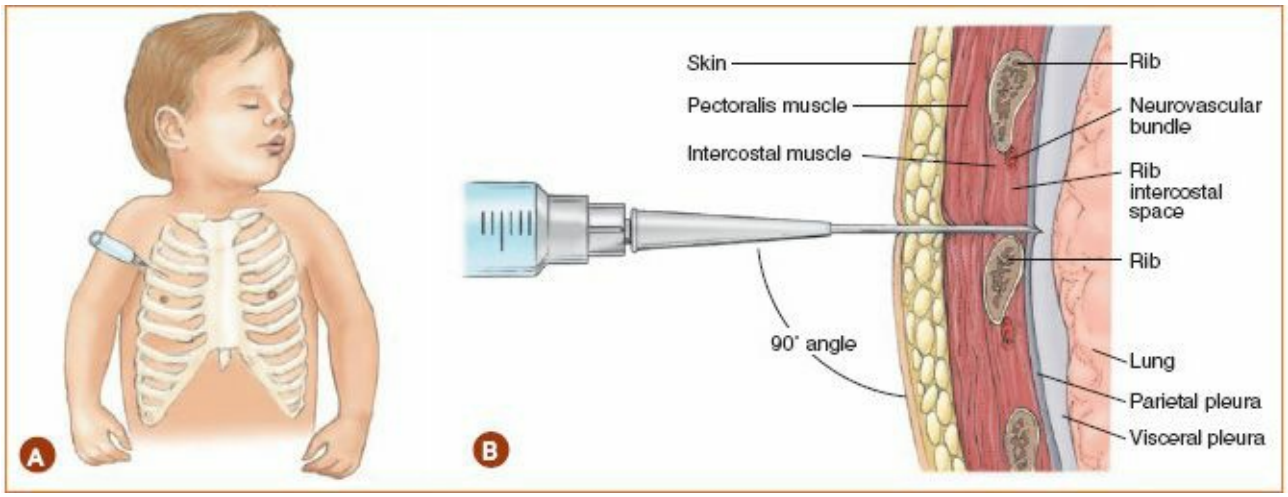

Figure 23-28 Decompression of a tension pneumothorax. **A.** The needle should be inserted into the second or third intercostal space, midclavicular on the affected side. **B.** Insert the needle at a 90° angle and listen for the release of air.

Transport Management

Hemothorax

- Insert an appropriately sized chest tube in the fifth intercostal space, anterior, and midaxillary.
- If available, attach the chest tube to a fluid collection or suction system that allows for autotransfusion.
- If autotransfusion collection is not possible, follow ATLS guidelines regarding how much blood should be accumulated before clamping the thoracostomy tube.
- Fluid resuscitation with blood or crystalloids may be necessary.

Circulation Abnormalities

Life threats related to circulation that must be assessed include bleeding, hypovolemia, arrhythmias, and pericardial tamponade. Hypovolemia and arrhythmias were discussed earlier in this chapter, and bleeding management follows the same principles and procedures learned in earlier training. Pericardial tamponade is a hemodynamic emergency, usually the result of a blunt or penetrating injury to the chest and, more specifically, the sternum.

Acute pericardial tamponade is assessed clinically, typically with a history of trauma, tachycardia, hypotension, jugular venous distention, and muffled heart tones. Muffled heart tones may be difficult to assess in the transport setting. Treatment in the field, pericardiocentesis, may also be challenging. Pericardiocentesis treatment involves inserting a 14-to 20-gauge IV or spinal needle to a syringe. The needle is inserted just inferior to the xiphoid process and advanced slowly toward the left shoulder. When the needle pierces the pericardial sac, blood is aspirated. Removing as little as 15 to 20 mL of blood may improve the patient's condition, because it allows improved cardiac filling and cardiac output. This intervention may be beyond the scope of some CCTPs depending on protocol. It is further recommended that this procedure not be done in a moving ground or air vehicle. The appropriate treatment during transport is a rapid IV fluid bolus (20 mL/kg of a crystalloid solution for a total of three infusions, then blood products or colloids at 10 mL/kg). An IV fluid bolus greatly increases preload and improves cardiac filling and cardiac output.

Transport Management

Circulation Abnormalities

- Administer a rapid IV fluid bolus of a crystalloid solution for a total of three infusions, followed by blood products or colloids.

Fractures

As mentioned earlier, children have less calcified bones. The immature bones of children are more porous than those of adults and tend to respond to kinetic forces by buckling rather than fracturing. Therefore, if the forces are great enough to cause a fracture, investigation of underlying organ injury is warranted. However, with reduction of misaligned structures and immobilization by either the physician or the CCTP (depending on agency protocol and extremity vascular status), most of these injuries have excellent outcomes.

The ligaments in children are stronger and more resilient to tensile forces, thereby providing strength to the joints. As mentioned earlier, this is the reason that dislocations are more rarely found in pediatric populations compared with adult populations.

The pediatric spine has incomplete ossification and epiphyseal growth plates and may have hypermobility. As a result, the cervical spine in children is highly flexible and young children tend to have avulsions rather than fractures. However, this hypermobility does not always protect the child's cervical spine. Compounding factors are that children have relatively large, heavy heads with weak neck muscles. Rapid deceleration injuries resulting in hyperflexion and torsion forces are most commonly seen in children.

Pelvic fractures can be responsible for massive internal hemorrhaging that is difficult to quantify and will lead to profound deterioration of the pediatric patient.

Although they are falling out of fashion, the PASG is one option for stabilizing a pelvic fracture for transport. Depending on the CCTP's protocols, a sheet can be wrapped around the pelvis or a commercial device known as a SAM splint can be used to stabilize an unstable pelvis and prevent further vascular and neurologic damage while en route to the trauma center. Remember that with pelvic fractures, a significant amount of blood can be located in the retroperitoneal cavity. The CCTPs must be prepared for a rapid decline in the patient's condition as a result of this blood loss and have crystalloids and colloids available.

A femur fracture, although rare in young children, is an injury that can lead to a significant amount of blood loss in the pediatric population **Figure 23-29**. Older children and adolescents are most at risk for femur fractures. The two most common causes are motor vehicle collisions and pedestrian injuries. For a femur fracture in the very young child who has not started walking, traction splinting may not be necessary as a result of the underdeveloped quadriceps muscles. In children, these muscles will not contract and cause the pain commonly seen in older children and adults. Other techniques for immobilization for transport include an air splint, rigid splint, PASG, or simple padding using pillows or blankets **Figure 23-30**. The ultimate goal is patient comfort; remember crystalloids for fluid replacement of a 20-mL/kg bolus and of a 10-mL/kg colloid bolus, as well as adequate pain management with analgesics and sedatives.

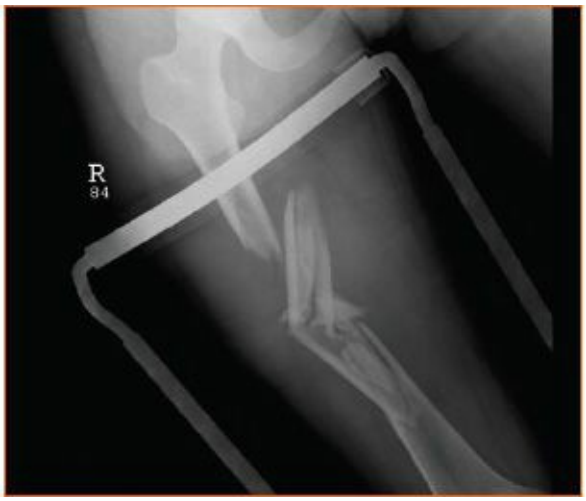

Figure 23-29 Radiograph of a femur fracture.

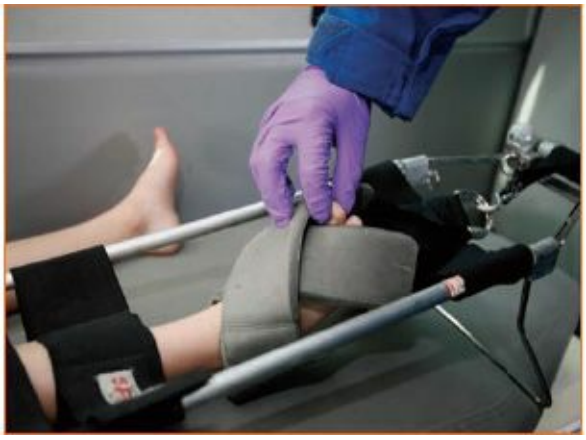

Figure 23-30 Transporting a child with a femur fracture can be accomplished using a portable traction splint like the one pictured. Distal sensory and motor function should be checked frequently during transport of these patients.

Transport Management

Fractures

Pelvic Fracture

- Stabilize the pelvic fracture.
- Be prepared to administer crystalloids and colloids if necessary.

Femur Fracture

- Immobilize the injury.
- Administer analgesics and sedatives as necessary for pain management.
- Administer crystalloids and colloids for fluid replacement.

Figure 23-31 With orthopaedic injuries from motor vehicle crashes, children experience similar injury patterns as adults.

Orthopaedic Injuries

Anatomic differences in relation to injury patterns are the main differences when dealing with the pediatric population and orthopaedic injuries. In vehicles, children younger than 8 years or weighing less than 80 lb should be in some sort of weight-appropriate child restraint system. These seats are designed to decrease morbidity and mortality when used correctly. Because newer vehicles are equipped with front passenger airbags, current National Highway Traffic Safety Administration recommendations (and laws in many states) require that children in this group occupy the rear seat of the vehicle (if equipped). A child who weighs less than 40 lb, depending on the make and model of the car seat, must be restrained in a 4-point harness. The goal of the booster seat is to protect the older child from damage caused by the seatbelt being improperly positioned over the child's shoulder. The booster does what it says; it boosts the child up so that the vehicle's seatbelt is in the correct position across the child's chest and resting over the clavicle. The CCTP should still be aware that children will experience similar injury patterns as adults **Figure 23-31**. These seats are designed to decrease injury but not prevent injury completely.

The main differences with regard to a pedestrian crash of an adult vs a child are the injury patterns that will be seen. As discussed in Chapter 10, an adult's primary injuries are to the pelvis and lower extremities; however, the pediatric patient's most common injuries involve the head and chest. A child is commonly run over by the vehicle or thrown by the impact. It is important for the CCTP to find out what occurred and be alert for these injury patterns.

Burns

Burns are the second most common cause of accidental death in children, second only to injuries from motor vehicle collisions. The peak incidence of burn injuries occurs in children between 1 and 5 years of age. Most of these injuries involve burns from scalding liquids. The incidence of house fires has decreased in recent history, but they still account for half of all burn-related deaths. Other sources of burns include chemical, electrical, and contact burns. Children have a higher mortality rate than adults for the same total body surface area and severity of the burn injury **Figure 23-32**.

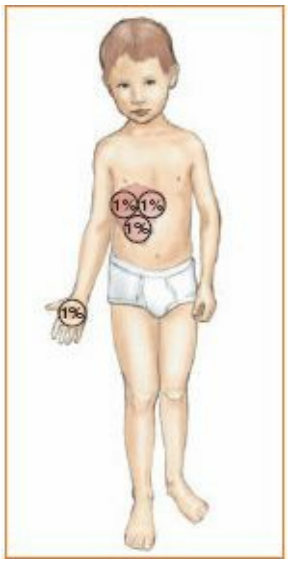

Figure 23-32 Use the size of the child's palm to estimate the burned body surface area. The palm generally represents 1% of the child's body surface area.

Direct thermal, chemical, or electrical injury causes cell death. There is immediate release of tissue local mediators such as histamine. These mediators are responsible for the profound increase in vascular permeability. This vascular permeability is further increased by the release of serotonin and prostaglandins. As the intravascular volume moves into the interstitial spaces, profound peripheral edema is noted. This edema not only promotes tissue ischemia to already damaged tissue, it also causes a relative hypovolemia within the intravascular space. Left unresolved, profound hypovolemic shock ensues for patients experiencing moderate to severe burns. The final depth of a burn may be delayed for 4 to 5 days because the ischemic injury may extend 3 to 7 times more deeply than the level of the original injury.

Regardless of the degree of burn, initial burn therapy always begins with removing the source of the burn. Prompt removal of burning or scalding clothing should occur. Remove constrictive jewelry such as rings, earrings, or necklaces.

Small area burns may be initially cooled with tepid water for a few minutes only and then a moist dressing may be applied. However, larger surface area wounds should have dry sterile dressings applied, because the risk of hypothermia is high in pediatric burn patients. Skin integrity is lost as a result of the burn, thereby eliminating one purpose of the skin, which is to maintain body temperature. In addition, recall that children have a large body surface area, leaving more area exposed for cooling.

Chemical burns should be copiously rinsed with warm water for a minimum of 20 to 30 minutes or until the skin no longer has a "slippery" feel to it. While you are irrigating a chemical burn, ensure that the chemical is not spread to other body areas of your patient or to yourself.

Initial burn assessment includes an evaluation of airway issues related to facial and laryngeal edema. Even if no body surface burns are visible, inhalation burns can be responsible for rapid loss of the airway. Assess for singed eyebrows, nasal hairs, or carbon material in the oral or nasal airways. Early intubation is usually the treatment of choice because progressive edema increases breathing difficulty and loss of airway control.

Breathing concerns for burn patients include injury to the lungs related to a direct thermal burn or injury related to the indirect effects of smoke inhalation and CO production. In addition, significant burns to the chest, especially circumferential burns, may impede chest movement during respiration; 100% oxygen is administered to all children with a chance of smoke inhalation. Carbon monoxide toxicity may lead to severe neurologic deficits as the carboxyhemoglobin level rises. CO will displace oxygen on the

hemoglobin molecule; 100% oxygen administration cuts the CO level in half every 30 minutes.

Circulation concerns result from dramatic increased vascular permeability and fluid losses from the intravascular space. Methods for replacing depleted volumes of fluid should be based on the practices and standards of the local referral burn center, and collaborative guidelines should be discussed and in place before encountering the care of a burned child. As discussed in Chapter 12, the Parkland formula may be used (4 mL/kg per percentage total body surface area administered over the first 24 hours; half of the total fluids are administered over the first 8 hours, and the remainder are administered over the next 16 hours).

After initial life threats are addressed, the CCTP may consider other treatments or procedures, including insertion of an indwelling urinary catheter because this will be the primary means of assessing urine output, assessing for myoglobinemia (port-wine-colored urine), and measuring the success of fluid resuscitation. For the child with suspected inhalation injuries, a beta-2 agonist medication such as albuterol (2.5 to 5.0 mg via hand-held nebulizer) may be warranted for bronchospasm and/or wheezing.

Transport Management

Burns

- Remove the source of the burn.
- Cool a small area burn with tepid water for a few minutes, then apply a moist dressing.
- Rinse chemical burns copiously with warm water for a minimum of 20 to 30 minutes or until the skin no longer has a slippery feel.
- Assess airway tissues and look for singed eyebrows, nasal hairs, or carbon material in the oral or nasal airways.
- Administer 100% oxygen to children who may have inhaled smoke.
- Administer fluids based on the practices and standards of the local burn center.
- Insert an indwelling urinary catheter after initial life threats have been addressed.
- Wrap the burn in dry, sterile gauze. Follow agency policies and procedures regarding applying a topical antimicrobial agent to the burn dressing.
- Cover the child with a warm blanket.
- Administer analgesia and sedation for pain.
- Monitor the patient's pain scale, and respiratory, hemodynamic, and mental status, during transport.

Wound care involves wrapping the burn in dry sterile gauze. For large surface area burns, a sterile burn dressing or trauma dressing may be used. Warmed blankets may then be used to maintain or improve the child's body temperature. The application of a topical antimicrobial agent to the burn dressing is controversial for transport. Often, the burn center team prefers to have an unobstructed view of the patient's burns and will apply the antimicrobial agent after patient evaluation and debridement. Refer to your own specific system policies and procedures.

Most burns are extremely painful, and adequate analgesia and sedation must be delivered to these children. Morphine sulfate should be administered at a dosage of 0.1 to 0.2 mg/kg IV/IO for analgesia while the CCTP monitors the patient's pain scale and respiratory, hemodynamic, and mental status while en route to a burn center for definitive treatment. Continued reassessment and medication administration should occur.

Abuse and Neglect

The incidence of child abuse and neglect has unfortunately been regarded as an epidemic in the 21st century. The probability of transporting such a victim of these horrific events is common in this profession. The primary responsibility of the transport crew is to their own safety first, and then to provide a safe environment for their patient. Forms of abuse that the CCTP must be aware of while caring for the pediatric population include psychological (emotional) abuse, physical abuse, economic (financial) abuse, sexual abuse, and neglect.

When there are no obvious signs of abuse, such as bruises or broken bones **Figure 23-33**, the patient may have suffered neglect. This is the intentional or unintentional omission of needed care and support, which may present as a child who appears unkempt, appears malnourished, or has recently been diagnosed as having failure to thrive. A key component of the physical assessment at the bedside is the child's behavior. Children younger than 6 years who have experienced neglect usually appear markedly passive to their environment, and children 6 years or older may seem aggressive on initial evaluation.

Signs and Symptoms

Neglect

- Child appears unkempt, malnourished, or has a diagnosis of failure to thrive
- Younger than 6 years: Child is markedly passive to the environment
- Older than 6 years: Child seems aggressive

Transport Management

Abuse and Neglect

- Conduct a thorough assessment.
- Adhere to agency protocol regarding reporting of suspected abuse or neglect.
- Document the events and transport details.

The transport crew should conduct a thorough assessment and gather all necessary transfer documentation and radiographic studies for the receiving hospital. If a detailed assessment cannot be completed based on the response of the pediatric patient, this should be noted in the transfer documentation. As with any patient population, if the provider's assessment and knowledge of age-appropriate behavior do not correlate, and a suspicion of abuse or neglect is present, then the CCTP must adhere to agency protocol regarding the reporting of suspected abuse or neglect. In the case of confirmed abuse, all measures should be taken to preserve evidence. Transfer documentation must reflect findings from the physical exam and statements made by the victim or suspected perpetrator. CCTPs may be called to testify in court regarding such cases, so it is imperative to document the events and transport details with the utmost completeness and clarity.

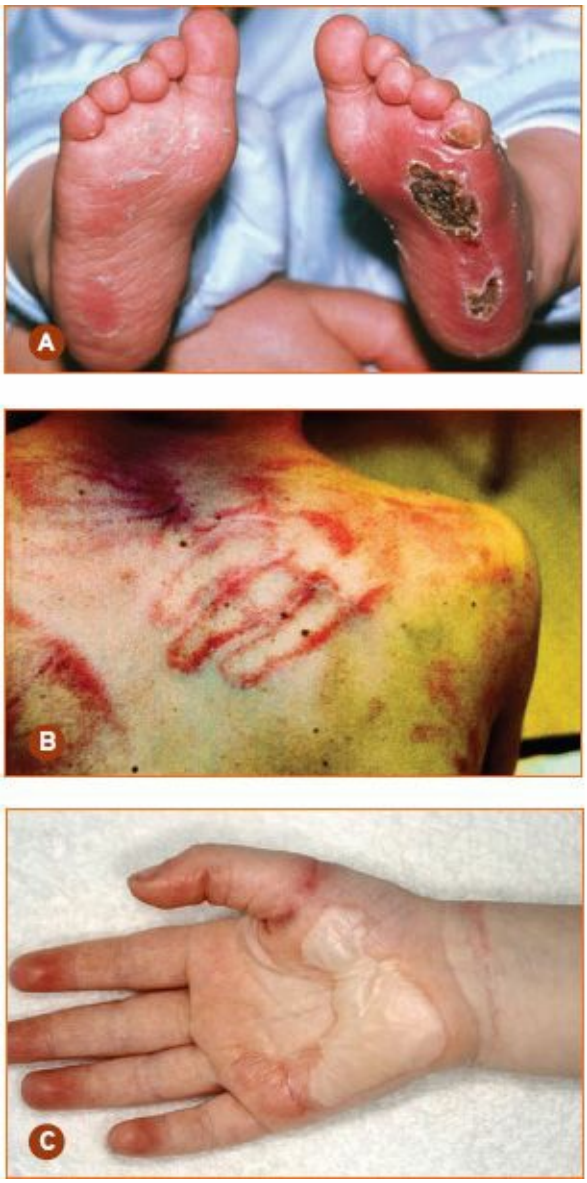

Figure 23-33 Obvious signs of abuse. **A.** Burns like these on the bottom of the feet are highly suggestive of abuse. **B.** Imprints of a hand on the skin suggest assault or other forms of abuse. **C.** Burns on the palm in susceptible patients can be signs of abuse.

Environmental Emergencies

Hypothermia

Hypothermia exists when the patient's body core temperature is 95°F (35°C) or less. It can result from a systemic disease process, prolonged environmental exposure, or when heat production is less than heat loss by convection, conduction, or radiation. The pediatric population is at an increased risk for hypothermia secondary to their relatively larger body surface area, proportionally large head, immature temperature-regulating mechanisms, decreased energy stores, and less body fluid volume and protective fat. Low-birth-weight (premature) infants, newborns, and children involved in trauma are at high risk for hypothermia.

The management of these patients starts by removing them from the cold environment, removing wet clothing, placing under a heat lamp, and providing warm blankets and liquids **Figure 23-34**. As with any patient, the ABCs must be addressed during the initial/primary assessment. Warm humidified oxygen and warmed IV fluids should be administered based on the level of hypothermia. Rapid fluid replacement in this population may in turn cause or increase the state of hypothermia. Regardless of the outside ambient

temperature, the CCTPs should provide additional heat from the vehicles' (rotor wing, fixed wing, or ground) heaters. In the event of severe hypoglycemia, the incidence of cardiopulmonary arrest may occur. Cardiac medication and defibrillation may not be effective when body core temperatures are below 82.4°F (28°C); therefore, CCTPs must adhere to their agency protocols or resuscitation guidelines (Neonatal Resuscitation Program, Pediatric Advanced Life Support).

Figure 23-34 To manage a hypothermic pediatric patient, remove wet clothing, place him or her under a heat lamp, and provide warm blankets and liquids.

Transport Management

Hypothermia

- Remove the child from the cold environment.
- Remove wet clothing.
- Place the child under a heat lamp.
- Provide warm blankets and liquids.
- Address the ABCs.
- Administer warm humidified oxygen and warmed IV fluids based on the level of hypothermia.
- Provide additional heat from the vehicle.
- Follow agency protocols regarding cardiac medication and defibrillation.

Heat Stroke

Heat stroke is classified as a life-threatening emergency in the field with morbidity rates ranging from 17% to 70%. Heat gain results from a mixture of metabolic activity, the environment (high ambient temperature and humidity), and vigorous exercise (exertional heat stroke). This gain is counteracted by several mechanisms, such as conduction, convection, evaporation, and radiation. The pediatric population acclimates more slowly to heat production from exercise than the adult populace. Such variables as a greater skin surface area, decreased sweating capacity, and the inability to shunt heat via the blood from the core to the periphery make the pediatric population more susceptible to this medical emergency. Heat stroke is the result of the body's compensatory heat dissipation system (sweating, radiation, and convection) being overwhelmed by heat production and absorption. Underlying conditions may contribute to this imbalance, which include, but are not limited to, the following: immature thermoregulation of the newborn, burns, and cystic fibrosis.

The classic signs of heat stroke are hyperpyrexia with rectal temperatures of greater than 104°F (40°C); skin that is red, hot, and dry to the touch; severe CNS dysfunction with complaints of headache

and confusion, leading to coma and posturing; tachycardia with hypotension; severe dehydration; nausea and vomiting; hypoglycemia; and polyuria with electrolyte imbalances. The patient may or may not be sweating, depending on the degree of fluid loss.

It is imperative that the management of these patients start in the field by removing the patient from the environment and into an air-conditioned vehicle. On initial contact with the patient, the clothing should be removed or drenched in cool water (conductive heat loss), and the active cooling process should be started per agency protocol. Ice packs can be placed around the neck, axilla, and groin. If time permits during the transport, attempt to fan the patient with misting water until the body temperature reaches 101.3°F (38.5°C) (rectal). IV fluid replacement should be based on the patient's presentation, cardiac status, and laboratory results. Be prepared to administer 0.9% normal saline at a rate of 5 to 20 mL/kg over 20 to 60 minutes depending on the patient's hemodynamic status. If intractable seizures or muscle cramps are noted, a hypertonic solution (3% saline) may be ordered to administer while en route to the receiving facility. The initial dose of 3% saline solution is 5 mL/kg, and additional dosages should be infused over the next 4 to 6 hours. If the patient begins to shiver, diazepam, 0.2 to 0.3 mg/kg IV, may be administered depending on protocol. The patient's cardiac status (arrhythmias) and neurologic status (GCS score) should be closely monitored continuously for any changes while en route to the receiving faculty.

Signs and Symptoms

Heat Stroke

- Rectal temperature greater than 104°F (40°C)
- Skin that is red, hot, and dry to the touch
- Severe CNS dysfunction with complaints of headache and confusion, leading to coma and posturing
- Tachycardia with hypotension
- Severe dehydration
- Nausea and vomiting
- Hypoglycemia
- Polyuria with electrolyte imbalances

Transport Management

Heat Stroke

- Move the patient into an air-conditioned vehicle.
- Remove the child's clothing or drench the child in cool water.
- Start the active cooling process per agency protocol.
- Place ice packs around the neck, axilla, and groin.
- If time permits, fan the child with misting water until the body temperature reaches 101.3°F (38.5°C) (rectal).
- Administer IV fluids, hypertonic solution, or diazepam, if necessary, according to agency protocol and medical orders.
- Monitor the patient's cardiac and neurologic status for changes during transport.

Drowning

Drowning is a top cause of death for the pediatric populations between the ages of 1 and 14 years. Over the past few years, the amount of education has increased to alert the general public about the hazards of drowning in pools and lakes for our pediatric population. However, there is still a lack of awareness about bathtubs and buckets because a child can drown in as little as an inch of standing water.

Figure 23-35 The diving recovery position should be used when treating patients with cold water drowning.

The primary insult is hypoxemia that may or may not be attributed to aspiration. Wet drowning accounts for 85% of incidents. Hypoxemia as a result of aspiration begins the insult to these patients, and is followed by metabolic acidosis, hypothermia, and cardiac compromise. The key to a successful outcome is good resuscitation and a brief submersion. Secure the airway early, provide positive-pressure ventilations, and add PEEP early. Rewarm the patient and work to reverse metabolic acidosis. These patients might be on fluid restriction per the physician or at least half maintenance infusions, and furosemide might be needed. There is a possibility that vasopressors might also be needed. Antibiotics are only needed if a bacterial infection is suspected. Patients can experience a wet drowning incident in fresh or salt water, although the exact etiology does not change the initial treatment. In recent research, it is believed the mammalian dive reflex occurs mostly with sudden submersion in extremely cold water **Figure 23-35**.

Approximately 10% to 15% of drowning incidents are related to dry drowning. Dry drowning occurs when the patient has a laryngospasm that prevents water from entering the lungs. The duration of the laryngospasm determines the extent of the hypoxemia. Death from dry drowning is the result of asphyxiation vs aspiration.

Patients With Special Needs

Children with special health care needs may be even more of a challenge in the transport environment. With all the advances in the care of the critically ill child and in neonatal resuscitation (with respect to babies being born and surviving as early as 24 weeks), the frequency of a CCTP having to take care of a special needs child is increasing. These children are being sent home with more complex equipment, such as ventilators, apnea monitors, nagogastric tubes, gastrostomy tubes or buttons, jejunostomy tubes, and ventriculoatrial or ventricu-loperitoneal shunts. It would be difficult for the CCTP to be familiar with every different illness or congenital disease seen in the pediatric community, so parents and caregivers are the best resource for understanding their child's illness and using the specialized equipment. **Figure 23-36** and **Figure 23-37** show a special needs patient with a tracheostomy tube and a feeding tube, respectively.

Figure 23-36 A special needs patient with a tracheostomy tube.

The CCTP might encounter special needs children either on a transport to the hospital before surgery or on a transport back to a tertiary hospital after surgery. Children with congenital heart disease, for example, might be sent to a large hospital for the first of multiple operations and then sent back home. The CCTP might also encounter these children if equipment malfunctions or as the time nears for the child's next surgery.

If your protocols and the child's condition allow, it might be possible to let the parent or caregiver assist you with care during transport. Sometimes having the parent suction or supply information about the child's illness can calm the parent and the patient, and also decrease anxiety they may feel regarding the child's hospital stay.

Flight Considerations

There are no specific flight considerations for pediatric critical care transport. The same flight considerations presented in other chapters apply with the pediatric patient. A key point to remember in all transports is that children are not small adults. The CCTP should be familiar with age-specific development and deviation from the norm; for example, pediatric patients will quickly decompensate without warning. Also remember that "all that wheezes is not asthma" and that cardiopulmonary resuscitation is usually caused from a respiratory, not a cardiac, etiology.

Figure 23-37 A special needs patient with a feeding tube.

In most circumstances, a parent/caregiver should be offered the opportunity to be transported with the patient, thus putting the pediatric patient at ease with the environment. As with adult patients, never lie to pediatric patients. Use every opportunity to build their trust.

Summary

The emergency care of children is challenging and difficult under the best of circumstances. Well-guided, standardized preparation for the transport of a critically ill or injured child is essential. Working collaboratively with various organizations, such as Emergency Medical Services for Children and pediatric components of emergency medical professional organizations, allows the CCTP to provide appropriate care to this vulnerable population.

Case Study

IT IS 1:00 AM AND THE PARENTS of an 11-year-old boy call EMS because he is "sucking for air."

EMS arrives on the scene 6 minutes later and immediately completes an initial/primary assessment, establishes that he has no known allergies, and begins treatment with an albuterol nebulizer. The boy is a cooperative and responsible child, but the parents have been noncompliant with administering his rescue inhalers and nebulizers. He is alert and cooperative but has audible wheezing with intercostal, subcostal, and substernal retractions with tracheal tugging. The cardiac monitor reveals sinus tachycardia with a rate of 118 beats/min, a blood pressure of 110/62 mm Hg, respirations of 36 breaths/min, and an initial SpO_2 of 84% on room air. En route, IV access is obtained. During that time, the SpO_2 rapidly increases to 94% and his work of breathing begins to decrease.

At 1:18 AM, the patient arrives at the local emergency department (ED) and care is transferred to the ED nurse and ED physician. A physical examination reveals a patent airway, but he has thick secretions that he cannot cough up. Breathing is rapid and forced. He continues to have retractions and an SpO_2 of 95% while on the nebulizer. Diminished breath sounds with expiratory wheezing are auscultated. Pulses are bounding, the skin is pale pink and diaphoretic, and capillary refill is 3 seconds centrally and peripherally. He is cooperative but very fatigued.

At 1:25 AM, he is started on a continuous albuterol/ipratropium nebulizer to a maximum of three doses, and he is given methylprednisolone and magnesium sulfate for management of his asthma. A chest radiograph is ordered. The ED physician calls the pediatric intensivist at the children's hospital for a possible transfer as a result of the child's history of asthma and previous intubations. The children's hospital accepts the patient to their pediatric floor, and you depart in a critical care ground ambulance.

Thirty minutes later, the patient's work of breathing is reported as "improving" and the chest radiograph shows the hyperinflation of the lungs typically seen in children with asthma. You arrive, begin assessment, and obtain a report from ED personnel.

At 2:02 AM, the patient becomes unresponsive and has an SpO_2 of 78%. You and your partner initiate bag-mask ventilation. You immediately prepare your equipment and intubate the child with a 6.5 ET tube.

After successful intubation and ET tube placement, confirmation with visualization of the tube passing the cords, fogging of the ET tube, and $ETCO_2$ monitoring, the child is given adequate pain medication sedation and paralytics, and is placed on a ventilator. His SpO_2 is now 93% and his vital signs are stable. You and your partner start an in-line nebulizer and prepare to move the patient to the stretcher for transport.

During transport, you observe that the child's stomach is distended with air.

At 2:21 AM, the child suddenly deteriorates as his SpO_2 decreases to 81% and his heart rate increases to 150 beats/min. The alarm on the ventilator is displaying "high pressure." The ventilator is disconnected, and manual ventilation with a bag-valve device is initiated.

A few minutes later, the patient's ET tube placement was reconfirmed, he was suctioned, and all equipment was working properly. He remains difficult to ventilate as a result of increased resistance, and the $ETCO_2$ increased to 60 mm Hg. Reassessment now reveals decreased breath sounds on the right side, jugular venous distention, and a decreasing blood pressure.

At 2:30 AM, you perform needle decompression on the patient's right side. There is a rush of air and resistance to bagging decreases. He is then placed back on the ventilator. His vital signs are blood pressure of 112/61 mm Hg and heart rate of 102 beats/min; he is being ventilated at a rate of 12 breaths/min. His SpO_2 is now 98% on an FIO_2 of 60%. He is resting comfortably under adequate sedation and pain medicine, and his condition is greatly improved. The rest of the transport is uneventful. You and your colleagues advise the receiving physician of the changes in the patient's condition during transport. All paperwork, treatments, and medications given are reported to the pediatric intensive care unit nurse with a copy of the transport report to be left with the patient's chart.

1. How was the ET tube size calculated?
2. What is the treatment priority after the patient is intubated and placed on the ventilator?
3. What is the treatment priority after the child deteriorates to an SpO_2 of 81%?
4. What mnemonic can be used initially to help address the acute change in your patient's status?
5. What procedure should be performed when you observe that the child's stomach is distended with air?

Analysis

Endotracheal tube size is calculated with the formula:

$$\frac{\text{Age} + 16}{4}$$

A length-based resuscitation tape, also called the Broselow tape, can also be used. Remember that ET tube size formulas are only guides for ET tube selection. An ET tube that is 0.5 mm smaller and larger must be readily available.

After the child is intubated and placed on the ventilator, the treatment priority is sedation and possibly pain medication. When you observe the patient's stomach distended with air, a nasogastric or orogastric tube should be inserted to decompress the stomach and reduce the risk of aspiration.

When the child deteriorates to an SpO_2 of 81%, the treatment priority is assessment of the ABCs. At this point, the CCTP should take the patient off of the ventilator and manually ventilate the patient with a bag-valve device.

The DOPE acronym is used to help remember how to care for this deteriorating patient. Recall that it stands for *Dislodgement* of the tube, *Obstruction* of the tube, *Pneumothorax*, and *Equipment* failures.

When the crew discovers a pneumothorax, the next treatment priority is needle decompression. In pediatric populations, the child may still have an equal rise/fall of the chest and audible lung sounds on both sides of the chest. Children typically have thin chest walls and lung sounds "echo" to the affected side from the unaffected side. In addition, the inflated lung may still elevate the side of the chest with the collapsed lung. Again, this is related to pliable and compliant chest walls.

Asthmatic children are at a greater risk for pneumothorax because of hyperinflation of the lungs and high airway pressures. Remember, in most cases, the air can get in, but it has difficulty getting out. Particular caution must be taken with children during bag-mask or mechanical ventilation. Slow down your expiration time for these patients. Some children may need I:E times of 1:3 to 1:5. Assess, treat, and *reassess*!

Prep Kit

Ready for Review

- The basic anatomy of the cardiovascular system is the same in children as in adults, but there are physiologic differences, such as differences in normal ranges for pulse rate and blood pressure and children's limited duration of compensatory mechanisms.
- Children's respiratory systems—lungs that continue developing through adolescence, smaller lower airways, smaller number and size of peripheral airways, immature alveoli, and decreased collateral ventilation—put them at higher risk for severe symptoms caused by respiratory diseases and for atelectasis.
- Infants' highly reactive airways make them more vulnerable to environmental allergens, viral respiratory diseases, and hereditary factors.
- Children are more susceptible to brain injuries because their brains are not well compartmentalized, which means there is more room for the brain to move in the skull.
- A normal fontanelle is soft and flat but has a feeling of fullness. The posterior fontanelle closes at 2 months of age, and the anterior fontanelle closes at 16 to 18 months of age.
- Cephalohematoma occurs in 1% to 2% of live births involving mechanical trauma, and is found predominately in the parietal region of the skull. It usually resolves in 6 to 8 weeks and can be associated with linear fractures. The CCTP should note the size and location of the hematoma during initial assessment and during transport. Possible complications are rare but include infection, hyperbilirubinemia, meningitis, and osteomyelitis.
- Caput is soft-tissue swelling that usually resolves within 24 hours.
- Children's bones are more porous than adults' bones, and tend to buckle rather fracture. Epiphyseal-metaphyseal growth plate injuries account for 10% to 15% of fractures in children; with reduction and immobilization, most have excellent outcomes.
- The child's thorax is pliable and able to absorb a significant amount of energy without fracturing. A small or suspected rib fracture may indicate a severe pulmonary contusion, which is more life threatening to the child than the rib fracture.
- Children's ligaments are strong and more resilient than those of adults, making the joints strong and dislocations rare.
- Because of a highly flexible cervical spine, young children tend to have avulsions rather than fractures. They are more prone to head injuries because they have relatively large, heavy heads with weak neck muscles.
- Children have a higher risk of aspiration as a result of an immature cardiac sphincter, are more susceptible to blunt abdominal trauma because they have weaker abdominal muscles, and are susceptible to hypoglycemia as a result of immature liver function.
- Children are more prone to dehydration than adults because they have a higher percentage of body water. Minimum urine outputs are 2 mL/kg/h for infants and 1 mL/kg/h for children, compared with 0.5 mL/kg/h for adults.
- Infants and children exposed to extremes of heat and cold are at higher risk than adults for hypothermia and hyperthermia because their ability to regulate temperature is not fully developed and they have a larger body surface to mass ratio.
- Close monitoring of oxygen and medication consumption is essential with children because they have a higher rate of metabolism and consume more oxygen than adults. The rate of onset of hypercapnia and

hypoxemia is accelerated.

- The CCTP should monitor the pediatric patient closely for hypoglycemia, which can manifest at a rapid onset with an asymptomatic or classic presentation. Treatment depends on the weight and age of the patient. Administration of 50% dextrose is contraindicated in the pediatric population.
- An understanding of normal growth and development, as well as the individual pediatric patient's developmental level, helps guide assessment, safety considerations, and communication techniques.
- Care strategies for psychosocial concerns vary with the age of the child. Speaking at eye level, using first names, and explaining medical procedures with age-appropriate words are techniques that can be used at all age levels. Maintaining a calm and confident manner also helps to reassure both the child and parent.
- CCTPs need to be academically, technically, and emotionally prepared to care for pediatric patients. They must recognize and address the needs of the parents, guardians, or caregivers, as well as those of the patient.
- Pediatric assessment begins with the PAT, which assesses the child's appearance, work of breathing, and circulation.
- After using the PAT to form a general impression of the patient, perform an initial/primary assessment. Any abnormal finding indicates the need for immediate treatment and may require deferral of the next step. Perform a secondary assessment on medical and trauma patients.
- Assess all children for pain. A numeric scale can be used for older children and adolescents; the FLACC or FACES scale can be used for infants and younger children.
- The CCTP can get extensive information for the child's history from the child's parents or caregivers. Scene or situation information may also be useful.
- Children with neural tube defects may have a latex allergy that can be life threatening. All latex products must be removed from care areas when treating these patients.
- Pediatric medication doses often have to be calculated, which increases the possibility of errors; doses of high-alert medications require an independent recheck before they are administered.
- The CCTP should be familiar with pediatric medications used for critical care transport and the different methods of medication delivery. The best option for administering resuscitation medications is a vascular access device.
- Nasal congestion can be life threatening to an infant because infants are obligatory nose breathers. Normal saline spray and a bulb syringe can be used to clear the nasal airways. Positioning the infant with the head up at 30° to 45° also aids in keeping the nasal airways clear. Administration of oxygen via a nasal cannula may also work well.
- Airway differences and other factors, such as infection, trauma, or foreign body airway obstruction, can impair the child's airway patency. Proper choice of blade size, proper positioning, and effective sweeping of the tongue are essential for successful intubation. Assess the corneal reflex to determine if the child has a gag reflex before inserting the oral airway.
- Tools used to identify the work of breathing in children include assessing stridor, snoring, retractions, head bobbing, accessory muscle use, tripoding, nasal flaring, wheezing, and grunting. The CCTP should know the parameters for normal pediatric vital signs but should remember to assess the patient, not the numbers or monitors. Assessing the child's level of consciousness, palpating the child's chest, and auscultating are used to assess breathing adequacy. Measurements used to determine breathing adequacy include SpO_2, $ETCO_2$, and arterial blood gas readings.

- Assessment of a child's cardiovascular system includes observing the child's general appearance and level of consciousness, including assessing the child's fingers and toes for evidence of clubbing; obtaining vital signs; assessing the child's skin, which should be warm, dry, and the appropriate color; and measuring the child's blood pressure. For some disease processes, MAP may be a more important measure in guiding treatment than blood pressure.
- Any trauma or medical condition that causes fluid losses requires IV access for volume replacement. Intraosseous IO insertion is recommended when rapid venous access cannot be obtained. The most common site for IO insertion is the anterior tibia. Contraindications include osteogenesis imperfecta, osteopetrosis, and fractures of the ipsilateral extremity.
- Urine output can be an objective indicator of pediatric circulatory status and the effectiveness of volume replacement. Low urine output indicates poor kidney perfusion.
- Neurologic assessment includes evaluating the child's general appearance, assessing the fontanelles, checking the child's pupil size and response to light, and evaluating muscle tone and reflexes, including the Moro reflex, the stepping reflex, and the Babinski reflex.
- In children who are not yet walking, the normal Babinski reflex is the opposite of normal for adults—flaring toes are normal, and toes pointing downward indicate brain injury. Protective reflexes are lost in ascending order.
- The GCS is the most sensitive indicator of mental status; it is modified when examining infants and children.
- Assessment of the renal system includes evaluation of hydration status and physical assessment, including assessment of the fontanelles in infants, assessment of skin turgor, the presence or absence of tears when crying, and sunken eyes. Daily weights are an objective measurement; acute weight loss may indicate fluid loss.
- Fluid overload is less common in children, but it may occur with congenital heart defects or renal insufficiency. Hypoxia and grunting are early signs of pulmonary edema. Additional signs of fluid overload include weight gain, edema, history of intake greater than output, and pulmonary edema. Early signs of pulmonary edema may be discounted because of subtle presentation. Pulmonary edema is confirmed by a chest radiograph.
- Resuscitation of infants and children must include measures to maintain body temperature because of children's physiologic differences. Measures must be taken to prevent hyperthermia and hypothermia.
- CCTPs who anticipate caring for critically ill or injured children need knowledge and skills specific to caring for infants and children as well as practice in pediatric care and mentoring from professional colleagues who care for pediatric patients on a regular basis.
- Equipment necessary for pediatric care should be available when transporting infants and children. The transport environment should also be latex free because of the high likelihood of latex allergies in special needs children. All equipment and supplies should be secured prior to transport to reduce the possibility of injury in the event of a crash.
- A team approach to interfacility transfer is best. Communication tools such as SBAR help to standardize the exchange of information among providers. The team should have a contingency plan for resuscitating the child if necessary during transport, and the driver or pilot should be prepared to respond if changes in the patient's condition or weather prevent completion of the transport.
- For safe transport, pediatric patients and health care providers must be secured in the vehicle and appropriate protective gear should be used. Interventions needed to stabilize the patient should be performed before transport. CCTPs need to be aware of local, regional, and national protocols for

addressing the increased risks of air medical transport and be prepared to take measures to reduce them.

- Guidelines for transporting parents or caregivers with a child should be in place before a child needs transport. Safe transport of the child is the priority, and each situation must be assessed for the caregiver's comfort level and potential for disruption of care during transport.
- The AAP recommends that self-inflating bags be used for resuscitation of children. CCTPs who will be caring for children should be familiar with their use as well as the use of flow-inflating bags, oxygen hoods, shortened tracheal tubes, and blade options.
- Respiratory illness is a common reason for pediatric transport. Upper airway conditions, lower airway conditions, parenchymal diseases, and abnormal control of ventilation can lead to upper airway failure.
- Respiratory distress is characterized by an increase in the work of breathing. Although the effort to breathe is increased, the child may still have adequate exchange of oxygen and carbon dioxide. The additional effort may also be for compensatory reasons.
- There are no strict guidelines for defining respiratory failure because it depends on the child's baseline respiratory function. Intervention to prevent respiratory or cardiac collapse is paramount. The CCTP should take all measures (per protocol) to maintain and preserve a patent airway and hemodynamic status before transport begins.
- Croup is a common viral infection characterized by a hoarse, "barking seal" cough that is often worse at night. Management ranges from the administration of humidified oxygen to the administration of racemic epinephrine to oral intubation in severe cases of obstruction.
- Epiglottitis is a bacterial infection that most commonly occurs in children aged 3 to 5 years. Symptoms include the four Ds: dysphagia, dysphonia, drooling, and distress. Management is controversial, but there is consensus that a calm environment should be maintained and a definitive airway should be secured prior to transport. Use of racemic epinephrine is contraindicated.
- Foreign body aspiration causes more than 90% of pediatric deaths in children younger than 5 years. Onset may be dramatic with an immediate life threat or with varying severity. Removal of the foreign body should be attempted only by following AHA and AAP guidelines.
- Early recognition of upper airway inflammation and intervention is imperative to lessen morbidity and mortality. Therapeutic measures are the same as for all upper airway obstructions: maintain a calm environment, administer humidified oxygen, provide a position of comfort, and prepare for deterioration of the airway status.
- Asthma is a chronic inflammatory disorder of the lower airways, resulting in a significant number of pediatric transports. Usually when transport is required, traditional treatments have failed and more advanced therapies are required. If a child has a history of requiring intubations for severe asthma, all treatments should be given during rapid transport to the hospital. Management includes oxygen therapy and the administration of a bronchodilator and corticosteroids.
- Bronchiolitis is a viral infection caused by RSV or another virus that primarily affects infants and young children. Symptoms may vary from mild wheezing to severe respiratory distress. Treatments are similar to treatments for asthma, but bronchodilators should be used with caution. Ribavirin, an antiviral drug, should be administered. Diligent assessment and monitoring of the child's work of breathing before and during transport are critical.
- Bronchopulmonary dysplasia is a chronic lung disease that develops in preterm infants who have been treated with oxygen and positive-pressure ventilation. The pathologic progression of the disease and

the healing process are not well understood. Children with BPD are at very high risk of pulmonary morbidity and mortality during the first 2 years of life.

- Pneumonia is a parenchymal lung disease. Infants and children may not tolerate pneumonia as well as older children and adults. Transport treatment includes vigilant assessment of the work of breathing and hydration status. Antibiotic therapy is required and may have been started at the initial facility.
- Near-SIDS is an acute episode of apnea accompanied by choking or gagging, skin color change, and a noted change in muscle tone. Etiologies associated with near-SIDS are gastroesophageal reflux disease, viral lower respiratory tract infections, seizures, sepsis, and meningitis. Near-SIDS patients usually only require supportive care and immediate transfer to a higher level of care for further studies or consultation.
- SIDS is the sudden, unexplainable death of an infant younger than 1 year. If the infant arrests while en route to the receiving facility, follow standing orders or contact medical control to request orders to continue or discontinue resuscitative measures. Be prepared to care for the parent if a parent accompanies the child during transport.
- Pediatric cardiac conditions are common indications for transport. CCTPs will routinely be involved with four classifications of heart disease or conditions, including a known cardiac defect, an unknown or suspected cyanotic defect, a cardiac arrhythmia, or some form of shock.
- Shock may lead to rapid death or may be more progressive. It is categorized as compensated or uncompensated, determined by the child's blood pressure.
- Hypovolemic shock is the most common cause of shock in pediatric populations. It is characterized by inadequate intravascular volume.
- There are four stages of hypovolemic shock: in stage 1, the child appears asymptomatic; in stage 2, the child maximizes compensatory mechanisms; in stage 3, the child is no longer able to compensate and hypotension occurs; in stage 4, death is imminent if the volume loss is not eliminated and aggressive volume replacement is not initiated. Resuscitation is successful when there is a return to normal heart and respiratory rates, normal blood pressure, and improved mental status and skin condition.
- The child with cardiogenic shock presents with traditional signs of shock, pulmonary congestion, and jugular venous distention. Treatment focuses on improving cardiac function with fluid resuscitation, inotropic support, and pharmacologic support.
- Neurogenic shock, anaphylactic shock, and dissociative shock are types of distributive shock. Treatment focuses on stopping vasodilation, returning volume to the intravascular space, and improving tissue perfusion.
- Symptoms of anaphylactic shock include increased general body edema, hypotension, rash, urticaria, anxiety, and warm, flushed skin. A child in anaphylactic shock may also experience bronchospasm and laryngeal edema, which may be life threatening. Management involves removing the allergen, replacing volume, and administering epinephrine.
- In dissociative shock, the hemoglobin molecule is unable to give up oxygen to the tissues. Dissociative shock is rare in the pediatric population. Examples of dissociative shock include CO poisoning and cyanide poisoning.
- There are two phases of septic shock: the warm and the cold phases. If not treated, a patient will progress from the warm to the cold phase. The CCTP should be able to diagnose septic shock based on the American College of Critical Care Medicine criteria. The goal of treatment is to resolve the hypovolemia and improve the perfusion status caused by cardiogenic shock.

- Approximately one of every 100 infants has a congenital heart defect. Cardiac anomalies may not be diagnosed until the neonate is 1 to 8 weeks old. Depending on their severity, cardiac defects may require immediate surgery or supportive care until the child is older and able to tolerate surgical intervention.
- Cardiac anomalies may be cyanotic or acyanotic. Acyanotic cardiac anomalies include VSDs, atrioventricular canal defects, ASDs, aortic stenosis, patent ductus arteriosus, and pulmonary stenosis.
- Aortic stenosis, hypoplastic left heart syndrome, and coarctation of the aorta cause impaired left ventricular output that may be treated by the CCTP.
- Persistent pulmonary hypertension (PPHN) in the newborn is caused by abnormally elevated pulmonary vascular resistance, resulting in hypoxia. Extracorporeal membrane oxygenation is used for short durations for patients with PPHN and congenital diaphragmatic hernia. Venoarterial ECMO is the form used most with neonates. Only a handful of teams in the United States have the expertise to perform ECMO transport.
- Cardiac defects that cause cyanosis include TOF and TGA. TOF is a cardiac anomaly consisting of four defects: ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and an overriding aorta. TOF is the most common heart defect seen beyond infancy. TGA is a defect in which the great vessels are reversed.
- A hypercyanotic spell, also called a TET spell, presents with marked cyanosis, irritability, tachypnea, pallor, and even loss of consciousness. Treatment may include supplemental oxygen, morphine, propranolol, and placing the patient in a knee-chest position.
- ECG rhythm interpretation is different for children than for adults. Normal values vary with the child's age. It may be helpful to increase the ECG monitor sweep speed to identify subtle nuances in the components of the ECG.
- Causes of acute renal failure are divided into three categories: prerenal, intrarenal, and postrenal.
- Prerenal causes are the most common in children and are primarily related to dehydration and decreased renal perfusion.
- Intrarenal or parenchymal causes are diverse, but all lead to nephron damage and loss of glomerular function. Hemolytic uremic syndrome is one of the most common causes of intrarenal failure in infants and children younger than 4 years. Acute glomerulonephritis, APSGN, and ATN are also intrarenal causes of renal failure.
- Postrenal or obstructive failure can cause irreversible parenchymal damage if not treated.
- Potentially life-threatening complications of acute renal failure include electrolyte imbalances such as hyponatremia, hypocalcemia, and hyperkalemia; volume losses or pulmonary edema and fluid overload; volume overload; hypertension; and septic shock.
- Trauma is the leading cause of death for children from the age of 6 months to young adulthood. The leading causes of trauma are motor vehicle, pedestrian, and bicycle accidents; submersions; burn; and firearm injuries.
- The two main resources for CCTPs assessing pediatric trauma patients are the GCS, which has been altered for pediatric patients, and the pediatric trauma score.
- Head trauma is the leading cause of morbidity and mortality in children. Injuries to the brain are evaluated as primary or secondary injuries.
- Primary injuries occur at the moment of impact and cause physical and mechanical destruction of brain tissue. Secondary injuries are injured areas of the brain that may remain viable but are vulnerable to

hypoxic, ischemic, and inflammatory responses. If untreated, secondary brain injury leads to irreversible brain damage and death.

- Concussion is the most commonly seen head injury in the pediatric population. The CCTP provides supportive measures during transport; performs and documents a neurologic assessment; and documents the patient's pain level and tolerance of transport.
- Other head injuries that CCTPs may encounter with children are cerebral contusion, epidural hematoma, and subdural hematoma. Subdural hematoma can be caused by child abuse, such as shaken baby syndrome.
- The incidence of spinal cord injuries in children is relatively rare, but mortality related to spinal cord injury in children is more than twice that in adult populations. As with brain injuries, spinal cord injuries may occur with a primary insult and then a secondary insult.
- The most important intervention with spinal cord injuries is proper immobilization. A properly fitting cervical collar and, ideally, a pediatric backboard should be used. If a pediatric backboard is not available, a standard backboard can be used with adaptations to accommodate the child.
- Children may sustain damage to the spinal cord and spinal ligaments without damage to the spinal vertebrae. This damage is called SCIWORA, which stands for spinal cord injury without radiographic abnormalities. SCIWORA is suspected when there are clinical signs or symptoms of spinal cord injury but the cervical spine radiographs are normal.
- Facial trauma, soft-tissue swelling, hanging injuries, and clothesline injuries can cause upper airway obstruction. The patient's tongue can also obstruct the airway. Simple pneumothorax, tension pneumothorax, open pneumothorax, hemothorax, and flail chest may threaten the child's life. Management of these conditions is generally the same for children as for adults. The CCTP should be aware of differences in managing these conditions in children.
- Pericardial tamponade is a hemodynamic emergency. Pericardiocentesis may be challenging in the field. It may be beyond the scope of some CCTPs and it should not be performed in a moving ground or air vehicle.
- Children's bones tend to buckle, rather than fracture, because they are more porous than adults' bones. Children also have stronger and more resilient ligaments than adults, making dislocations rare in pediatric populations.
- The child's cervical spine is highly flexible, and children tend to have avulsions rather than fractures. Because of their large, heavy heads and weak neck muscles, children tend to have rapid deceleration injuries, resulting in hyperflexion and torsion forces.
- Pelvic fractures can cause massive internal hemorrhaging and can lead to profound deterioration of the child. PASG is one option for stabilizing a pelvic fracture for transport.
- A femur fracture can cause significant blood loss in children. Older children and adolescents are most at risk for femur fractures.
- Children will experience similar injury patterns as adults in motor vehicle accidents despite the use of car seats and restraints. In a pedestrian crash, children's injuries are most likely to involve the head and chest. The CCTP must find out what occurred and be alert for these injury patterns.
- Burns are the second most common cause of accidental death in children. The peak incidence of burn injuries occurs in children aged 1 to 5 years. Most burn injuries in children are from scalding liquids. Children have a higher mortality rate than adults from burns.
- The CCTP should remove the burn source, address initial life threats, care for the wound, manage the

child's pain, and monitor the child closely during transport to the burn center.

- CCTPs must adhere to agency protocol regarding reporting suspected child abuse or neglect. In cases of confirmed abuse, CCTPs must preserve evidence. CCTPs may be called on to testify in court, so it is important to document findings from the physical exam, statements made by the victim or suspected perpetrator, and transport details.
- Children are at higher risk for hypothermia than adults. Hypothermia exists when the patient's core body temperature is 95°F (35°C) or less. CCTPs should adhere to agency protocols or resuscitation guidelines for children with hypothermia.
- Heat stroke is a life-threatening emergency with morbidity rates ranging from 17% to 70%. The patient should be moved immediately into an air-conditioned vehicle, and measures should be taken to lower body temperature.
- Drowning is the second leading cause of death for children aged 1 to 14 years. Wet drowning accounts for 85% of incidents. The key to a successful outcome is brief submersion and good resuscitation. Approximately 10% to 15% of drowning incidents are related to dry drowning. Death from dry drowning is the result of asphyxiation vs aspiration.
- The frequency of caring for children with special needs is increasing. Parents and caregivers are the best resource for understanding their child's illness and for using the child's specialized equipment.

Vital Vocabulary

acute glomerulonephritis (AGN) A histopathologic diagnosis associated with edema, hypertension, and hematuria; results from the deposits of circulating immune complexes in the kidney basement membrane, which ultimately causes reduced glomerular filtration.

acute poststreptococcal glomerulonephritis (APSGN) A form of acute glomerulonephritis that occurs following an infection by streptococci.

acute tubular necrosis (ATN) A condition that results in damage to the tissue of the kidneys' tubules.

aortic stenosis Narrowing of the aortic valve.

atrial septal defect (ASD) A hole in the atrial septal wall that allows blood to flow from the left atrium into the right atrium.

atrioventricular canal defect A hole in both the atrial and ventricular septal walls that allows blood to flow from the left side of the heart to the right side of the heart; usually involves defects of both the mitral and tricuspid valves.

Babinski reflex When the toe(s) move upward in response to stimulation to the sole of the foot. Under normal circumstances, the toe(s) move downward.

bronchiolitis A condition seen in children younger than age 2 years, characterized by dyspnea and wheezing.

bronchopulmonary dysplasia (BPD) A chronic lung disease that develops in preterm neonates who have been treated with oxygen and positive-pressure ventilation; infants who survive this have pulmonary hypertension and abnormal pulmonary vascular development.

Brudzinski sign Passive flexion of the legs and thighs when the examiner flexes the patient's neck to the chest. If this causes pain or causes the knees and hips to flex involuntary, the sign is positive.

cerebral contusion A focal brain injury in which brain tissue is bruised and damaged in a defined area.

coarctation of the aorta (CoA) Pinching or narrowing of the aorta that obstructs blood flow from the heart to the systemic circulation.

concussion A transient interruption of normal neurologic function.

croup A childhood viral disease characterized by edema of the upper airways with barking cough, difficult breathing, and stridor; also called laryngotracheobronchitis.

epiglottitis Inflammation of the epiglottis.

extracorporeal membrane oxygenation (ECMO) A supportive intervention which provides oxygenation to a patient by removing the patient's blood to a heart and lung machine that does the work normally done by the lungs and heart. The blood is then transfused back into the patient's circulation.

FLACC scale A pain assessment scale that scores points for five categories, including face, legs, activity, cry, and consolability.

hemolytic uremic syndrome (HUS) A serious condition in which the patient develops acute renal failure, and which also causes anemia and thrombocytopenia; thought to be caused by several bacterial and viral infectious organisms, most often verotoxin-producing strains of *E coli*.

hyperkalemia An increased level of potassium in the blood.

hypocalcemia A low level of calcium in the blood.

hyponatremia A blood serum sodium level that is below 135 mEq/L, and a serum osmolarity level that is less than 280 mOsm/kg.

hypoplastic left heart syndrome (HLHS) Underdevelopment of the aorta, aortic valve, left ventricle, and mitral valve; this defect involves the entire left side of the heart.

intrarenal A type of acute renal failure caused by damage of an actual nephron.

Kernig sign Resistance and pain elicited on extension of the leg at the knee, with the patient in the supine position and the hips flexed perpendicular to the trunk; when pain occurs as a result of this test, the test result is positive.

Moro reflex An infant reflex in which, when an infant is caught off guard, the infant opens his or her arms wide, spreads the fingers, and seems to grab at things.

near–SIDS An acute episode in which an infant experiences apnea accompanied by choking or gagging, skin color change, and a noted change in muscle tone; also called an ALTE (apparent life-threatening event).

patent ductus arteriosus (PDA) A situation in which the ductus arteriosus, which assists in fetal circulation, does not transition as it should after birth to become the ligamentum arteriosum; the result is that the connection between the pulmonary artery and the aorta remains, allowing some oxygenated blood to move back into the heart rather than all of it moving out of the aorta and into the systemic circulation.

Pediatric assessment triangle (PAT) An assessment tool that allows rapid formation of a general impression of the type and level of illness or injury in an infant or child without touching him or her; consists of assessing appearance, work of breathing, and circulation of the skin.

persistent pulmonary hypertension (PPHN) A condition in the newborn caused by an abnormally elevated pulmonary vascular resistance resulting in hypoxia. The typical presentation consists of low APGAR scores, hypoglycemia, or a congenital diaphragmatic hernia.

postrenal A type of acute renal failure caused by downstream obstruction of urine flow from the kidneys,

with initially intact nephrons; commonly caused by a blockage of the urethra by prostate enlargement, renal calculi, or strictures.

prerenal A type of acute renal failure caused by decreased perfusion of an intact nephron; often reversible if the underlying condition can be found and perfusion can be restored to the kidney.

pulmonary stenosis Narrowing of the pulmonary valve.

reactive airway disease (RAD) A term used to describe any condition that causes hyperreactive bronchioles and bronchospasm.

SCIWORA An acronym that stands for spinal cord injury without radiographic abnormalities.

stepping reflex An infant reflex that occurs when the infant is held up in the air and moves his or her legs up and down as if marching.

sudden infant death syndrome The sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation.

tetralogy of Fallot (TOF) A cardiac anomaly that consists of four defects: a ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and an overriding aorta.

transposition of the great arteries (TGA) A defect in which the great vessels are reversed; the aorta is connected to the right ventricle, and the pulmonary artery is connected to the left ventricle.

ventricular septal defect A hole in the septum separating the ventricles, allowing blood from the left ventricle to flow into the right ventricle.

Bibliography

Brierley J, Carcillo JA, Choong K, et al. Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. *Crit Care Med.* 2009;37(2):666–688.

Caroline N. In: Pollak AN, Elling B, Smith M, eds. *Nancy Caroline's Emergency Care in the Streets* , 6th ed. Sudbury, MA: Jones and Bartlett Publishers; 2008.

Dieckmann R, ed. *Pediatric Education for Prehospital Professionals*, 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2006.

Emergency Nurses Association. *Emergency Nurse Pediatric Course.* Des Plaines, IL: Emergency Nurses Association; 2004.

Gausche-Hill M, Fuchs S, Yamamoto L, eds. *APLS: The Pediatric Emergency Medicine Resource*, 4th ed. Elk Grove Village, IL: American Academy of Pediatrics and American College of Emergency Physicians; 2004.

Hazinski MF, ed. *Pediatric Advanced Life Support Provider Manual.* Dallas, TX: American Heart Association; 2006.

Hohenhaus S, Powell S, Hohenhaus J. Enhancing patient safety during hand-offs: standardized communication and teamwork using the "SBAR" method. *Am J Nurs.* 2006;106(8):72A–72B.

Merkel SI, Voepel-Lewis T, Shayevitz JR, Malviya S. FLACC: a behavioral scale for scoring postoperative pain in young children. *Pediatr Nurse.* 1997;23(3):293–297.

Willinger M, James LS, Catz C. Defining the sudden infant death syndrome (SIDS): deliberations of an expert panel convened by the National Institute of Child Health and Human Development. *Pediatr Pathol.* 1991;11(5):677–684.

Glossary

3-3-2 rule A method used to predict difficult intubation. A mouth opening of less than three fingers wide, a mandible length of less than three fingers wide, and a distance from hyoid bone to thyroid notch of less than two fingers wide indicate a possibly difficult airway.

abandonment The termination of an established patient-care provider relationship by the care provider without simultaneous transfer of health care responsibility to another care provider of equal or greater competence.

Abbreviated Injury Scale (AIS) A trauma scoring system that ranks injury severity by assigning an individual injury score of 1 to 6 to six body regions, with 1 being a minor injury and 6 being an injury with a high mortality rate; does not account for multisystem injuries.

abdominal compartment syndrome (ACS) A condition that can result from intra-abdominal hypertension, and that includes a range of deleterious effects, including decreased end-organ perfusion; if untreated, it can lead to death.

ablation Removal of a pathway or function by electrocautery or radiofrequency.

ABO-incompatible transfusion reactions A type of transfusion reaction in which the patient possesses antigens to a blood type and receives that blood type.

absolute refractory period The early phase of cardiac repolarization, wherein the heart muscle cannot be stimulated to depolarize.

absorption Acquisition of additional heat, radiation, or other energy from the environment; or, a substance's molecules are moved from the site of entry on the body into the systemic circulation.

accuracy A measure of the likelihood that an average of a set of test values will be the same as individual values in the set.

acetylcholine (ACh) A chemical neurotransmitter in the parasympathetic nervous system.

acetylcholinesterase An enzyme that breaks down acetylcholine at the neuromuscular junction.

acquired immunity The immunity the body develops as part of exposure to an antigen.

acrocyanosis A decrease in the amount of oxygen delivered to the extremities. The hands and feet turn blue because of the lack of oxygen.

acromegaly A syndrome that results from excessive growth hormone secreted by the pituitary gland after the epiphyseal plate has closed; characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate.

activated charcoal A carbon-based liquid with an incredible absorptive ability that is typically administered orally or via nasogastric or orogastric tube to decrease the available quantity of a toxic substance.

activated partial thromboplastin time (aPTT) A value that represents the intrinsic coagulation pathway's clotting ability; also known as partial thromboplastin time (PTT).

active immunity Production of specific antibody in response to an infection or antigen.

acute coronary syndrome (ACS) The term used to describe any group of clinical symptoms consistent with acute myocardial ischemia.

acute gastritis A type of gastritis characterized by a rapid onset of mucosal inflammation, frequently

accompanied by mild to severe upper GI bleeding.

acute glomerulonephritis (AGN) A histopathologic diagnosis associated with edema, hypertension, and hematuria; results from the deposits of circulating immune complexes in the kidney basement membrane, which ultimately causes reduced glomerular filtration.

acute poststreptococcal glomerulonephritis (APSGN) A form of acute glomerulonephritis that occurs following an infection by streptococci.

acute radiation syndrome Radiation poisoning.

acute renal failure (ARF) Decreased renal function in the absence of preexisting renal disease. Classified into three categories: prerenal, intrarenal, and postrenal.

acute tubular necrosis (ATN) A condition that results in damage to the tissue of the tubules of the nephron, preventing proper ion and fluid exchanges in the kidneys.

adaptive immune system The secondary mechanism that protects the host by reacting with and eliminating specific antigens after requiring more time than the innate immune system to mobilize its defense against unknown pathogens.

Addisonian crisis The sudden appearance of symptoms, especially shock, in a patient with chronic adrenal insufficiency; may appear suddenly as a result of an increased period of stress, trauma, surgery, or severe infection; other symptoms include weakness, altered mental status, hyperthermia, and severe pain in the lower back, legs, or abdomen.

Addison's disease A chronic hormonal or endocrine disorder caused by a deficiency of cortisol and/or aldosterone and characterized by weakness, fatigue, hypotension, unexplained weight loss, and darkening of the skin.

adhesions Bands of connective tissue that can distort the normal GI anatomy. They are the result of improper healing or scar tissue growth following surgery.

adrenal insufficiency (AI) Underproduction of cortisol and aldosterone caused by a decreased function of the adrenal cortex; occurs when at least 90% of the adrenal cortex has been damaged.

adsorbtion The process of attracting molecules of a substance to the surface of that substance.

adventitious breath sounds Abnormal breath sounds that are heard in addition to, or in place of, normal sounds.

aerobic metabolism A form of energy production in which mitochondria utilize glucose, amino acids, and fatty acids combined with oxygen and ADP to produce ATP, carbon dioxide, water, and heat.

afferent pathways Ascending pathways that carry sensory impulses toward the central nervous system.

afterload An expression of cardiac work related to the forces that must be overcome to eject blood from the ventricle; the tension or stress that develops in the ventricles during systole.

agonal respirations Slow, shallow, irregular respirations or occasional gasping breaths; results from cerebral anoxia.

Air Medical Physician Association (AMPA) An organization of physicians and air medical professionals that promotes safe patient transport.

alanine aminotransferase (ALT) An intracellular enzyme that is found in large amounts in the liver and in the kidney, skeletal muscle, and heart; formerly known as SGPT.

albumin The most common protein in the body, which acts as a transport protein, is a free radical

scavenger, and serves as the main source of protein-generated oncotic pressure; also a blood product containing this specific protein found in the blood, and prepared by the fractionation of pooled plasma; used for volume replacement in certain conditions.

alcoholic ketoacidosis A type of acidosis characterized by a buildup of ketones in the blood and caused by a large intake of alcohol.

aldosterone One of the two main hormones responsible for adjustments to the final composition of urine; it increases the rate of active resorption of sodium and chloride ions into the blood and decreases the resorption of potassium.

aldosteronism A syndrome of high blood pressure and low blood potassium levels caused by an excess of aldosterone; there are two main types (primary and secondary).

alkaline phosphatase An enzyme that is essential for proper digestion and absorption through the mucous membrane in the gastrointestinal tract; clinically useful for testing liver function and for diagnosing a common bile duct obstruction.

Allen test A technique in which the patient's hand is initially held above the head while the fist is clenched and the radial and ulnar arteries are compressed; the hand is then lowered and the fist is opened, ulnar pressure is released, and radial pressure is maintained; after ulnar pressure is released, color should return to the hand within 6 seconds.

alveolar ventilation (V_A) The volume of air that comes into contact with the alveolar capillary membrane surfaces and participates in the exchange of gases between the lung and blood.

amniotic fluid embolism A rare condition that can occur antepartum and postpartum, in which fetal tissue crosses over the placental barrier into maternal circulation, thereby causing obstruction to the mother's organs with organ failure or respiratory and cardiovascular compromise.

amylase A key enzyme used by the body to metabolize carbohydrates; it is produced primarily by the salivary glands and the pancreas.

amyloidosis A group of diseases that result from abnormal deposits of the protein amyloid in various tissues of the body; can occur in a localized area or may be systemic.

anaerobic metabolism A less efficient form of energy production in which an alternative pathway converts glucose to pyruvic acid with the simultaneous production of ATP and that also results in the production of lactate.

anaphylactic shock A severe hypersensitivity reaction that involves bronchoconstriction and cardiovascular collapse.

anaphylactoid reaction A non–IgE-mediated response that causes the rupture of mast cells and basophils, which then release histamine and other defense mediators.

anatomic dead space (V_D) Space in airway structures such as the trachea, bronchi, and bronchioles that does not participate in gas exchange. It is defined physiologically as ventilation without perfusion.

aneurysm A weakened portion of the wall of an artery where the blood creates a localized dilation or bulge; can involve the wall intact or be classified as dissecting, in which the artery wall ruptures and blood pools between the inner and outer artery wall.

angiodysplasia Deformed submucosal blood vessels in the GI tract that are prone to bleeding.

angiography Radiologic observation of dye injected into the bloodstream for leakage.

anion A negatively charged ion.

anion gap (AG) A summary of the relationship among the three major contributors to the overall electrical charge (Na^+, Cl^-, and HCO_3^-); abnormal AG values may signal disturbances in the overall electrical and acid-base balance of the serum and presence of disease.

anisocoria A condition in which the pupils are not of equal size.

anode The electrode in a pacing circuit that is positively charged when current is flowing.

antegrade conduction Conduction in the normal direction between cardiac structures.

anterior cord syndrome Displacement of bony fragments into the anterior portion of the spinal cord, often as the result of flexion injuries or fractures, that disrupts blood flow in the anterior spinal artery.

antiarrhythmic A type of medication used to treat and prevent cardiac rhythm disorders.

anticholinergic syndrome A syndrome that occurs following excessive exposure to medications such as antihistamines, atropine, and benztropine (Cogentin), or other substances such as Jimson weed, resulting in muscarinic receptor blockade at the neuromuscular junction; characterized by tachycardia, hyperthermia, dilated pupils, warm (or hot) dry skin, ileus, delirium, seizures, psychosis, and urinary retention.

antigen An agent that, when taken into the body, stimulates the formation of specific protective proteins called antibodies.

anxiolysis The reduction of anxiety by the administration of an antianxiety agent.

aortic regurgitation Backward flow of blood through the aortic valve from the aorta into the left ventricle.

aortic stenosis Narrowing of the aortic valve.

APGAR score A scoring system for assessing the status of a newborn that assigns a number value to each of five areas of assessment (the total possible score is 0 to 10).

aphasia Any loss or impairment of language function as a result of brain damage.

apnea Respiratory pause greater than or equal to 20 seconds; the absence of respiration.

apneustic breathing A condition in which lesions in the respiratory center of the brain stem lead to a breathing pattern characterized by prolonged, gasping inspiration, followed by extremely short, ineffective expiration.

arachnoid mater The middle layer of the meninges, which contains blood vessels that give it the appearance of a spider web.

arterial blood gas (ABG) Analysis of the following characteristics of blood: pH, partial pressure of carbon dioxide (in arterial blood), partial pressure of oxygen (in arterial blood), concentration of bicarbonate ion, base excess (indicating whether the patient is acidotic or alkalotic), and oxygen saturation of the hemoglobin molecule.

arterial lines Catheters inserted into the patient's arterial vascular system for the purpose of producing a waveform with pressure measurements; also called an A-line.

arterial oxygen content (CaO_2) The total amount of oxygen in the arterial blood.

arteriovenous malformation (AVM) Abnormally formed blood vessels that have a higher rate of bleeding than average vessels.

ascites A buildup of fluid in the abdominal cavity that can impede lung expansion and put pressure on

other organs.

aspartate aminotransferase (AST) An intracellular enzyme that is found in large amounts in the liver and in skeletal muscles, the brain, red blood cells, and the heart; formerly known as SGOT.

aspartate aminotransferase/alanine aminotransferase (AST/ALT) ratio A ratio used as an indicator of liver damage; this ratio can determine if the elevation is due to a liver injury or injury elsewhere in the body.

assault The act of putting someone in reasonable apprehension of imminent harmful or offensive contact.

assist ratio A setting on the IABP that allows the operator to determine how often the pump inflates the balloon. A 1:1 setting means the balloon inflates with each heartbeat; and 1:2, with every other heartbeat, etc; also called IABP frequency.

assisted systole The pressure waveform that follows the diastolic augmentation peak.

AST:ALT A calculated index that is useful for determining the cause of liver dysfunction.

atmosphere Gases that extend from the earth's surface to space; composed primarily of nitrogen, oxygen, argon, and trace gases.

atresia Complete absence of lumen.

atrial septal defect (ASD) A hole in the atrial septal wall that allows blood to flow from the left atrium into the right atrium.

atrioventricular canal defect A hole in both the atrial and ventricular septal walls that allows blood to flow from the left side of the heart to the right side of the heart; usually involves defects of both the mitral and tricuspid valves.

atrioventricular (AV) valves The valves (mitral and tricuspid) that separate the atria from the ventricles.

auto launch A protocol that launches an aircraft at the time it is placed on standby rather than waiting for the formal launch request. An auto launch is frequently activated when the distance to be covered for the response is greater than 30 nautical miles.

autonomic dysreflexia A potentially life-threatening complication of spinal cord injury that results from the loss of parasympathetic stimulation. It is characterized by a massive, uninhibited, uncompensated cardiovascular response as the result of some stimulation of the sympathetic nervous system below the level of injury. Also called autonomic hyperreflexia.

auto-PEEP The nonintended increase in end alveolar pressure due to air trapping.

axonal transport The movement of organelles and proteins along a nerve cell axon into and out of the cell body.

azotemia A combined elevation of blood urea nitrogen and serum creatine levels often seen in acute renal failure.

azygos system A network of blood vessels that connects the superior and inferior vena cava; it also drains a portion of the esophageal venous blood.

Babinski reflex When the toe(s) move upward in response to stimulation to the sole of the foot. Under normal circumstances, the toe(s) move downward.

bacteriocidal Capable of killing bacteria.

bacteriostatic Capable of inhibiting bacterial growth.

barometric pressure The weight per unit area of all of the molecules of the gases above the point at which the measurement was taken.

barotitis media Inflammation and possible petechial hemorrhage in the middle ear and possible rupture of the eardrum that results from the failure of the middle ear space to ventilate when going from low to high atmospheric pressure; also known as ear block.

barotrauma Injury to tissues, organs, or structures within the body, resulting from rapid or significant changes in environmental air pressure.

basal ganglia Masses of nuclei located deep in the cerebral hemispheres that play a major role in fine motor function.

basal metabolic rate (BMR) The heat energy produced at rest by normal body metabolic reactions, determined mostly by the liver and skeletal muscles.

base excess (BE) A measure of metabolic derangement that is part of the arterial blood gas panel; also known as base deficit (BD) as the value can be either positive (excess) or negative (deficit).

basilar skull fracture A fracture along the base of the skull.

battery Harmful or offensive touching of another without consent.

Battle's sign Migration of blood to the mastoid region, posterior and slightly inferior to the ear, resulting in discoloration; also called retroauricular ecchymosis.

Beck's triad The combination of a narrowed pulse pressure, muffled heart tones, and jugular vein distention associated with cardiac tamponade; usually resulting from penetrating chest trauma.

bicarbonate (HCO_3^-) An ion (HCO_3^-) that is present in the blood; measurement represents the metabolic component of the arterial blood gas panel.

bicarbonate–carbonic acid buffer system The principal extracellular buffer system, which operates primarily in the lungs and kidneys, and by which Co_2 and H_2O are reversibly converted into H_2CO_3 (carbonic acid), which in turn is reversibly converted into H^+ and HCO_3^- (bicarbonate).

bifascicular block The combination of a right bundle branch block and a block of one of the fascicles of the left bundle, the left anterior or left posterior fascicle.

bi-level positive airway pressure (BiPAP) The use of two separate pressures—inspiratory positive airway pressure and expiratory positive airway pressure—to raise the breathing baseline above ambient pressure. The pressure gradient enhances ventilation, and the reduced expiratory pressure makes exhalation easier and increases patient tolerance.

bilirubin A waste product of heme formed during erythrocyte metabolism. It is moved to the small intestine within bile and then converted into urobilinogen, resulting in brown pigmented feces.

bioavailability The amount or percentage of a medication that reaches systemic circulation without being altered following administration.

Biot's (ataxic) breathing Breathing characterized by three patterns: (1) slow and deep, (2) rapid and shallow, and (3) apnea. Causes include meningitis, increased intracranial pressure, and central nervous system dysfunction.

biphasic A term used to describe a wave with negative and positive components; usually used in conjunction with P and T waves.

bipolar lead A conduction lead comprising two electrodes attached at specific body sites with different

polarity used to examine electrical activity by monitoring changes in the electrical potential between them.

bipolar system A closed system consisting of bipolar leads and a module to generate impulses and measure response.

bleb A blister or bladderlike structure that may be filled with fluid.

blood urea nitrogen (BUN) A test used to measure urea, which is a biomarker for adequate kidney function.

blood–brain barrier A network of endothelial cells and astrocytes (neuroglia) in the brain that regulate the transport of nutrients, ions, water, drugs, and waste products to and from the brain through the process of selective permeability.

body packers People who swallow carefully packaged capsules for smuggling purposes.

body stuffers People who hastily swallow illicit substances to avoid impending arrest.

Boyle's law States that the volume of a gas is inversely proportional to the pressure to which it is subjected. Gases trapped in body cavities will expand with increases in altitude and will contract with decreases in altitude.

bradypnea A respiratory rate that is slower than normal.

brain herniation Displacement of a portion of the brain from its correct location within the cranial cavity to a different location.

brain tissue compliance The change in brain volume resulting from a change in pressure.

brain tissue oxygen tension ($P_{br}O_2$) A method of monitoring temperature and oxygenation via placement of a commercial probe into the brain tissue through a bolt; can be done simultaneously with ICP monitoring placement.

breach of duty to act A case in which a health care provider does not conform to the standard of care by providing inappropriate care, failing to act, or acting beyond the scope of practice.

breech A delivery presentation in which the buttocks emerge first.

Broca's area Part of the frontal lobe that is located at the inferior frontal gyrus and that participates in the formulation of words.

bronchiolitis A condition seen in children younger than age 2 years, characterized by dyspnea and wheezing.

bronchopulmonary dysplasia (BPD) A chronic lung disease that develops in preterm neonates who have been treated with oxygen and positive-pressure ventilation; infants who survive this have pulmonary hypertension and abnormal pulmonary vascular development.

bronchovesicular sounds A combination of the tracheal and vesicular breath sounds, heard in places where airways and alveoli are found, the upper part of the sternum and between the scapulae.

Brown-Sequard syndrome Loss of function as a result of penetrating trauma accompanied by hemisection of the spinal cord and complete damage to all spinal tracts on the involved side; characterized by loss of motor function and sensation of light touch, proprioception, and vibration on the ipsilateral side and temperature and pain sense on the contralateral side.

Brudzinski sign Passive flexion of the legs and thighs when the examiner flexes the patient's neck to the chest. If this causes pain or causes the knees and hips to flex involuntary, the sign is positive.

B-type natriuretic peptide (BNP) A polypeptide whose value that is indicative of abnormal ventricular function and congestive heart failure.

buffer Any substance that can alternately bind or release H_+ depending on outside conditions.

BUN:Cr A calculated index that is used to determine the cause of increased levels of blood urea nitrogen and creatinine.

cable The physical wire that connects the electrode to the ECG monitor.

caloric test A method for assessing vestibular function that involves the raising and lowering of the temperature in the external auditory canal; also called Bárány test.

capnography The measurement of exhaled Co_2, which in most cases correlates with the Co_2 levels in arterial blood, can be done with two different types of devices—an electronic monitor that displays a waveform or a colorimetric device that should turn yellow during exhalation, indicating proper tube placement.

capnometry The measurement of exhaled Co_2, performed the same way as capnography, but which provides a light-emitting diode readout of the patient's exhaled Co_2.

caput succedaneum In the newborn, a subcutaneous collection of fluid with poorly defined margins within the scalp, resulting from pressure on the head during delivery.

carboxyhemoglobin (COHb) A measure of the amount of hemoglobin–carbon monoxide complexes in the blood.

cardiac electrophysiology The cardiac specialty that involves evaluation and management of rhythm disturbances.

cardiac index A hemodynamic value that adjusts a patient's cardiac output to take into account his or her total body surface area.

cardiac monitoring The continuous observation of the patient's condition in relation to his or her cardiac rhythm.

cardiac output (CO) The amount of blood pumped by the heart per minute, calculated by multiplying the stroke volume by the heart rate per minute.

cardiogenic shock A condition caused by loss of 40% or more of the functioning myocardium; the heart is no longer able to circulate sufficient blood to maintain adequate oxygen delivery.

cathode The electrode in a pacing circuit that is negatively charged when current is flowing.

cation A positively charged ion.

cauda equina The collection of individual nerve roots into which the spinal cord separates at the L2 vertebra.

caudal Pertaining to or in the direction of the feet.

central cord syndrome A syndrome in which cavities form in the central portions of the spinal cord, usually in the cervical area; may be due to a tumor, genetic origin, or trauma; presents along with hemorrhage or edema to the central cervical segments.

central cyanosis Bluish coloration of the skin due to the presence of deoxygenated hemoglobin in blood vessels near the skin surface.

central diabetes insipidus A result of damage to the posterior part of the pituitary gland, which causes a

lack of the hormone vasopressin, an anti-diuretic hormone, to be produced; because of the lack of anti-diuretic hormone, patients frequently experience polyuria and polydipsia.

central lumen The lumen or port of the IAB catheter used to guide initial catheter insertion and to monitor arterial pressure during operation.

central neurogenic hyperventilation A pattern of very deep, rapid respirations at rates of 40 to 60 breaths/min, caused by a midbrain lesion or dysfunction.

central venous lines IV access catheters that terminate in central circulation, usually just proximal to the right atrium.

central venous pressure (CVP) The pressure in the superior vena cava (average 2 to 6 cm H_2O); reflects the pressure in the venous system when the blood is returned to the right atrium; indicative of a patient's fluid volume status and right-sided heart performance.

cephalad Toward the patient's head.

cephalohematoma A subperiosteal collection of blood; may be accompanied by a linear skull fracture.

cerebral angiography A procedure that uses imaging and a contrast material or dye to view the blood vessels in the brain and is used to find abnormalities.

cerebral aqueduct The narrowest portion of the brain's ventricular system; it provides communication with the fourth ventricle, which lies between the brain stem and the cerebellum.

cerebral blood flow (CBF) The amount of blood flow the brain requires to maintain homeostasis. In a 24-hour period, the brain requires 1,000 L of blood to obtain 71 L of oxygen and 100 g of glucose.

cerebral contusion A focal brain injury in which brain tissue is bruised and damaged in a defined area.

cerebral cortex The outermost layer of the cerebrum.

cerebral function analysis monitor (CFAM) A device that can provide summed, averaged, and analyzed outputs of the general state of brain activity.

cerebral function monitor (CFM) A device that can provide summed and averaged outputs, but not analysis, of the general state of brain activity.

cerebral hemispheres The name for each half of the brain (right or left), each of which contains one of the paired lobes (occipital, parietal, temporal, and frontal).

cerebral metabolic rate for oxygen ($CMRO_2$) A measurement used to determine neuronal demand for oxygen. Neurons with high activity rates require greater amounts of oxygen.

cerebral perfusion pressure (CPP) The pressure gradient across the brain; it provides an estimate of perfusion adequacy: CPP = MAP – ICP.

Charcot's triad Fever, jaundice, and right upper quadrant abdominal pain suggestive of choledocholithiasis.

Charles' law States that when pressure is constant, the volume of a gas is very nearly proportional to its absolute temperature. Thus, the volume is directly proportional to the temperature when it is expressed on an absolute scale where all other factors remain constant.

chelating agents Chemical substances that bind to heavy metals to remove them from the body.

Cheyne-Stokes respiration A cyclic pattern of increased respiratory rate and depth with periods of apnea. Causes include increased intracranial pressure, renal failure, meningitis, drug overdose, or hypoxia secondary to congestive heart failure.

choanal atresia A congenital narrowing or blockage of the nasal airway by membranous or bony tissue.

cholangitis An infective form of biliary tract obstruction in which white blood cell levels rise.

cholecystitis Inflammation of the gallbladder.

choledocholithiasis Gallstones in the biliary tract system, which put the person at high risk for developing a biliary tract obstruction.

cholelithiasis Gallstones in the gallbladder.

cholinergic syndrome Cholinesterase inhibitor toxicity.

cholinesterase An enzyme that hydrolyzes acetylcholine to acetic acid and choline.

chordae tendineae The thin, fibrous strands stretching out from the apexes of some of the papillary muscles of the ventricles, extending upward and attaching to the edges of the AV valves, which help keep the cusps of the valves from turning inside out when pressure builds up in the ventricles.

choroid plexus A cluster of nerve roots at the lateral and the third and fourth ventricles of the brain that produce cerebrospinal fluid.

chronic renal failure (CRF) A gradual decrease in renal function resulting from irreversible damage to the nephrons. It is characterized by a glomerular filtration rate (GFR) of less than 30 mL/min, and will eventually progress to end-stage renal disease.

chronotropic Altering the rate of contraction of the heart.

circle of Willis A system of arteries located at the base of the skull that (in most people) is able to compensate for reduced blood flow from any one of the major contributors to cerebral circulation.

cirrhosis Irreversible structural changes to the liver that impair its proper functioning.

cluster breathing An abnormal respiratory pattern in which a cluster of irregular respirations that vary in depth are followed by a period of apnea at irregular intervals.

coagulation necrosis The death of tissue resulting from a blockage of blood flow to that tissue, in which the blockage is caused by clots preventing blood from reaching the tissue.

coarctation of the aorta (CoA) Pinching or narrowing of the aorta that obstructs blood flow from the heart to the systemic circulation.

cocaine washout syndrome Profound exhaustion with the ability to regain normal mental status and orientation when aroused; common with heavy cocaine users.

cold zone The area surrounding the warm zone at a hazardous materials scene. No environmental hazards should be present in this zone.

colonoscopy Similar to endoscopy, but used for the lower GI tract.

comminuted fracture A type of skull fracture in which the skull is splintered or shattered into many pieces.

Commission on Accreditation of Air Medical Transport Services (CAMTS) An organization dedicated to improving the quality of patient care and safety of the transport environment for both rotor-wing and fixed-wing providers. CAMTS also provides voluntary accreditation of critical care transport agencies.

compartment syndrome A condition that develops when edema and swelling result in increased pressure within soft tissues, causing circulation to be compromised, possibly resulting in tissue necrosis.

compensatory stage A stage of hypoxia in which the physiologic adjustments that occur in the respiratory and circulatory systems are adequate to provide defense against the effects of hypoxia. Factors such as environmental stress and prolonged exercise can potentiate certain effects of hypoxia.

competence Capacity or ability to understand the nature and effects of one's decisions or actions.

complete breech A delivery presentation in which the buttocks present first and the legs are crossed inside the uterus, oriented transversely to the birth canal; in this case, the mother is unable to vaginally deliver.

complete spinal cord injury A complete disruption of all tracts of the spinal cord, with permanent loss of all cord-mediated functions below the level of transaction.

computed tomography (CT) An imaging study in which focused beams examine an area that is then reconstructed by computer to give a high-resolution view; multiple "slices" or views are created; performed on almost every patient with abnormal neurologic findings; also called CAT scanning.

concentration The amount of a substance present in a given volume of fluid.

concentration gradient The natural tendency for substances to flow from an area of higher concentration to an area of lower concentration, within or outside the cell.

concussion A transient interruption of normal neurologic function.

conduction Transfer of heat to a solid object or a liquid by direct contact.

congenital adrenal hyperplasia A disease group in which the adrenal glands do not function properly, and as a result do not produce a sufficient amount of the hormone cortisol.

conjugate movement Movement of both eyes together in the same direction.

consent Voluntary agreement by a patient with sufficient mental capacity to capably choose assessment, treatment, or transport offered by the care provider; a patient not accepting care demonstrates the right to refuse.

contact phenomena injuries Injuries that occur as the direct result of trauma to the head, including local effects such as scalp laceration, skull fracture, hematoma, and intracerebral hemorrhage.

contamination A state in which a certain substance, such as blood, infectious material, or a toxic substance, is present on an item or surface; in the case of radiation, it is important to distinguish between this and irradiation.

continuous positive airway pressure (CPAP) A means of raising the breathing baseline above ambient pressure. The increased pressure across the entire breathing cycle increases the mean airway pressure, stents the airway, and increases the functional residual capacity, thereby improving oxygenation.

contrecoup injury A situation in which an impact occurs on one side of the head, causing the brain to move within the cranial vault and forcibly contact the opposite side of the skull, resulting in damage on that side of the brain; also called transitional injury.

convection The mechanism by which heat (body heat in the context of this chapter) is picked up and carried away by moving air currents.

Coombs' test A test used to identify antibodies that may bind to the red blood cells and cause hemolysis.

corpus callosum A large tract of transverse fibers that provides a communication link between the two cerebral hemispheres.

crackle Breath sound produced as fluid-filled alveoli pop open under increasing inspiratory pressure;

can be fine or coarse.

cranial nerves (CN) The 12 nerves arising directly from the brain that govern many of the senses and the functions of muscles in the eyes, face, and pharynx.

C-reactive protein (CRP) An acute phase protein synthesized in the liver; an indicator of inflammation.

creatine kinase (CK) An enzyme that cleaves the high-energy phosphate from creatine in muscle tissues and transfers it to adenosine diphosphate to yield adenosine triphosphate; measurement is used in the assessment for a myocardial infarction.

creatine A major storehouse of intramuscular high-energy phosphate.

creatinine (Cr) The metabolized form of creatine resulting from muscle metabolism and eliminated through the urine. The rate of creatinine clearance is calculated as follows: [(140 – age) × weight in kilograms], divided by [plasma creatinine level × 72].

crew resource management A system that originated as the result of a NASA workshop in 1979 as a means to improve air safety. It incorporates equipment, procedures, and crew concerns to make the best decision during flight operations, focusing on interpersonal communication, leadership, and decision making.

critical care Constant, complex, detailed health care as provided in various acute life-threatening conditions; the ability to deal with crucial situations rapidly and with precision using various advanced machines and devices for treating and monitoring the patient's condition.

critical care patient Any patient who experiences an actual or potential life-threatening illness or injury that requires continual monitoring and care by a specially trained physician, registered nurse, or paramedic.

critical care transport The transport of a patient from an emergency department, critical care unit, or incident scene during which the patient receives the same level of care as was provided in the hospital or originating facility.

critical care transport professional (CCTP) A health care professional who has successfully completed a recognized critical care program and meets the minimum qualifications set forth by the employing transport program.

critical stage The stage of acute hypoxia in which there is almost complete mental and physical incapacitation, resulting in rapid loss of consciousness, seizures, respiratory arrest, and death.

Crohn's disease An inflammation of the GI tract in which all layers of the mucosa may be affected. It results in scattered ulcerations and fibroses throughout the large and small intestines.

cross-tolerance A form of drug tolerance in which patients who take a particular medication for an extended period can build up a tolerance to other medications in the same class.

croup A childhood viral disease characterized by edema of the upper airways with barking cough, difficult breathing, and stridor; also called laryngotracheobronchitis.

cryoprecipitate A blood product created from plasma and in which clotting factors, especially factor VIII, are concentrated; used to treat patients with coagulation disorders.

cumulative effect An effect that occurs when several successive doses of a medication are administered or when absorption of a medication occurs faster than excretion or metabolism.

current The movement of electrons through an electrical circuit over time, measured in amperes.

Cushing's syndrome A condition caused by an excess of cortisol production by the adrenal glands or by

excessive use of cortisol or other similar steroid (glucocorticoid) hormones.

Cushing's triad A cascade of events provoked when intracranial pressure rises to the level of the arterial pressure, vasoconstriction occurs in an effort to shift fluid volumes in the cranium, and the ensuing brain displacement puts pressure on the medulla oblongata by pushing it into the foramen magnum, resulting in disturbances in breathing, heart rate, and blood pressure.

cytokines Chemical messengers that enhance cell growth, promote cell activation, direct cellular traffic, stimulate macrophage function, and destroy antigens; interleukins are a type of cytokine.

daily safety briefing A meeting held at the beginning of each critical care transport shift, in which the daily plan, crew member duties, equipment and aircraft issues, weather expectations, and emergency situations are reviewed.

Dalton's law States that the total pressure of a gas mixture is the sum of the individual or partial pressures of the gases in the mixture; also referred to as the law of partial pressure.

damage In a legal context, damage must result from a breach of duty for a health care provider to be found negligent. Usually, the injury is physical.

dampened waveform A hemodynamic pressure waveform that appears to have lost crisp deflections.

D-dimer test A test of hypercoagulability that detects a fragment from the fibrinolysis process; the test can be used to help diagnose and monitor diseases and conditions related to inappropriate clotting; for example, it can be used to test for deep venous thrombosis.

decompression sickness A condition resulting from exposure to low barometric pressure, causing inert gases normally dissolved in body fluids and tissue to come out of physical solution and form bubbles.

decontamination Use of chemical, physical, or other means to remove, inactivate, or eradicate harmful microorganisms from persons, surfaces, or objects.

deep frostbite A type of frostbite in which the affected part looks white, yellow-white, or mottled blue-white and is hard, cold, and without sensation.

deep partial-thickness burn A burn in which the skin is blistered but not charred and is painful to the touch, usually the result of steam, oil, or flames and involving the deeper layers of the dermis.

delayed hemolytic transfusion reactions A type of transfusion reaction that does not occur until 3 to 7 days after transfusion.

delayed traumatic intracranial hemorrhage (DTICH) Hemorrhage that occurs within the first 3 to 10 days following an injury to the occipital-parietal region via a coup-contrecoup mechanism.

delta wave The slurring of the upstroke of the first part of the QRS complex that occurs in Wolff-Parkinson-White syndrome.

depolarization The process of discharging resting cardiac muscle fibers by an electrical impulse that causes them to contract.

depolarizing paralytics Medications that cause neuromuscular blockade by binding and briefly activating receptor sites at the neuromuscular junction, preventing further activation of these sites and causing chemical paralysis.

depressed skull fracture A type of skull fracture in which a portion of the skull is depressed; the scalp and/or dura may or may not be torn.

dextrocardia A congenital cause of right axis deviation, in which the heart develops in a right-facing

position, creating a mirror image of the normal left-facing heart.

diabetic ketoacidosis (DKA) A form of acidosis in uncontrolled diabetes in which certain acids accumulate when insulin is not available.

diaphragma sellae An extension of the dura mater that forms a roof over the sella turica, which contains the pituitary gland.

diaphragmatic hernia Passage of loops of bowel with or without other abdominal organs, through the diaphragm muscle; occurs as the bowel from the abdomen "herniates" upward through the diaphragm into the chest (thoracic) cavity.

diastatic stellate fracture A fracture involving injury to a bone with separation of an epiphysis; prevalent in abused children.

diastole The relaxation phase of the heart cycle, in which the ventricles are dilated and filling with blood.

diastolic augmentation The increase in aortic pressure during diastole that IAB inflation produces, improving coronary and peripheral perfusion, and that may be thought of as a "second systole."

diastolic augmentation waveforms The waveforms caused by IAB inflation during diastole.

diastolic blood pressure (DBP) The trough or resting pressure in the arterial system that occurs during ventricular diastole.

dicrotic notch The brief increase in aortic pressure reflected in a notching of the wave; caused by the sudden closure and spring back of the aortic valve leaflets; signals the start of diastole.

diencephalon Portion of the cerebrum consisting of the thalamus, the hypothalamus, the subthalamus, and the epithalamus.

differential diagnosis A list of all possible diagnoses, aside from the projected diagnosis, that could be causing a patient's symptoms.

diffuse axonal injury (DAI) A deep-brain injury in which shearing forces damage the integrity of the axon at the node of Ranvier, which consequently alters the axoplasmic flow.

digoxin A cardiac glycoside that produces positive inotropic and negative chronotropic activity in the heart and is primarily indicated in the treatment of chronic heart failure and to control the ventricular rate in atrial tachyarrhythmias.

dilation Opening of the cervix to a maximum of 10 cm, at which point it is considered complete.

dilation and curettage (D&C) A surgical procedure in which the cervix is opened and the uterus scraped of tissue.

diplopia Double vision.

direct bilirubin Conjugated bilirubin; the result of bilirubin's conjugation in the liver, which is ultimately excreted in the bile.

discordant A term that describes T waves that are in the opposite direction from the terminal portion of the QRS complex in bundle branch blocks.

disseminated intravascular coagulation (DIC) A complex condition arising from different causes that activate coagulation mechanisms, resulting in obstructed blood flow as a result of microclots; in fibrinolysis, the attempt by the body to reopen the microcirculation. Bleeding and thrombosis and, potentially, organ dysfunction result.

distal intestinal obstruction Partial or complete obstruction of the distal small bowel (ileum) or large

intestine.

distributive shock A condition that occurs when there is widespread dilation of the resistance vessels, the capacitance vessels, or both.

disturbance stage A stage of hypoxia in which physiologic responses are inadequate to compensate for the oxygen deficiency, and hypoxia is evident.

diverticula Small pouches of tissue that develop as outcroppings of the large intestine, typically in the descending colon and sigmoid colon.

diverticulitis Inflammation of diverticula.

diverticulosis Development of diverticula that may then become inflamed or bleed.

do not resuscitate (DNR) order A type of advance directive that describes which life-sustaining procedures should be performed in case of a sudden deterioration in a patient's medical condition.

doll's eye test An oculocephalic reflex test that is performed on the unconscious patient by rapidly rotating the head from side to side and observing the eye movement.

dorsal Toward the back surface of an object; posterior.

drag The resistance of an airplane to forward motion, directly opposed to thrust.

dromotropic Influencing the conduction rate within the heart.

drug antagonism Medications that bind with a receptor site and prevent activation of these receptors by other medications, without causing receptor activation or related clinical effects.

drug interaction The alteration of the action of a particular medication when combined with another medication.

dual-chamber pacemaker An artificial pacemaker with two leads (one in the atrium and one in the ventricle) so electromechanical synchrony can be achieved.

DUMBELS Mnemonic for symptoms of anticholinesterase inhibitor toxicity: Diaphoresis/diarrhea, Urination, Miosis, Bradycardia/bronchospasm/bronchorrhea, Emesis, Lacrimation, and Salivation.

dura mater The outer membrane of the meninges.

dural venous sinuses Endothelial-lined spaces between the periosteal and meningeal layers of the dura mater.

duration of action How long the medication concentration can be expected to remain above the minimum level needed to provide the intended action.

duty to act A legal obligation of public and certain other ambulance services to respond to a call for help in their jurisdiction. When the duty to act applies varies from state to state; it can be when a CCTP initiates treatment or when the dispatch center confirms that a transport vehicle is being sent.

dysbarism A condition resulting from the effects (excluding hypoxia) of a pressure differential between the ambient barometric pressure and the pressure of gases within the body.

dysconjugate movement Lack of symmetric movement between the two visual axes.

eclampsia The condition that exists once a patient with preeclampsia develops seizures; warning signs of the transition include severe headache, scotomata, hyperreflexia, epigastric pain, and anxiety; the condition causes serious maternal complications.

ectopic pregnancy A pregnancy in which the ovum implants somewhere other than the uterine

endometrium.

Edinger-Westphal nucleus Part of the midbrain that is responsible for mediating the autonomic reflex centers for pupillary accommodation to light.

effective performance time The amount of time an individual is able to perform useful duties in an environment of inadequate oxygen; also known as expected performance time.

efferent pathways Descending pathways that carry motor impulses away from the central nervous system.

ejection fraction The portion of the blood ejected from the ventricle during systole.

electrical alternans An ECG pattern in which the QRS vector changes with each heartbeat. This pattern is pathognomonic of cardiac tamponade.

electrode In the context of a 12-lead ECG, an electrical sensor placed on the chest to record the bioelectrical activity of the heart. In the context of a pacemaker, a conductor in contact with cardiac tissue at the end of a pacing lead; it delivers impulses to that tissue.

electroencephalography (EEG) A procedure that records the electrical activity of the brain by measuring brain waves.

emancipation A legal status in which a person who is younger than the legal age in a given state is legally considered an adult; he or she gives up the right to maintenance and support from a parent, and the parent relinquishes the right to control the child.

embolic stroke Occurs when a blood clot, known as an embolus, forms in one part of the body and travels through the bloodstream to the brain or neck.

emergency locator transmitter (ELT) A device that activates upon the crash of an aircraft and broadcasts a signal that rescuers can use to locate the downed aircraft.

Emergency Medical Treatment and Active Labor Act (EMTALA) A federal law passed by Congress in 1986, with a primary focus of preventing hospitals from transferring patients to other facilities because they are unable to pay for services. Initially, EMTALA was a portion of the Consolidated Budget Reconciliation Act of 1985 (COBRA).

endocardial (transvenous) leads Pacemaker leads guided by angiography and attached to the endocardium.

endocrine hypertension Significant high blood pressure caused by a hormonal disorder; often related to excess hormone produced by a tumor.

endometrium The inner mucous membrane of the uterus.

endoscopy The method of choice for visualization of the GI tract; it allows for observation, diagnosis, and treatment of an insult to the GI tract.

end-stage renal disease (ESRD) A loss of proper kidney functioning, with renal replacement therapy becoming a requirement for survival.

enhanced elimination A process that replaces or augments the body's normal method of eliminating, modifying, or breaking down toxic substances.

enteroclysis Infusion of barium contrast dye into the GI tract to observe for obstruction.

enzymes Proteins that act as catalysts for biochemical reactions within the body.

epicardial leads Pacemaker leads attached to the epicardium (outer surface of the myocardium);

placement and troubleshooting are done in the hospital and require surgery and anesthesia.

epidemiology The study of disease distribution within populations and the factors that determine that distribution.

epidural hematoma (EDH) An injury resulting in the accumulation of blood between the inner periosteum and the dura mater; also called extradural hematoma.

epiglottitis Inflammation of the epiglottis.

epithalamus An area of the cerebrum that is located in the dorsal portion of the diencephalon and contains the pineal gland.

epitope The specific portion of the antigen that is recognized by the antibody or B cell.

eschar The leathery covering of a burn injury, formed after the burned tissues dry out.

escharotomy A surgical incision in an eschar to lessen constriction; sometimes necessary (although rarely performed in a prehospital setting) to prevent edema from building up, impairing capillary filling, and causing ischemia.

esophageal atresia A congenital defect in which the esophagus ends in a blind pouch, often associated with other anomalies such as tracheoesophageal fistula.

esophageal detection device (EDD) A bulb or syringe that is attached to the proximal end of the ET tube; a device used to confirm proper ET tube placement.

esophageal varix Swelling of esophageal veins into the lumen of the esophagus.

etiology The cause of a disease.

eupnea Normal breathing at a rate of 12 to 20 breaths/min in the adult patient.

evaporation The conversion of a liquid to a gas.

exertional hyponatremia A low sodium level as a result of prolonged exertion in hot environments coupled with excessive hypotonic fluid intake that leads to nausea, vomiting, and, in severe cases, mental status changes and seizures.

expiratory reserve volume (ERV) The air that can be expelled from the lungs after a normal exhalation.

external fetal monitoring Electronic heart monitoring of the fetus while in utero, performed via electrodes on the pregnant woman's abdomen.

extinction A test of sensation discrimination in which the CCTP simultaneously touches opposite, corresponding areas of the body, and asks the patient where the touch is felt; it is intended to identify sensory inattention.

extracorporeal membrane oxygenation (ECMO) A supportive intervention which provides oxygenation to a patient by removing the patient's blood to a heart and lung machine that does the work normally done by the lungs and heart. The blood is then transfused back into the patient's circulation.

extradural space A potential space between the cranial bones and the periosteal layer of the dura that becomes a real space only when blood from torn vessels pushes the periosteum from the cranium and accumulates; also called epidural space.

face-to-face intubation Performing intubation at the same level as the patient's face; used when the standard position is not possible. In this position, the laryngoscope is held in the provider's right hand and the ET tube in the left.

false imprisonment Intentional and unjustifiable detention of a patient.

falx cerebelli A fold of dura mater that forms the division between the two lateral lobes of the cerebellum.

falx cerebri A double fold of dura mater that divides the cerebrum into right and left hemispheres by descending vertically into the longitudinal fissure that extends from the frontal lobe to the occipital lobe.

fasciotomy A surgical incision into an area of fascia, for example to relieve pressure between two compartments; not usually performed in a prehospital setting.

FAST An ultrasound examination directed at identifying the presence of free intraperitoneal or pericardial fluids, performed by transducing four distinct areas of the abdomen; stands for Focused Assessment with Sonography for Trauma.

fecal-oral route The route of transmitting infectious organisms from one individual to another; commonly, enteric bacteria or viruses are shed in the feces, spread via contamination of food or water sources, and ingested by other individuals.

Federal Aviation Administration (FAA) A US governmental agency within the Department of Transportation that regulates and oversees civil aviation within the United States; established and enforces the Title 14 CFR that govern aircraft operation within the United States.

Fick principle A method of indirectly determining cardiac output, in which the amount of oxygen uptake of blood as it passes through the lungs is equal to the oxygen concentration difference between mixed venous and arterial blood; the formula uses assumed values for oxygen consumption derived from basal metabolic studies on healthy subjects, which may or may not be valid in critically ill patients.

Fick's law States that the net diffusion rate of a gas across a fluid membrane is proportional to the difference in partial pressure, proportional to the area of the membrane, and inversely proportional to the thickness of the membrane.

first-order elimination The plasma concentration of a medication or substance is proportional to its rate of elimination.

first-pass effect Medications absorbed from the intestine that are partially inactivated as they pass through the hepatic circulation into the liver before entering the systemic circulation; also called first-pass metabolism.

fissures Deep grooves between adjacent gyri of the brain.

fixed-wing aircraft Airplanes; these are capable of transporting critically ill and injured patients a distance of 150 miles or greater.

FLACC scale A pain assessment scale that scores points for five categories, including face, legs, activity, cry, and consolability.

flat fracture The least common type of depressed skull fracture, in which the depressed segment does not have any connection with the cranial vault.

flexion-extension injury A spinal cord injury that results from forward movement of the head, typically as the result of rapid deceleration, or from a direct blow to the occiput.

flicker vertigo An imbalance in brain cell activity caused by exposure to low-frequency flickering or flashing light. Light flickering from 4 to 20 times per second can precipitate reactions, including nausea, migraines, unconsciousness, and seizures.

flight following The practice of maintaining constant contact with other local traffic and air traffic controllers.

flight surgeon A physician who specializes in flight medicine and has been trained extensively in various aspects of aviation and the effects of flight on the human body; also specializes in working with pilots and flight crew members.

flow-cycled ventilator A positive-pressure ventilator that ends inspiration when a predetermined flow rate is achieved.

flow rate The speed of the gas at which the tidal volume is delivered.

focal ischemic stroke Occurs when an area of marginally perfused tissue, the ischemic penumbra, surrounds a core of ischemic cells, and the cells will eventually die without medical intervention.

fomite An inanimate object that is capable of transmitting infectious organisms from one individual to another.

footling breech A delivery presentation in which both feet extend into the birth canal; this is the only malpresentation in which field delivery may be considered.

foramen magnum The opening at the base of the skull through which the bundle of nerve fibers constituting the spinal cord exits.

foramen of Luschka An opening at the base of the fourth ventricle that leads to the subarachnoid space and is essential for the normal flow of cerebrospinal fluid; part of the brain's ventricular system.

foramen of Magendie An opening at the base of the fourth ventricle that leads to the subarachnoid space and is essential for the normal flow of cerebrospinal fluid; part of the brain's ventricular system.

foramen of Monro An opening in the skull that connects the two lateral ventricles with the third ventricle, a central cavity; part of the brain's ventricular system.

Foster–Kennedy syndrome A tumor or abscess at the base of the frontal lobe that impacts the olfactory nerve.

fraction of inspired oxygen (F_{IO_2}) Percentage of inhaled oxygen expressed as a decimal. For example, 40% oxygen = F_{IO_2} of 0.40.

frank breech A delivery presentation in which the buttocks present first, both of the infant's hips are flexed, and the feet are near the fetus' head.

Frank-Starling curve States that the force of the cardiac muscle contraction is proportional to the amount of stretch placed on the muscle fibers (meaning that the more the heart is stretched by the incoming blood, the more forcefully the ventricles contract and the more blood that is ejected); demonstrates how changes in ventricular preload lead to changes in stroke volume.

Frank-Starling law A law of physiology that states that the greater the myocardium is stretched, the greater the force of contraction.

fresh-frozen plasma (FFP) A blood product in which uncoagulated plasma has been separated from the red blood cells; primarily composed of water, proteins, salts, metabolites, and clotting factors.

frontal lobe The largest of the four lobes of the brain; it lies underneath the frontal bone of the skull and is separated posteriorly from the parietal lobe by the central fissure and inferiorly from the temporal lobe by the lateral fissure; responsible for a variety of cognitive and motor functions.

frostbite Localized damage to tissues resulting from prolonged exposure to extreme cold.

full-thickness burn A burn that extends through the epidermis and dermis into the subcutaneous tissues beneath, in which skin is pale, painless, leathery, and charred; also called a third-degree burn.

fulminant hepatic failure A sudden and significant insult to the liver characterized by encephalopathy and a mortality rate of up to 90%.

functional residual capacity (FRC) The amount of air remaining in the lungs after normal expiration; the sum of residual volume and expiratory reserve volume.

functioning adenoma A type of pituitary lesion in which overproduction of hormones occurs.

fundus The optic disk, macula, and blood vessels on the back wall of the internal eyeball.

Galveston formula A formula used to calculate fluid resuscitation for children, which takes into consideration the BSA burned and the TBSA of the child, with half of the amount given in the first 8 hours after injury and the second half over the next 16 hours.

gas lumen The lumen or port of the IAB catheter that carries helium between the IAB and the pump console to inflate and deflate the balloon.

gastritis Inflammation of the gastric mucosa that occurs when the equilibrium of offensive and defensive mechanisms for the lining is altered.

gastroschisis A centrally located, full-thickness abdominal wall defect.

Gay-Lussac's law States that the pressure of a gas when volume is maintained constant is directly proportional to the absolute temperature for a constant amount of gas. Simply stated, as pressure increases, volume increases.

gestation Period of time elapsed from conception to birth. For humans, the full period is normally 40 weeks.

gigantism A syndrome that results from excessive growth hormone, secreted by the pituitary gland before the epiphyseal plate has closed; characterized by the growth of bones, muscles, and many internal organs at an abnormally fast rate.

Glasgow Coma Scale (GCS) An evaluation tool used to determine level of consciousness, which evaluates and assigns point values (scores) for eye opening, verbal response, and motor response, which are then totaled; effective in helping predict patient outcomes.

global ischemic stroke Results when severe hypotension or cardiac arrest produces a transient drop in blood flow to all areas of the brain.

glomerular filtration rate (GFR) A benchmark for comparison of nephron function, based on the amount of fluid filtered by the glomerulus per minute.

gluconeogenesis Glucose formation.

glycogenolysis The breakdown of glycogen to glucose.

glycosuria The presence of glucose in the urine.

Graham's law States that the rate at which gases diffuse is related inversely to the square root of their densities.

gravida The number of pregnancies a woman has had, including those not carried to term.

gravitational forces Force changes that occur with acceleration and deceleration.

gyri Convolutions on the surface of the cerebrum that functionally increase the cortical surface area.

half-life (T1/2) The time period required to eliminate one half of the plasma concentration. During the second "half-life," an additional 25% of the original plasma concentration is eliminated. After three half-lives, one-eighth of the original plasma concentration remains.

halo test A test for leaking CSF that is accomplished by collecting and assessing fluid that drains from the nose, mouth, or ears; a "halo" consisting of a dark red circle surrounded by a lighter yellowish one is a positive halo sign.

hapten A small-molecular-weight nonprotein molecule that is capable of reacting with a specific antibody but cannot elicit the formation of antibodies unless bound to a carrier protein; also called a partial or incomplete antigen.

hard landing An excessively firm contact with the ground, potentially resulting in injury or aircraft damage.

Hashimoto's disease A condition that occurs when the immune system attacks the patient's thyroid gland. The leading cause of hypothyroidism; also known as chronic lymphocytic thyroiditis.

health care–associated pneumonia (HCAP) A pneumonia that occurs in nonhospitalized patients who have contact with health care facilities or personnel.

Health Insurance Portability and Accountability Act (HIPAA) A law enacted in 1996, providing for criminal sanctions and civil penalties for releasing a patient's protected health information (PHI) in a way not authorized by the patient.

heat cramps Acute and involuntary muscle pains, usually in the lower extremities, the abdomen, or both, that occur because of profuse sweating and subsequent sodium loss in sweat.

heat exhaustion A clinical syndrome characterized by volume depletion and heat stress that is thought to be a milder form of heat illness and on a continuum leading to heat stroke.

heat stroke The least common and most deadly heat illness, caused by a severe disturbance in thermoregulation, usually characterized by a core body temperature of more than 104°F (40°C) and altered mental status.

heat syncope An orthostatic or near-syncopal episode that typically occurs in nonacclimated people who may be under heat stress.

Helicobacter pylori A bacterium that causes most peptic ulcers.

helicopter emergency medical service (HEMS) Use of a rotor-wing aircraft to deliver air medical service, for which the goals are to rapidly transport CCTPs to a patient, stabilize the patient's condition, and rapidly transport that patient to a tertiary care center.

helicopter shopping The practice of making sequential calls to numerous air medical providers in an attempt to find a service that will accept a mission request that has been declined by other services based on safety factors such as poor weather, limited landing zone availability, exceptional distances, or other factors.

HELLP syndrome A hemolytic disorder that occurs during the latter stages of gestation, usually after the 20th week, and whose clinical findings include hemolytic anemia, elevated liver enzyme levels, and a low platelet count; patients will present with blurred vision, abdominal pain, headache, and edema.

hematemesis Coffee-ground emesis containing partially digested blood.

hematochezia Stool streaked with bright red blood, originating from the lower GI tract.

hematocrit The percentage of formed elements (that is, cells) in a venous blood sample.

hematuria The presence of red blood cells in the urine.

hemiblocks Blocks of one of the fascicles of the left bundle branch, the left anterior or left posterior fascicle.

hemoconcentration Decreased fluid in the blood, which means that concentrations of other blood components increase.

hemodialysis (HD) Use of vascular access through a fistula or AV shunt to access the blood directly. Solute separation occurs across an artificial semi-permeable membrane within the dialysis machine.

hemoglobin (Hgb) The protein responsible for carrying oxygen to the body's cells and, to a lesser extent, carbon dioxide back to the lungs.

hemoglobinuria The presence of hemoglobin in the urine.

hemolysis A situation in which red blood cells are destroyed, resulting in the release of hemoglobin and red blood cell remnants into the bloodstream; sometimes follows massive blood transfusions or blood transfusion reactions.

hemolytic transfusion reaction A type of transfusion reaction caused by ABO or Rh incompatibility, intradonor incompatibility, improper crossmatching, or improper blood storage.

hemolytic uremic syndrome (HUS) A serious condition in which the patient develops acute renal failure, and which also causes anemia and thrombocytopenia; thought to be caused by several bacterial and viral infectious organisms, most often verotoxin-producing strains of *E coli*.

hemoperfusion A treatment in which a patient's blood is filtered outside the body through a substance that removes toxic substances from it; a method of removing toxic substances from the blood.

hemorrhagic conversion After the brain tissue surrounding the stroke has died, renewed blood flow to the region, for example as triggered by medication, is no longer held in place by the tissue, resulting in hemorrhage.

Henry's law States that the amount of gas dissolved in solution is directly proportional to the pressure of the gas over the solution.

hepatic encephalopathy Encephalopathy secondary to hepatic disease.

hepatitis Inflammation of liver cells that can impede proper functioning of the liver and lead to chronic conditions such as cirrhosis.

hepatitis C virus (HCV) The virus that is the most common cause of cirrhosis and is especially pathogenic in causing hepatitis.

hepatoiminodiacetic acid (HIDA) scan Administration of a radiologically labeled chemical specific for bile incorporation to a patient; the path taken by the marker is observed through radiography to ascertain whether an obstruction is present.

hepatojugular reflex A reflex tested in assessment in which midabdominal pressure is applied while observing for jugular venous distention; if noted, the test result is positive and indicates volume overload.

hepatotoxicity Capable of damaging the liver.

hernia A protrusion of an organ from its tissue lining.

hexaxial system The system developed to describe the coronal plane that is created by the limb leads (I, II, III, aVR, aVL, and aVF).

high-altitude cerebral edema (HACE) An altitude illness in which there is a change in mental status and/or ataxia in a person with acute mountain sickness or the presence of mental status changes and ataxia in a person without acute mountain sickness.

high-altitude pulmonary edema (HAPE) An altitude illness characterized by dyspnea at rest, cough, severe weakness, and drowsiness that may eventually lead to central cyanosis, audible rales or wheezing, tachypnea, and tachycardia.

high-level disinfection Treatment used on inanimate objects to kill all microorganisms but which does *not* kill bacterial spores.

hippus phenomenon A pattern of pupil response to light in which there is rapid constriction of the pupil followed by dilation; can be normal or signify compression of cranial nerve III.

Hirschsprung's disease A congenital lack of ganglion nerve cells in a portion of the distal intestines leading to poor intestinal peristalsis, constipation, and, if not diagnosed and treated, potentially death.

histotoxic hypoxia Hypoxia caused by the inability of the tissues to use oxygen, usually as a result of poisoning by toxins such as carbon monoxide and cyanide.

homeostasis Stability in the body's internal environment.

hospital-acquired pneumonia (HAP) Pneumonia that occurs in a hospitalized patient within 48 hours or more after admission to the hospital that was not apparent at the time of admission.

hot loading Loading a patient into a helicopter while the helicopter is running.

hot zone The area immediately surrounding the chemical source at a hazmat scene.

human leukocyte antigen (HLA) An antigen present on the cell membrane surface of circulating platelets, white blood cells, and most tissue cells.

humidity The degree of moisture in the air, expressed as a percentage.

hydroureteronephrosis Dilation and obstruction of a ureter.

hypemic hypoxia Hypoxia caused by a decrease in the oxygen-carrying capacity of the blood due to a reduced amount of hemoglobin in the blood or a reduced number of red blood cells; also known as anemic hypoxia.

hypercalcemia An increased level of calcium in the blood.

hypercapnia Greater than normal amounts of carbon dioxide in the blood.

hyperchloremia An abnormally high level of chloride in the blood.

hyperchloremic acidosis A form of metabolic acidosis in which low bicarbonate (base) concentration occurs along with an increase in chloride concentration.

hyperdynamic state A condition that occurs as the first stage of distributive shock and is characterized primarily by high cardiac output and low peripheral vascular resistance; also known as warm shock.

hyperemic An increase in blood flow into a tissue or organ; congested with blood.

hyperkalemia An abnormally high level of potassium in the blood.

hyperosmolar hyperglycemic nonketotic syndrome (HHNS) A metabolic derangement that occurs principally in patients with type 2 diabetes, and is characterized by hyperglycemia, hyperosmolarity, and absence of significant ketosis.

hyperpnea A breath that is deeper than normal; can lead to low levels of Co_2.

hyperreflexia A condition of overactive reflexes that can involve excessive twitching or spasms.

hyperresonance An abnormal respiratory sound that is exaggerated or increased beyond normal and low in pitch.

hypersensitivity Occurs when a patient reacts with exaggerated or inappropriate allergic symptoms after coming into contact with a substance the body perceives as harmful.

hyperthyroidism A condition caused by an increased production of T3 (triiodothyronine) and T4 (thyroxine) from the thyroid gland, resulting in an increase in the body's organ function, and characterized by an increase in body temperature, gradual weight loss, increased and/or irregular heart rate, sweating, and irritability.

hypertrophy An increase in the size of the cells as the result of synthesis of more subcellular components, leading to an increase in tissue and organ size.

hypoalbuminemia An abnormally low level of albumin in the blood.

hypocalcemia A low level of calcium in the blood.

hypochloremia An abnormally low level of chloride in the blood.

hypodynamic state A condition that occurs as the second stage of distributive shock and is characterized primarily by a subnormal temperature, a low white blood cell count, profound hypotension and hypoperfusion, and a sudden drop in cardiac output; also known as cold shock.

hypokalemia An abnormally low level of potassium in the blood.

hyponatremia An abnormally low level of sodium in the blood.

hypoplastic left heart syndrome (HLHS) Underdevelopment of the aorta, aortic valve, left ventricle, and mitral valve; this defect involves the entire left side of the heart.

hypopnea A shallow breath; can lead to increased Co_2 levels and decreased oxygen levels.

hypothalamic-pituitary axis An interrelationship between the hypothalamus and pituitary gland in which a releasing or an inhibiting factor is sent from the hypothalamus to the pituitary, resulting in an increase or a decrease in metabolism and other functions throughout the body.

hypothalamo-hypophyseal portal system The venules between the capillaries in the hypothalamus and pituitary gland by which the hypothalamus sends releasing or inhibiting factors to the pituitary gland, thereby increasing or decreasing metabolism.

hypothalamus The most inferior portion of the diencephalon; it is responsible for control of many body functions, including heart rate, digestion, sexual development, temperature regulation, emotion, hunger, thirst, and regulation of the sleep cycle.

hypothermia A condition in which the core body temperature decreases to less than 95°F (35°C).

hypothyroidism A condition caused by a deficiency of T3 (triiodothyronine) and T4 (thyroxine) from the thyroid gland, resulting in a slowing of the body's organ function, and characterized by a decrease in body temperature, gradual weight gain, and increased risk for acute myocardial infarction and cerebrovascular accident.

hypotonia Low or poor (floppy) muscle tone.

hypovolemic shock A condition that occurs when the circulating blood volume is inadequate for delivering adequate oxygen and nutrients to the body.

hypoxia A state of oxygen deficiency in the body, which is sufficient to cause an impairment of function.

Hypoxia is caused by the reduction in partial pressure of oxygen, inadequate oxygen transport, or the inability of the tissues to use oxygen.

hypoxic hypoxia Hypoxia caused by a decrease in the amount of oxygen in the blood due to a reduction in oxygen pressure in the lungs, a reduced gas exchange area, exposure to high altitude, or lung disease.

hypoxic ischemic encephalopathy (HIE) Damage to cells in the central nervous system (the brain and spinal cord) from inadequate oxygen.

I time The time frame for the delivery of the tidal volume.

I:E ratio An expression for comparing the length of expiration to inspiration. The normal ratio is 1:2. This means that expiration is twice as long as inspiration. It is not measured in seconds.

iatrogenic Any adverse physical condition that results from medical treatment.

iatrogenic response An adverse condition inadvertently induced in a patient by the treatment given.

idiosyncrasy An abnormal (and usually unexplained) reaction by a person to a medication to which most other people do not react.

ileus A lack of movement of GI contents in the absence of an obstruction.

immediate hemolytic transfusion reactions A type of transfusion reaction that usually occurs soon (between 1 and 2 hours) after the transfusion of incompatible red blood cells.

immunocompromised Unable to mount a normal immune response as the result of disease (such as AIDS) or chemotherapy treatment.

immunodeficiency An abnormal condition in which some part of the body's immune system is inadequate, and consequently resistance to infectious disease is decreased.

impedance Resistance to the flow of current along an electrical pathway, measured in ohms.

imperforate anus A congenital condition where the anal opening is either absent or displaced.

implantable cardioverter-defibrillator (ICD) A small, battery-powered electrical impulse generator that is implanted in patients at risk for sudden cardiac death as the result of ventricular fibrillation or pulseless ventricular tachycardia.

implantable pulse generator (IPG) The largest implanted element in a pacemaking system, containing the battery and control circuitry.

implied consent Assumption on behalf of a person unable to give consent that he or she would have consented.

inborn error of metabolism A class of genetic diseases mostly due to defects of single genes that code for enzymes that facilitate conversion of various substances into others leading to accumulation of substances which are toxic or interfere with normal function.

incomplete breech A delivery presentation in which the buttocks present first and one foot extends into the birth canal.

incomplete spinal cord injury A disruption of the tracts of the spinal cord in which the patient retains some degree of cord-mediated function.

incorporation In the radiologic context, a process in which radioactive materials enter the body during radiation exposure, causing ongoing internal exposure and creating devastating individual patient toxicity.

incubator An enclosed, clear plastic, heated bed that keeps the infant warm.

indicator conditions Rare or unusual diseases associated with an immunocompromised state. Also called AIDS-related diseases.

indifferent stage The stage of altitude hypoxia in which the body is able to compensate for the hypoxia induced by low barometric pressures.

indirect bilirubin A by-product of the metabolism of red blood cells that is unconjugated and, therefore, not water soluble.

inflammatory bowel disease (IBD) Term covering two colon inflammation pathologies: ulcerative colitis and Crohn's disease.

informed (express) consent A patient's voluntary agreement to be treated after being told about the nature of the disease, the risks and benefits of the proposed treatment, alternative treatments, and the choice of no treatment at all.

inhibition The presence of one medication that decreases the effect of another medication.

Injury Severity Score (ISS) A trauma scoring system that adds the squares of the three highest abbreviated injury scale scores to create a score between 1 and 75 that accounts for multiple injuries, with 1 being a minor injury and 75 being an injury with a high mortality rate.

innate immune system The primary antigen and immunogennonspecific defense mechanism that protects the host by eliminating microbes to prevent infection and other antigens in an attempt to prevent allergic reactions.

innate immunity Immunity to diseases inherent in the genetic makeup of an individual.

inotropic Affecting the force with which muscle tissue contracts, especially cardiac muscle.

inspiratory capacity (IC) The maximum amount of air that can be inspired; the sum of inspiratory reserve volume and tidal volume.

inspiratory reserve volume (IRV) The amount of air that can be inhaled after a tidal volume is inhaled.

instrument flight rules (IFR) A mode of flight used when adverse weather conditions exist, visibility is poor, or cloud cover is low. The pilot may not be able to see outside the aircraft, must rely on instruments, and must be in constant contact with an air traffic controller who assists in maintaining proper separation from other air traffic.

instrument meteorological conditions (IMC) Weather conditions (eg, cloudiness or low visibility) in which a pilot must fly under instrument flight rules, depending on instruments to guide the aircraft.

insulin resistance A condition in which the pancreas produces enough insulin but the body cannot effectively use it.

interference A direct biochemical interaction between two drugs.

intermediate-level disinfection Treatment used on inanimate objects to kill *M tuberculosis*, vegetative bacteria, and most viruses and fungi, but which does *not* kill bacterial spores.

internal capsule A massive bundle of efferent and afferent fibers connecting the various subdivisions of the brain and spinal cord.

International Civil Aviation Organization (ICAO) An agency of the United Nations that defines standards for international air navigation and, as part of its role in developing safe practices, requires crew resource management.

international normalized ratio (INR) A comparative rating of a patient's prothrombin time to help standardize the prothrombin time when planning treatment.

intervertebral foramen A space in the middle of the vertebra that allows the exit of a peripheral nerve root and spinal vein as well as the entrance of a spinal artery on both sides at each vertebral junction.

intra-aortic balloon pump (IABP) therapy A procedure involving a balloon inserted into the aorta and connected to a pump via a catheter, helping to increase the blood flow to the coronary arteries during diastole (inflation) and decrease afterload of blood from the left ventricle (deflation).

intra-atrial conduction delay Delayed conduction within one of the atria, often associated with left or right atrial enlargement.

intra-atrial pathways The anterior or Bachman bundle, middle bundle, and posterior internodal system, through which the electrical impulse passes after the SA node; represented by the P wave on the ECG; also called intranodal pathways.

intracerebral hemorrhage Direct bleeding into the brain parenchyma.

intracranial pressure (ICP) The pressure exerted by brain tissue, intracranial vascular contents, and cerebrospinal fluid in the closed, nondistensible cranial cavity.

intracranial temperature (ICT) Core brain temperature or homeostatic mean gradient temperature of 38.4°C.

intrarenal A type of acute renal failure caused by damage of an actual nephron.

intussusception An event where one part of the intestine folds into another part of the intestines, leading to a blockage.

invasive hemodynamic monitoring A term describing methods for assessing the physiologic condition of the three principle components of the cardiovascular system: heart, vascular network, and fluid volume; mainly assesses the capability of a patient's heart to pump the requisite amount of blood to the body, but also can assess compliance, tone, resistance of the vascular network, and fluid status; includes a variety of pressure values and other measurements.

invasive ventilation Application of mechanical ventilation through an artificial airway such as a tracheostomy or ET tube.

involuntary (autonomic) nervous system The sympathetic and parasympathetic branches of the nervous system, whose fibers connect the structures of the CNS with smooth muscle, cardiac muscle, and glands.

ionic bond A type of chemical bond formed between oppositely charged ions.

ionized calcium Calcium that is not bound or chelated (also called free calcium); its value is useful in assessing for renal failure, nephrotic syndrome, acid-base derangements, and decreases or elevations in chelating compounds.

irradiation Exposure to radiation; it is important to distinguish between this and contamination because health care providers and emergency responders are not placed at any risk by patients who have been exposed but are not contaminated.

ischemic stroke Occurs when an artery to the brain has been blocked by a thrombus, embolus, trauma, or vasospasm (often due to drugs).

isobaric pressurization system A system in which the aircraft cabin is pressurized and the cabin altitude is maintained at a constant pressure even as ambient pressure decreases. This type of pressurization is

found in most cargo planes and passenger aircraft.

isoelectric When referring to a wave, the wave is neither positive nor negative.

isovolumetric contraction The early stage of ventricular contraction during which ventricular blood volume is unchanging because all valves are closed (the semilunar valves have not yet opened).

isovolumetric relaxation The early stage of ventricular relaxation during which ventricular blood volume is unchanging because all valves are closed (the AV valves have not yet opened).

Jackson-Pratt drain A surgical drain used to remove fluid buildup from the wound site during the postoperative healing process.

jejunostomy tube (J tube) A feeding tube placed through the abdominal wall into the jejunum.

joule A measurement of energy.

jugular venous bulb oximetry A technique in which a sampling catheter is placed in the internal jugular vein and directed upward so that its tip rests in the jugular venous bulb at the base of the brain; samples of blood can then be drawn to measure mixed venous oxygen saturation (SVO_2).

JumpSTART triage A sorting system for pediatric patients younger than 8 years or weighing less than 100 lb. There is a minor adaptation for infants because they cannot ambulate on their own.

Kehr's sign Left shoulder pain that may indicate a ruptured spleen. Right shoulder pain may indicate trauma to the liver.

Kernig sign Resistance and pain elicited on extension of the leg at the knee, with the patient in the supine position and the hips flexed perpendicular to the trunk; when pain occurs as a result of this test, the test result is positive.

ketone bodies Organic products of fat catabolism—specifically, acetoacidic acid, acetone, and beta-hydroxybutyric acid.

ketonuria The presence of ketone bodies in the urine.

Kussmaul's respiration A fast and deep respiratory pattern without any periods of apnea. The rate and depth are greater than the normal rate expected; breathing is labored, with periods of deep breaths punctuated by sighs.

lactate The form of lactic acid that is physiologically present in the body.

lactate dehydrogenase (LDH) An enzyme that catalyzes the metabolism of pyruvate (the end product of glycolysis) to lactate in the absence of a functioning citric acid cycle.

lactic acidosis A form of acidosis caused by an excess accumulation or impaired excretion of lactate, leading to an elevated anion gap; can result from exposure to various toxic substances, inadequate tissue perfusion in various shock states, dysfunction of certain organs, nutritional deficiency, infection, malignancy, diabetes, or hereditary metabolic disorders.

lead In the context of the 12-lead ECG, the designated position of the electrode, or the name of the electrode placement. In the context of a pacemaker, an insulated wire that carries signals in a pacemaking system between the implantable pulse generator and the heart tissue.

Le Fort criteria A categorization of facial fractures involving the maxilla that are differentiated based on the location of fracture lines and the extent of mobility of facial structures on physical examination.

left anterior fascicle The portion of the electrical conduction system responsible for innervating the anterior and superior areas of the left ventricle. It is a single-stranded cord terminating in the Purkinje

cells.

left posterior fascicle The portion of the electrical conduction system responsible for innervating the posterior and inferior areas of the left ventricle. It is a widely distributed, fanlike structure terminating in the Purkinje cells.

left ventricular stroke work index A calculation of the contractility of the left ventricle indexed to the patient's body surface area; same as stroke volume index.

left ventricular end-diastolic pressure The pressure exerted on the left ventricle at the end of diastole; the normal value is 4 to 12 mm Hg and is measured by using a pulmonary artery catheter.

leukocytosis An abnormally high number of white blood cells.

leukotrienes A class of biologically active compounds that occur naturally in leukocytes and that produce allergic and inflammatory reactions.

Lhermitte phenomenon A condition in which forward flexion of the neck produces an electric shock feeling, usually running down the back.

lift The upward force created by the wings moving through the air, which sustains the airplane in flight.

ligament of Treitz A small ligament supporting the small intestine at the junction between the duodenum and jejunum. It serves as the dividing point between the upper and lower GI tract.

limb leads The ECG lead electrodes attached to the limbs that form the hexaxial system, dividing the heart along a coronal plane into the anterior and posterior segments.

limbic lobe (rhinencephalon) Part of the temporal lobe that is the seat of emotions and instincts.

linear skull fracture A type of skull fracture characterized by a single fracture line.

linear stellate fracture A fracture with multiple linear fractures radiating from the site of impact.

lipase A pancreatic hormone that metabolizes lipids.

lipid disorders A group of disorders that cause a change in the production or use of cholesterol and that also may cause a change in the way cholesterol is circulated or processed in the body.

liquefactive necrosis Tissue death caused by bacterial or fungal infections in which cellular destruction has occurred, leaving a lesion filled only with pus and the liquid remains of the tissue.

lithotripsy Use of external vibrations to break up gallstones.

liver function test (LFT) A test for liver damage that measures enzymes that normally appear in liver cells but may spill out into the vasculature with parenchymal damage.

long QT syndrome A prolonged QT interval on the ECG primarily caused by a congenital disorder and that under certain conditions tends to deteriorate into ventricular tachyarrhythmias and can lead to syncope or sudden cardiac death; the patient loses consciousness, often without warning.

long-term variability A pattern of changes in the baseline fetal heart rate occurring within 30-second intervals rather than from beat to beat; due to the parasympathetic-sympathetic interplay in the fetal heart rate.

low-level disinfection Treatment used on inanimate objects to kill most bacteria, some viruses, and some fungi, but which does *not* kill *M tuberculosis* and does *not* kill bacterial spores.

lumbar puncture A procedure in which a needle is inserted into the lumbar portion of the back and into the subarachnoid space to obtain spinal fluid for testing or to administer drugs.

Lund and Browder chart A detailed version of the rule of nines chart that takes into consideration the changes in body surface area brought on by growth.

magnetic resonance imaging (MRI) A noninvasive diagnostic imaging technique used to visualize the internal structure and function of the body, which works by using a powerful magnet to align water molecules present in body compartments; an image is obtained from this alignment and the speed at which molecules alter or release.

major burn A classification from the American Burn Association that includes: (1) partial-thickness burns involving more than 25% of BSA in adults or 20% of BSA in children younger than 10 years and adults older than 50 years; (2) full-thickness burns involving more than 10% of BSA; (3) burns involving the face, eyes, ears, hands, feet, or perineum that may result in functional or cosmetic impairment; (4) burns caused by caustic chemical agents; (5) high-voltage electrical injury; (6) burns complicated by inhalation injury or major trauma; and (7) burns sustained by high-risk patients.

malignant hyperthermia A condition that can result from certain anesthesia medications (notably succinylcholine) and is characterized by muscle spasms, rigidity, acidosis, hyperthermia, tachycardia, tachypnea, myoglobinuria, rhabdomyolysis, and hyperkalemia.

Mallampati classification A system for predicting the relative difficulty of intubation based on the amount of oropharyngeal structures visible in an upright, seated patient who is fully able to open his or her mouth.

Mallory-Weiss syndrome Longitudinal laceration of the esophagus, often following severe vomiting.

malrotation A congenital anomaly of rotation of the midgut, in which the small bowel is found predominantly on the right side of the abdomen.

mass A tumor, aneurysm, or abscess.

mean airway pressure The amount of positive pressure in the airway, averaged over the inspiratory and expiratory phases of the breathing cycle.

mean arterial pressure (MAP) The average (or mean) of the systolic and diastolic blood pressures (SBP and DBP); MAP = DBP + 1/3(SBP – DBP).

mean electrical axis The sum of all electrical impulses.

mechanical ventilation The application of a device that provides varying degrees of ventilatory support.

mechanism of action The way in which a medication produces the intended response.

meconium A dark green fecal material that accumulates in the fetal intestines and is discharged around the time of birth.

medical control The oversight designed to ensure that actions taken by providers on behalf of patients are appropriate; divided into direct medical control, available in real time via radio, and indirect medical control such as standing orders and protocols.

medical direction Supervision of medical care, provided online (telephone or radio) or off-line (protocols, standing orders, education, and quality improvement).

medulla oblongata The lowermost portion of the brain stem.

melena Black, tarry stool containing partially digested blood, with bleeding originating from the upper GI tract.

metabolic acidosis A pathologic condition (blood pH < 7.35) resulting from the accumulation of acids in

the body caused by any number of systems in the body, including the gastrointestinal system, or major organ failure.

metabolic alkalosis A pathologic condition (blood pH > 7.45) resulting from the accumulation of bases in the body caused by any number of systems in the body, including the gastrointestinal system, or major organ failure.

metabolic syndrome A group of symptoms that together may lead to coronary artery disease, stroke, and type 2 diabetes; usually related to predisposition to a lipid disorder.

methamphetamine washout syndrome Excessive sleep, hunger, and depression as a result of excessive amphetamine use.

microcirculation Circulation that occurs in the microvasculature, the body's smallest vessels (arterioles, capillaries, and venules).

midbrain A small area of the brain stem extending between the diencephalon, the pons, and the third ventricle.

mild hypothermia A condition in which the core body temperature is between 90°F and 95°F (32.2°C and 35°C); at this stage, the body usually compensates with increased thermogenesis and interrupted thermolysis.

mild TBI Concussion; a traumatically induced physiologic disruption of brain function that occurs without structural damage.

Mini-Mental State Examination A simple, easily applied test of higher cognitive functions.

Minnesota esophagogastric tamponade tube A tube that is placed to stop bleeding of esophageal varices. It is similar to the Sengstaken-Blakemore tube, but has a built-in suction catheter.

minor burn A classification from the American Burn Association that includes: (1) partial-thickness burns involving less than 15% of BSA in adults or 10% of BSA in children and older adults; and (2) full-thickness burns involving less than 2% BSA that do not present a serious threat of functional or cosmetic risk to the eyes, ears, face, hands, feet, or perineum.

minute volume Total volume of air breathed in and out in 1 minute. It is calculated by multiplying the respiratory rate per minute by the tidal volume.

mittelschmerz pelvic pain Abdominal pain occurring midway between menstrual periods, at the time of ovulation and from the ovulation site.

mixed acidosis A pathologic condition in which there is a low pH (< 7.35), an elevated PCO_2 level (> 45 mm Hg), and a low bicarbonate level (< 23 mmol/L), which occurs when there is both a respiratory and a metabolic cause present at the same time.

mixed alkalosis A pathologic condition in which there is an elevated pH (> 7.45), a low PCO_2 level (< 35 mm Hg), and an elevated bicarbonate level (> 29 mmol/L), which occurs when there is both a respiratory and a metabolic cause present at the same time.

mixed central venous oxygen saturation ($ScvO_2$) The percentage of oxygen bound to hemoglobin in blood returning to the right side of the heart from the head and upper body, representative of oxygen extraction by the head and upper extremities.

mixed venous oxygen saturation (SvO_2) The percentage of oxygen bound to hemoglobin in blood returning to the right side of the heart, representative of global oxygen extraction.

mobile intensive care units Ambulances or helicopters that are used only for maintaining specialized or intensive care treatment; used primarily for interfacility transports.

mode The particular way in which a spontaneous or mechanical breath is delivered.

moderate burn A classification from the American Burn Association that includes: (1) partial-thickness burns of 15% to 25% of BSA in adults or over 10% to 20% of BSA in children and older adults; and (2) full-thickness burns involving 2% to 10% of BSA that do not present a serious threat to functional or cosmetic impairment of the eyes, ears, face, hands, feet, or perineum.

Monro–Kellie doctrine A theory developed by two Scottish anatomists, who stated that the central nervous system is enclosed in a rigid compartment along with cerebrospinal fluid, whose total volume tends to remain constant; an increase in any component—whether brain, blood, or CSF—will cause an increase in pressure and decrease the volume of one of the other elements.

morbidity The number of nonfatally injured or disabled people. Usually expressed as a rate, meaning the number of nonfatal injuries in a certain population in a given time period divided by the size of the population.

Moro reflex An infant reflex in which, when an infant is caught off guard, the infant opens his or her arms wide, spreads the fingers, and seems to grab at things.

mortality Deaths caused by injury and disease; usually expressed as a rate, meaning the number of deaths in a certain population in a given time period divided by the size of the population.

motor area Part of the frontal lobe that contains pyramidal cells that control voluntary motor function on the opposite side of the body.

mucosa The outermost layer of the alimentary canal. It consists of three sublayers: surface epithelium, lamina propria, and muscularis mucosae.

multiple organ dysfunction syndrome (MODS) Altered organ function in acutely ill patients, which is diagnosed when two or more organs stop functioning.

Murphy's sign Pain when pressure is applied to the right upper quadrant of the abdomen in a specific manner; helps detect gallbladder problems.

muscularis externa The third tissue layer of the alimentary canal. It contains two levels in most places: the circular layer and the longitudinal layer.

myocardial oxygen consumption (MVO_2) The volume of oxygen that the heart muscle consumes; an expression of the level of oxygen demand in the heart.

myoglobin An oxygen-carrying heme protein present in high concentrations in the cytoplasm of cardiac and skeletal muscles.

myoglobinuria The presence of myoglobin, a respiratory pigment of muscle tissue, in the urine.

myxedema coma A rare, life-threatening condition that can occur in patients who have severe, untreated hypothyroidism, and which is characterized by altered mental status and lethargy, failure of the thermoregulatory system, and a precipitating event; may be accompanied by auditory and visual hallucinations, seizures, or unresponsiveness.

National Transportation Safety Board (NTSB) An independent federal agency that promotes transportation safety, including aviation, railroad, highway, maritime, pipeline, and hazardous materials safety; investigates transportation crashes to identify the cause and make safety recommendations.

NBG code A code of five letters used to categorize pacemakers by their functions and capabilities, developed by a joint effort of North American and British electrophysiology groups.

near–SIDS An acute episode in which an infant experiences apnea accompanied by choking or gagging, skin color change, and a noted change in muscle tone; also called an ALTE (apparent life-threatening event).

necrotizing fasciitis A limb- or life-threatening infection of the soft tissue that affects the subcutaneous tissues, fat, and fascia.

negative-pressure ventilator A mechanical ventilator that operates using pressure that is less than ambient (atmospheric) pressure.

negligence Failure to provide the quality of care (as defined by applicable standards) usually expected with conditions requiring that care; negligence is established when the plaintiff proves four elements: duty to act, breach of the duty, injury to the patient, and the breach was the direct cause of the injury.

neonate An infant during the first 28 days after birth.

neurocranium The part of the skull that encloses and protects the brain.

neurogenic shock Circulatory failure caused by paralysis of the nerves that control the size of the blood vessels, leading to widespread dilation; seen in spinal cord injuries.

neuroleptic malignant syndrome (NMS) A potentially fatal reaction to antipsychotic, antischizophrenic, and antiemetic medications, characterized by patient hyperthermia, profound muscle rigidity, metabolic acidosis, and altered mental status.

neutrophils One of the types of leukocytes (white blood cells); these numerous phagocytic microphages usually are the first of the mobile phagocytic cells to arrive at an area of injury or infection.

newborn An infant within the first few hours after birth.

nicotinic receptors Cholinergic receptors that bind with the neurotransmitter acetylcholine.

nitrogen narcosis A state resembling alcohol intoxication produced by nitrogen gas dissolved in the blood at high ambient pressure.

nondepolarizing agents Medications designed to cause temporary paralysis by binding in a competitive but nonstimulatory manner to part of the ACh receptor; they do not cause fasciculations.

nonfunctioning adenoma A type of pituitary lesion in which the tumor does not secrete any hormones.

noninvasive ventilation Application of mechanical ventilation through a mask, mouthpiece, or other interfaces other than an artificial airway.

normal pressure hydrocephalus (NPH) An accumulation of CSF that causes the ventricles of the brain to enlarge. The enlarged ventricles of a patient with this condition may not cause increased intracranial pressure.

normal range A range of values encompassing the results that 95% of healthy people would have for the particular test.

nosocomial Infections acquired during hospitalization or a nursing home stay.

nuchal rigidity Marked resistance to head movement in any direction that is suggestive of meningeal irritation.

obstructive diseases Diseases that result in difficulty with moving air out of the lungs, such as asthma, COPD, cystic fibrosis, and bronchioectasis.

occipital lobe The lobe of the brain that occupies the most posterior portion of the cerebrum; it is the primary receptive area for vision, specifically the interpretation of visual stimuli.

off-line medical control Medical direction given through a set of protocols, policies, and/or standards.

Ohm's law The principle given by the equation V = IR, which states that applied voltage is equal to the current times the resistance of the circuit.

oligohydramnios Decreased volume of amniotic fluid during a pregnancy; a risk factor associated with abnormalities of the urinary tract, postmaturity (birth after a prolonged pregnancy), and intrauterine growth retardation.

omphalocele Herniation of abdominal contents into the base of the umbilical cord.

online medical control A type of medical direction in which the care provider is in direct contact with a physician, usually via two-way radio or telephone.

onset of action The time needed for the concentration of the medication at the target tissue to reach the minimum effective level.

operational control The person who has the ultimate authority to initiate, conduct, and terminate the EMS mission; in an air medical program, usually the chief pilot or operational manager.

opioid syndrome Toxicity that develops following illicit use and abuse of opioids.

opioids The class of chemicals that includes naturally derived opiates and newer artificial medications with the same properties.

opsonize To make bacterial cells more susceptible to the action of phagocytosis by the action of binding an antibody to the pathogen's cell membrane.

optic disk The most prominent structure visible in the eye; it represents the termination of the optic nerve.

osmolality The amount of dissolved substance in 1 kg of water.

osmolarity The amount of dissolved substance in 1 L of water.

osmosis The diffusion of water across membranes.

osmotic pressure The pressure created in a space divided by a semipermeable membrane owing to differences in concentrations of solutes found in the solutions on either side of the membrane.

ostomy A surgically created opening through which feces can be voided in the absence of some or all of the large intestine or rectum.

ovarian cyst A fluid buildup within an outcropping of tissue from the ovary. If such a cyst is ruptured, significant bleeding can occur.

ovarian torsion Twisting of an ovary about its ligaments, resulting in ischemia and possibly necrosis.

overtriage Considering a patient to be in more serious condition than he or she actually is.

oxygen consumption ($\dot{V}O_2$) The amount of oxygen used by the cells and tissues.

oxygen delivery (DO_2) The amount of oxygen delivered to the tissues each minute.

oxygen extraction fraction (OEF) The fraction of oxygen extracted from the blood as it passes by to maintain normal oxygen delivery and, consequently, normal brain functions.

oxygen extraction ratio (ERO_2) The relationship between oxygen consumption and oxygen delivery, or the cells' ability to utilize oxygen.

oxygen hood A tent placed over the head of an infant for the purpose of delivering supplemental oxygen.

oxygen paradox A condition that results when a hypoxic person rapidly breathes in 100% oxygen; he or she may experience sudden dizziness, which is quickly resolved followed by complete restoration of function. The condition is possibly due to the sudden constriction of dilated arteries in the brain.

pacemaker syndrome The occurrence of symptoms relating to the loss of atrioventricular synchrony in ventricularly paced hearts or symptoms caused by inadequate timing and ventricular contractions in paced hearts.

pacing circuit The conduction pathway along which the pacing impulse flows; formed by a power source, one or two lead-electrode pairs, and body tissue.

pacing impulse The electrical impulse sent to the heart to stimulate the heart to beat.

packed red blood cells (PRBCs) A blood product that retains all of the characteristics of whole blood with the exception of the extraction of approximately 250 mL of platelet-rich plasma from each unit of whole blood.

palpable purpura Larger areas of bleeding under the skin (greater than 3 mm).

pancreatitis Inflammation of the pancreas leading to tissue destruction, improper functioning of the pancreas, and chronic disease.

panel Groups of related tests that are performed as a single unit; also called profile.

papillary muscles A type of muscle in the ventricle from which the chordae tendineae extend and attach to the cusps of the AV valves.

paracentesis A "tapping" of the abdomen with a needle to draw off ascites.

parietal lobe The lobe of the brain situated directly posterior to the frontal lobe on the other side of the central fissure; it is largely responsible for sensory functions.

parity The number of pregnancies a woman has had that were carried to more than 28 weeks' gestation.

Parkland formula A formula that recommends giving 4 mL of lactated Ringer's solution or normal saline for each kilogram of body weight, multiplied by the percentage of body surface area burned; sometimes used to calculate fluid needs during lengthy transport times.

paroxysmal nocturnal dyspnea Severe shortness of breath occurring at night after several hours of recumbency, during which fluid pools in the lungs; the person is forced to sit up to breathe; caused by left-sided heart failure or decompensation of chronic obstructive pulmonary disease.

paroxysmal supraventricular tachycardia A supraventricular tachycardia that starts and ends abruptly.

partial pressure of oxygen (arterial) (Pa_{O_2}) The amount of the total pressure in the blood contributed by oxygen; a value measured when analyzing arterial blood gas level.

partial pressure of oxygen (P_{O_2}) A measurement of the amount of oxygen dissolved in the blood.

partial-thickness burn A burn that involves the epidermis and part of the dermis, characterized by pain and blistering; also called a second-degree burn.

partial thromboplastin time (PTT) A value that represents the intrinsic coagulation pathway's clotting ability; also known as activated partial thromboplastin time (aPTT).

passive immunity Short-lived immunity acquired from placental transfer, antibodies in the mother's milk, or antiserum administered IV.

passive leg raising (PLR) A technique used to assess fluid responsiveness in patients with suspected volume depletion, performed by raising both of the patient's legs to a 45° angle and then taking the blood pressure; improvement in blood pressure suggests that the patient will benefit from fluid administration.

patent ductus arteriosus (PDA) A situation in which the ductus arteriosus, which assists in fetal circulation, does not transition as it should after birth to become the ligamentum arteriosum; the result is that the connection between the pulmonary artery and the aorta remains, allowing some oxygenated blood to move back into the heart rather than all of it moving out of the aorta and into the systemic circulation.

pathogens The microorganisms responsible for activating the immune system and that cause disease; includes viruses, parasites, fungi, and bacteria.

pathologic edema Systemic swelling, which may have several causes, including preeclampsia.

peak airway pressure The amount of positive pressure generated by the ventilator to deliver the tidal volume.

Pediatric assessment triangle (PAT) An assessment tool that allows rapid formation of a general impression of the type and level of illness or injury in an infant or child without touching him or her; consists of assessing appearance, work of breathing, and circulation of the skin.

pelvic inflammatory disease (PID) An infection of the female upper organs of reproduction, specifically the uterus, ovaries, and fallopian tubes.

penile fracture A rupture of one of the blood-containing sacs in the penis, resulting in deformity and possible loss of function.

peptic ulcer An erosion of the mucosal lining of the GI tract.

percutaneous endoscopic gastrostomy (PEG) tube A feeding tube that is placed through the abdominal wall into the stomach.

perfusionists Highly trained technicians who are intimately familiar with the operation of intra-aortic balloon pumps and adult and pediatric extracorporeal membrane oxygenation machines and who may assist during any medical situation, including critical care transports, in which it is necessary to support or temporarily replace a patient's circulatory or respiratory function.

pericardial tamponade Impairment of diastolic filling of the right ventricle as a result of significant amounts of fluid in the pericardial sac surrounding the heart, leading to a decrease in the cardiac output.

pericardiocentesis A procedure in which a needle or angiocath is introduced into the pericardial sac to relieve cardiac tamponade.

pericarditis An inflammatory process involving the pericardium.

peripheral blood smear A glass slide containing a drop of blood; analysis allows the lab to microscopically examine the structure of the blood cells.

peristalsis A general term applicable to any concerted and directional movement in the body such as the movements that push food or urine through the body.

peritoneal dialysis Use of a catheter to introduce dialysate solution into the abdomen using the peritoneum as a natural semipermeable membrane to separate solutes.

persistent pulmonary hypertension (PPHN) The persistence of elevated pressures in pulmonary

vasculature after birth, associated with failure to transition from fetal circulation to postpartum or normal newborn circulation. The typical presentation consists of hypoxia, low APGAR scores, hypoglycemia, or a congenital diaphragmatic hernia.

petechial lesions Small areas of bleeding under the skin (pinpoint).

pharmacodynamics The branch of pharmacology that studies reactions between medications and living structures, including the processes of body responses to pharmacologic, biochemical, physiologic, and therapeutic effects.

pharmacokinetics The study of the metabolism and action of medications with the particular emphasis on the time required for absorption, duration of action, distribution in the body, and method of excretion.

pheochromocytoma A catecholamine-producing benign tumor of chromaffin cells located in the center of the adrenal gland, which can occur either sporadically or chronically as a result of genetic risk factors; causes stimulation of alpha-adrenergic and beta-adrenergic receptors, resulting in hypertension, increased cardiac contractility, glycogenolysis, gluconeogenesis, intestinal relaxation, and increased heart rate.

phlebostatic axis An imaginary point located at the fourth intercostal space, mid-chest level, which serves as an external landmark for the right atrium.

phosphate buffer system The buffer system that functions in the renal tubules and intracellular fluids to convert strong acids or bases into weak acids or bases so that there is only a minimal effect on overall pH.

phosphodiesterase An enzyme that helps break phosphodiester bonds, creating smaller nucleotides.

physiologic zone The zone that extends from sea level to 10,000′ and is the area of the atmosphere to which humans are well adapted. The barometric pressure is sufficient in this zone to facilitate adequate oxygenation. The changes in pressure encountered with rapid ascents or descents within this zone can produce ear or sinus trapped-gas problems.

physiologically deficient zone The zone that extends from 10,000′ to 50,000′. Noticeable physiologic deficits occur above 10,000′. A decrease in barometric pressure results in oxygen deficiency, causing hypoxic hypoxia. In this zone, the manifestation of trapped and evolved gases occurs. The use of pressurized aircraft and/or supplemental oxygen is necessary in this zone.

pia mater The innermost layer of the meninges, which rests directly on the brain or spinal cord.

Pierre Robin syndrome A condition present at birth marked by a very small lower jaw (micrognathia). The tongue tends to fall back and downward (glossoptosis) and there is a cleft soft palate.

ping-pong ball fracture A pediatric greenstick fracture of the skull.

plasma protein fraction A blood product that contains 83% albumin and 17% globulins.

platelet (Plt) count A measurement of the number of platelets in the blood, which is useful for assessing a patient's coagulation status.

pleural friction rub The result of an inflammation that causes the pleura to thicken, decreasing the pleural space and allowing the surfaces of the pleura to rub together.

plexus A cluster of nerve roots that permits peripheral nerve roots to function as a group.

P mitrale A double-humped, M-shaped P wave that is 120 milliseconds wide or greater with the tops of the humps 40 milliseconds apart or greater; found in limb leads I, II, and III; represents left atrial enlargement.

pneumomediastinum The collection of air within the mediastinum (the space within the chest that contains the heart, major blood vessels, vagus nerve, trachea, and esophagus; located between the two lungs).

pneumoperitoneum The collection of air within the peritoneum (the membrane in the abdomen encasing the liver, spleen, diaphragm, stomach, and transverse colon).

point-of-care (POC) testing Laboratory testing that is performed at the point of care, for example, the bedside, so that results can be quickly obtained and considered while decisions are being made about patient care.

polydipsia Excessive thirst, resulting in excessive intake of fluid.

polyhydramnios An excessive amount of amniotic fluid. May cause preterm labor.

polyphagia Excessive desire to eat, resulting in overconsumption of food.

polyuria Frequent and plentiful urination.

pons Part of the brain stem located between the midbrain and the medulla oblongata; it relays information to and from the brain and spinal cord along fiber tracts.

poor R-wave progression An abnormal R-wave pattern; one of the factors that may signify anterior infarction.

portal hypertension An increase in vascular resistance through the hepatic portal system. It can cause high venous pressure in gastric and esophageal veins, leading to varices, among other problems.

positive end-expiratory pressure (PEEP) The amount of pressure above ambient pressure present in the airway at the end of the respiratory cycle.

positive-pressure ventilator A mechanical ventilator that operates using pressure that is greater than ambient pressure.

posterior cord syndrome Extension injury that produces dysfunction of the dorsal columns, presenting as decreased sensation to light touch, proprioception, and vibration.

postrenal A type of acute renal failure caused by downstream obstruction of urine flow from the kidneys, with initially intact nephrons; commonly caused by a blockage of the urethra by prostate enlargement, renal calculi, or strictures.

potentiation The effect of increasing the potency or effectiveness of a drug or other treatment; may occur by administering two medications concurrently, where one increases the effect of the other.

P pulmonale A tall P wave that is 2.5 mm high or greater, found in leads II and III; indicates right atrial enlargement.

PR interval The interval of time that occupies the space between the beginning of the P wave and the beginning of the QRS complex.

precision A measure of how a value is likely to be the same every time a test is performed.

precordial leads Another term used to describe the chest leads in an ECG.

preeclampsia A condition of late pregnancy that involves gradual onset of hypertension, headache, visual changes, and swelling of the hands and feet; also called toxemia of pregnancy.

prefrontal area Part of the frontal lobe that provides control of thought, concentration, depth and ability to think abstractly, memory, and autonomic nervous system response, concomitant to emotional change.

pregnancy-induced hypertension (PIH) The occurrence of hypertension in a pregnant woman, usually after 20 weeks' gestation, defined as a blood pressure of 140/90 mm Hg or greater in a woman with a previously normal blood pressure, or a rise of 30 mm Hg systolic and/or 15 mm Hg diastolic in a woman with baseline hypertension; can lead to preeclampsia if uncontrolled.

preload The amount of blood that flows into the ventricle passively from the atria; related to the overall volume status of the patient; measured by right atrial pressure or central venous pressure and wedge pressure.

premature Underdeveloped; refers to infants born before 36 weeks from the first day of the last menstrual period.

premotor area Part of the frontal lobe that is adjacent to the motor area and helps coordinate certain movements.

prerenal A type of acute renal failure caused by decreased perfusion of an intact nephron; often reversible if the underlying condition can be found and perfusion can be restored to the kidney.

pressure ulcer A sore on the skin arising from prolonged pressure, classified in four stages, with stage 4 being the most severe with tissue necrosis and muscle and bone involvement. All stages are painful and prone to infection; commonly seen in patients confined to a bed.

pressure ventilator A type of positive-pressure ventilator that ends the delivery of the tidal volume based on a predetermined pressure; therefore, the volume is variable.

preterm labor Labor occurring before 36 weeks' gestation, leading to early birth.

priapism Prolonged, painful erection of the penis.

primary aldosteronism A type of aldosteronism usually caused by a tumor of a single adrenal gland that overproduces aldosterone; also known as Conn's syndrome.

primary amyloidosis The most common form of amyloidosis, affecting the heart, kidneys, tongue, nerves, and intestines; classified as apolipoprotein.

primary apnea The initial response to asphyxia at birth, characterized by increased respiratory rate and a slight decrease in heart rate with increased blood pressure; responds to stimulation and oxygen therapy.

primary decontamination A form of decontamination that usually occurs at a scene or outside a health care facility or transport vehicle to protect rescuers and health care providers from exposure to a toxic substance during patient care and transport.

primary MODS Multiple organ dysfunction syndrome that results from a direct insult such as trauma.

primary spinal cord injury Spinal cord injury that occurs at the moment of impact.

prodromal Referring to the early signs and symptoms that occur before a disease or condition, such as a toxic exposure, fully appears, for example, dizziness before fainting.

projected diagnosis An assumed decision on the medical condition of a patient from preliminary investigation; assists in the provision of initial treatment and projection of further diagnostic testing requirements.

prophylaxis Treatment of disease before it occurs (ie, to prevent the disease).

proprioception The ability to perceive the position and movement of one's own body or limbs.

proprioceptive Referring to information that comes from receptors located in the skin, muscles, tendons,

and joints; this information helps a person know the position of his or her body.

protected health information (PHI) Individually identifiable information (such as name, social security number, and date of birth), health information (such as laboratory results and medical history), and demographic information (such as address and telephone number) that is protected by HIPAA.

proteinuria Protein levels detected in the urine.

prothrombin time (PT) A value that represents the extrinsic coagulation pathway's clotting ability by taking into account various clotting factors, fibrinogen, the prothrombin ratio, and the international normalized ratio.

proximal intestinal obstruction Obstruction at or above the level of the jejunum.

proximate (direct) cause The reason an injury occurred; the final element that must be proven for a health care provider to be found negligent.

pseudo-Cushing's state A condition in which a person has higher cortisol levels from a cause other than actual Cushing's syndrome; these causes include depression, alcoholism, malnutrition, and panic attack.

ptosis Drooping of an eyelid.

pulmonary artery catheter (PAC) A catheter with a balloon near its tip that is passed through a vein into the right side of the heart, through the right ventricle, and into the pulmonary artery; records the pressure transmitted back from the left atrium; also referred to as a Swan-Ganz catheter.

pulmonary artery pressure The pressure measured in the pulmonary artery, usually displayed with a pressure waveform and digital systolic, diastolic, and mean values.

pulmonary capillary wedge pressure (PCWP) The mean pressure measured while occluding the pulmonary artery with a balloon-tipped catheter proximal to the site of measurement; reflects left atrial pressure (also called pulmonary artery wedge pressure and pulmonary artery occlusion pressure).

pulmonary overpressurization syndrome (POPS) Also called "burst lung," this diving emergency can occur during ascent and can cause pneumothorax, mediastinal and subcutaneous emphysema, alveolar hemorrhage, and the lethal arterial gas embolism.

pulmonary stenosis Narrowing of the pulmonary valve.

pulmonary vascular resistance (PVR) The resistance or impedance to ejection of the right ventricle of the heart.

pulse CO-oximetry A noninvasive screening and monitoring method for methemoglobinemia and carbon monoxide exposure.

pulse pressure The difference between systolic and diastolic blood pressures.

pyelonephritis An infection of the kidney, typically the result of a urinary tract infection that traveled up the ureter.

QRS complex Deflections in the ECG produced by ventricular depolarization.

quiescent A latent state as in a dormant virus or bacterium, in which an infected patient will exhibit little to no symptoms.

raccoon eyes Orbital fractures and hemorrhage into the surrounding tissue; also called periorbital ecchymosis.

radiation Emission of heat from an object into surrounding, colder air.

rapid decompression Occurs when a large leak or hole develops in a pressurized aircraft; can result in hypoxia and injury to people inside the aircraft and catastrophic failure of the aircraft.

reactive airway disease (RAD) A term used to describe any condition that causes hyperreactive bronchioles and bronchospasm.

reactive gastritis A chronic mucosal edema that results from recurring contact of the mucosa with antagonistic substances such as bile, pancreatic juice, or NSAIDs.

reciprocal changes An ECG pattern in which a lead shows a pattern that is the opposite of the one shown in the lead located 180° from the other; for example, the electrode over the area of infarction records ST-segment elevation, whereas the electrode over the lead that is 180° away records ST-segment depression.

red blood cell (RBC) count A measure of the total number of erythrocytes in the blood.

referencing The process of ensuring that the hemodynamic pressure transducer is at the level of the left atrium.

refractory period A short period immediately after depolarization in which the myocytes are not yet repolarized and are unable to fire or conduct an impulse.

refractory stage A stage in shock in which there is persistently low mean arterial blood pressure despite vasopressor therapy and adequate fluid resuscitation.

relative contraindication A condition that makes a particular treatment or procedure somewhat inadvisable but does not completely rule it out.

relative refractory period The period in the cell-firing cycle at which it is possible but difficult to restimulate the cell to fire another impulse.

renal tubular acidosis (RTA) A form of metabolic acidosis caused by dysfunction of the kidneys or renal system; H^+ cannot be excreted into the urine, even in the presence of overwhelming acidosis. Presents without an elevated anion gap; characterized into Type 1, 2, or 4.

repogle A double-lumen tube used for gastric decompression in infants with gastrointestinal issues; for an infant, usually an 8F size is used and is connected to low intermittent wall suction to collect gastric secretions.

repolarization A state in which the cell becomes more negative, moving away from equilibrium with the extracellular fluid; this is an active process.

residual volume (RV) The amount of air remaining in the lungs after the expiratory reserve volume is exhaled.

resonance Normal lung sound heard with percussion.

respiratory acidosis A pathologic condition (blood pH < 7.35) resulting from the accumulation of acids in the body caused by a breathing problem or insufficient function of the respiratory system.

respiratory alkalosis A pathologic condition (blood pH > 7.45) resulting from the accumulation of bases in the body caused by a breathing problem or insufficient function of the respiratory system.

respiratory depression A low respiratory rate (< 12 breaths/min in adults) for a prolonged period of time; also called hypoventilation.

respiratory failure A situation in which the respiratory system fails to meet the body's metabolic needs. If not reversed, it leads to respiratory or cardiopulmonary arrest.

respiratory insufficiency The inability of the respiratory system to keep up with the metabolic demands of the body.

respiratory syncytial virus (RSV) A labile paramyxovirus that produces its characteristic fusion of human cells in a tissue culture known as the syncytial effect; can affect both upper and lower respiratory tracts but is more prevalent with the lower, causing pneumonias and bronchiolitis.

restrictive diseases Diseases that result in difficulty moving air in to the lungs such as occupational lung diseases, idiopathic pulmonary fibrosis, pneumonia, atelectasis, chest wall deformities and injuries, and neuromuscular diseases that affect breathing.

reticular activating system (RAS) A diffuse system that extends from the lower brain stem to the cerebral cortex; it controls the sleep-wakefulness cycle, consciousness, the ability to direct attention to a specific task, and the perception of sensory input that might alter behavior.

reticular formation (RF) A set of neurons that extends from the upper level of the spinal cord, through the medulla, pons, and midbrain, and into the thalamus and cerebral cortex. It has both excitatory and some inhibitory capabilities, and can enhance, suppress, or modify impulse transmission.

retrograde intubation A technique in which a wire is placed through the trachea and into the mouth with a needle via the cricoid membrane. The ET tube is then placed over the wire and guided into the trachea.

reverse Trendelenburg Supine position with head higher than feet.

revised trauma score A trauma scoring system that rates injury severity by comparing the Glasgow Coma Scale score, the systolic blood pressure, and the respiratory rate and assigning a score between 1 and 13 based on these three values; in some cases the parameters are weighted, resulting in a score of between 1.0 and 7.8408.

Rh factors Antigens found on the cell membrane of red blood cells.

rhabdomyolysis The destruction of muscle tissue leading to a release of potassium and myoglobin, which then accumulate in the blood and urine and impair filtration; occurs most often with crush injuries, electrical burns, or large full-thickness burns. The urine becomes pink, dark red, or rust colored.

rhonchi Rattling vibrations produced as air flows through mucus or around obstruction in the larger airways.

rickettsial diseases Diseases transmitted via tick bite; the three major diseases in this class include Rocky Mountain spotted fever, ehrlichiosis, and anaplasmosis.

right ventricular pressure The pressure in the right ventricle that consists of systolic, diastolic, and mean pressures; important during insertion and use of a PAC.

right ventricular stroke work index A calculation of contractility of the right ventricle indexed to the patient's body surface area.

rotational force An injury-producing force in which the head moves around its center of gravity.

rotation–flexion injury A spinal cord injury to C1-C2, the only area of the spine that allows for significant rotation, in which rotation with abrupt flexion produces a stable dislocation in the cervical spine. In the thoracolumbar spine, rotation-flexion forces typically cause fracture rather than dislocation.

rotor-wing aircraft Helicopters; these are used to transport critically ill and injured patients in rural settings and can travel distances of up to 150 miles.

rubrospinal tract Part of the midbrain that controls the tone of flexor muscles.

rule of 3s A means of prioritizing the basic physiologic needs for survival: The average person can survive 3 minutes without oxygen, 3 hours without shelter, 3 days without water, and 3 weeks without food.

rule of nines A system that assigns percentages to sections of the body, allowing calculation of the amount of skin surface involved in the burn area.

scintigraphy An imaging technology that is similar to angiography, except that the red blood cells themselves are radiologically labeled to allow greater specificity.

SCIWORA An acronym that stands for spinal cord injury without radiographic abnormalities.

scope of practice The body of knowledge, skills, and therapies that health care providers can legally apply in patient care, based on training, certification, medical direction, and applicable law.

secondary aldosteronism A type of aldosteronism that occurs as a result of reduced renal blood flow, which stimulates hypersecretion of aldosterone; can be caused by obstructive renal artery disease, renal vasoconstriction, and edematous disorders.

secondary amyloidosis A form of amyloidosis that occurs as a result of another illness, and which primarily affects the kidneys, spleen, liver, and lymph nodes; classified as amyloid A protein.

secondary apnea The response if asphyxia at birth continues, characterized by a period of gasping respirations, falling heart rate, and falling blood pressure; does not respond to stimulation unless resuscitation is initiated immediately.

secondary decontamination A form of decontamination that is directed at minimizing patient absorption or injury from a toxic substance; occurs after initial decontamination has occurred outside a health care facility or transport vehicle.

secondary MODS Multiple organ dysfunction syndrome that is a slower, more progressive insult to organs and frequently results from the sepsis cascade.

secondary spinal cord injury Spinal cord injury in which multiple factors permit a progression of the primary spinal cord injury; the ensuing cascade of inflammatory responses may result in further deterioration.

Seldinger technique The most common technique for inserting a central venous line; involves inserting a needle with a syringe, then inserting a guide wire into the needle. Once the guide wire is in place, the needle is removed, an incision is made, and a catheter is inserted over the guide wire; the guide wire is then removed.

semilunar valves The valves (aortic and pulmonic) that are the exits from the left and right ventricles into the aorta and the pulmonary artery, respectively.

Sengstaken-Blakemore tube A tube with an inflatable balloon at its end that is inserted into the GI tract and inflated to tamponade bleeding.

sensitivity The ability of a certain test to indicate whether a person has a certain disease; also, a ventilator control that regulates the amount of negative pressure required by the patient to initiate or "trigger" a breath.

sepsis Systemic response to infection manifested by two or more symptoms noted with SIRS.

septic shock A form of shock that occurs in septicemia when endotoxins or exotoxins are released from certain bacteria in the bloodstream, resulting in a persistently low mean arterial blood pressure

despite adequate fluid resuscitation.

seroconversion Development of antibodies, measured in the serum, in response to an infection or vaccine.

serosa A protective layer of connective tissue over most of the alimentary canal; also called the visceral peritoneum.

serotonin syndrome An unusual response to serotonin-altering medications causing hyperserotoninergic symptoms.

severe acute respiratory syndrome (SARS) A highly contagious, potentially life-threatening lower respiratory tract illness that usually starts with flu-like symptoms, including fever, headache, and muscle aches, followed within 2 to 7 days by a dry cough and pneumonia.

severe hypothermia A condition in which the core body temperature drops to less than 90°F (32.2°C).

severe sepsis A type of sepsis with associated acute organ failure; hypoperfusion or hypertension is present.

shoulder dystocia A condition in which the infant's shoulders are too large to travel through the birth canal without additional clinical maneuvering of the infant.

shunt Perfusion without ventilation.

side effects Reactions that can manifest as signs or symptoms that are not desired but are expected based on how the medication works.

single-chamber demand pacemaker A pacemaker with the pacing lead placed in only one chamber of the heart, in which the generator stimulus is inhibited by a signal derived from the heart's depolarization, thus minimizing the risk of pacemaker-induced fibrillation.

SLUDGEM Acronym for Salivation, Lacrimation, Urination, Diarrhea, Gastroenteritis, Emesis, and Miosis, which are symptoms of anticholinesterase inhibitor toxicity.

somatogravic illusion An error in perception that occurs with acceleration, as the otolith organs are displaced rearward, similar to when a person is looking up. This perception of a nose-up altitude may cause the pilot to push the nose of the aircraft down inappropriately at night or in unlit terrain.

space equivalent zone The zone that begins at 50,000′. In this zone, 100% oxygen is not sufficient to prevent hypoxia without the use of a pressurized aircraft or suit. Unprotected personnel may experience boiling of body fluids at a level above 66,500′.

spatial disorientation An error in perception that may result from a person's inability to determine his or her position, altitude, and motion in relation to the surface of the earth or to a significant fixed object during flight.

specialty care transport (SCT) The term used by the Centers for Medicare and Medicaid Services to define ground transportation and medically necessary supplies and services for critically ill and injured patients.

specific gravity The chemical property of a fluid that relates its density to the density of water.

specificity The ability of a certain test to indicate whether a person does not have a certain disease.

spinal shock The temporary local neurologic condition that occurs immediately after spinal trauma; it is characterized by swelling and edema of the spinal cord and can lead to a physiologic transection, mechanically disrupting all nerve conduction distal to the injury.

SpO_2 The noninvasive pulse oximetry measurement of oxyhemoglobin saturation by means of a beam of light applied to a superficial capillary bed such as the digits or ear lobe.

spontaneous abortion Expulsion of the fetus that occurs naturally; also called miscarriage.

stagnant hypoxia Hypoxia caused by a malfunction of the circulatory system resulting in a decrease in blood flow.

standby The situation in which an HEMS provider is notified of a potentially life-threatening emergency call and makes preparations to respond to the call. During standby, the flight crew checks weather data, obtains landing coordinates, and conducts a preflight inspection of the aircraft.

START triage A patient sorting process that stands for simple triage and rapid treatment and uses a limited assessment of the patient's ability to walk, respiratory status, hemodynamic status, and neurologic status.

steady state A point in drug administration at which the rate of administration (frequency and dose) is equal to the rate of elimination, resulting in a constant plasma medication level.

ST-elevation myocardial infarction (STEMI) A myocardial infarction that shows ST-segment elevation on the ECG; patients with STEMI have a high probability of coronary thrombus occlusion.

stepping reflex An infant reflex that occurs when the infant is held up in the air and moves his or her legs up and down as if marching.

stereognosis The ability to sense an object's form through touch.

sterile cockpit The time when unnecessary communication that could distract the pilot is banned in the cockpit—usually during takeoffs, landings, and any other critical phase of flight at the discretion of the pilot in command.

Stevens-Johnson syndrome A milder form of toxic epidermal necrolysis, in which epidermal detachment involves less than 10% of the total body surface area; causes sloughing of the skin, mucous membranes, and cells lining the respiratory system.

strain pattern An ECG pattern that involves ST-segment changes and flipped, asymmetric T waves associated with right or left ventricular hypertrophy.

stress-related erosive syndrome A condition in which small, diffuse peptic ulcers appear in critically ill patients in the intensive care unit, including Cushing's ulcers from head injuries and Curling's ulcers from severe burns.

stridor High-pitched sound representing air moving past fluid or mechanical obstruction within or immediately above the glottic opening.

stroke Results from a disruption of blood flow to the brain that results in neurologic deficit persisting for more than 24 hours.

stroke volume (SV) The amount of blood ejected by the ventricles during each contraction; varies between 60 and 130 mL/beat, with the average being 70 mL.

stroke volume index A calculation of contractility of the left ventricle indexed to the patient's body surface area; same as the left ventricular stroke work index.

ST segment The section of the ECG complex from the end of the QRS complex to the beginning of the T wave; represents the period of inactivity between ventricular depolarization and repolarization; mechanically, represents the time that the myocardium is maintaining contraction.

subarachnoid hemorrhage (SAH) Bleeding between the arachnoid mater and dura mater of the brain.

subdermal burn A severe, life-threatening burn involving the deep structures of muscle, bone, larger blood vessels, and nerves; also called a fourth-degree burn.

subdural hematoma (SDH) Bleeding that accumulates between the dura mater and arachnoid mater.

subdural space The dura-arachnoid junction; this potential space may develop into a real one if a blow to the head causes a loss of blood into the cranial meninges.

submucosa The layer of connective tissue below the mucosa. Blood vessels, lymph, and nerves reside here.

subthalamus An area of the cerebrum that is located below the thalamus and is closely related to the basal ganglia in function.

sudden infant death syndrome The sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation.

sudden sniffing death syndrome Occurs when a surge of catecholamines on a sensitized myocardium causes ventricular fibrillation or ventricular tachycardia; can occur when a person is startled while huffing hydrocarbons.

sulci Grooves between adjacent gyri.

summation effect The process whereby multiple medications can produce a response that the individual medications alone do not produce.

superficial burn A burn involving only the epidermis, producing very red, painful skin; also called a first-degree burn.

superficial frostbite A type of frostbite characterized by altered sensation (numbness, tingling, or burning) and white, waxy skin that is firm to palpation, but the underlying tissues remain soft.

superficial partial-thickness burn A burn involving the epidermis and part of the dermis, but the deeper layers of the dermis are not involved; also called a second-degree burn.

supine hypotensive syndrome Low blood pressure resulting from compression of the inferior vena cava by the weight of the pregnant uterus when the mother is supine.

surfactant A substance formed in the lungs that helps keep the small air sacs or alveoli from collapsing and sticking together; a low level in a premature baby contributes to respiratory distress syndrome.

sympathomimetic Medication that mimics the body's sympathetic nervous system response (fight or flight response); includes epinephrine and norepinephrine.

sympathomimetic syndrome A syndrome that involves overstimulation of the adrenergic nervous system, resulting in tachycardia, hypertension, agitation, seizures, hyperthermia, dilated pupils, and diaphoresis.

synergism An interaction of two or more medications that results in an effect that is greater than the sum of their effects if taken independently.

systemic inflammatory response syndrome (SIRS) A widespread inflammatory process associated with infectious and noninfectious causes, without end-organ damage.

systemic vascular resistance (SVR) The resistance or impedance to ejection of the left ventricle of the heart.

systole The contraction phase of the heart cycle when the ventricles pump blood out of the heart through

the aorta and the pulmonary artery into the systemic and pulmonary circulatory systems.

systolic blood pressure (SBP) Peak pressure in the arterial system that occurs during ventricular ejection or systole.

T wave The upright, flat, or inverted wave following the QRS complex of the ECG, representing ventricular repolarization.

tachyphylaxis A condition in which the patient rapidly becomes tolerant to a medication.

tachypnea An abnormally fast respiratory rate.

tectospinal tract Part of the midbrain that controls reflex motor movements in response to visual and auditory stimuli.

temporal lobe The lobe of the brain that is located beneath the temporal bone of the cranium; its primary functions relate to hearing, speech, behavior, and memory.

tentorium cerebelli A fold of the dura mater that separates the occipital lobes of the cerebrum from the cerebellum and brain stem, thereby dividing the brain into upper and lower compartments.

term Used to describe an infant delivered at 37 to 42 weeks of gestation.

term newborn Used to describe an infant born between 37 and 42 weeks' gestation.

termination of action The amount of time after the medication's concentration falls below the minimum effective level until it is eliminated from the body.

testicular torsion Twisting of a testicle about the spermatic cord to the point of ischemia and possibly necrosis.

tetralogy of Fallot (TOF) A cardiac anomaly that consists of four defects: a ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and an overriding aorta.

thalamus The largest portion of the diencephalons; it acts as a relay station for motor and sensory activity; basic neuronal activity; and memory, thought, emotion, and complex behavior.

therapeutic index The ratio of a drug's lethal dose for 50% (LD_{50}) of the population to its effective dose for 50% (ED_{50}) of the population; a medication's margin of safety.

thermal burn A burn caused by heat, contact with hot objects, ignited liquids, steam, or hot liquids.

thermistor The apparatus used for quickly determining very small changes in temperature.

thermogenesis The production of heat in the body.

thermolysis The liberation of heat from the body.

thermoregulation The process by which the body maintains temperature through a combination of heat gain by metabolic processes and muscular movement and heat loss through respiration, evaporation, conduction, convection, and perspiration.

third spacing A loss of fluids from the intravascular space into the tissues caused by an increase in intravascular pressures and/or increased permeability of the cell membranes. Physical stressors of flight such as temperature, vibration, and changes in gravitational force can cause or aggravate this condition.

thrombocytopenia An abnormally low level of platelets.

thrombotic stroke Occurs when the blood supply to part of the brain is disrupted by a thrombus, or blood clot.

thromboxane A type of substance that causes blood vessels and bronchial smooth muscles to constrict, and promotes blood coagulation by causing platelets to collect.

thrust The force exerted by the aircraft engine, which pushes air backward with the objective of causing a reaction of the airplane in the forward direction.

thyrotoxicosis An excess of thyroid hormones resulting in a hypermetabolic crisis, including tachycardia over 140 beats/min; hyperthermia (sometimes greater than 103.9°F); coma with agitation, nausea, vomiting, diarrhea, and unexplained jaundice; and pulmonary edema; marked by an elevated thyroxine level; also called thyroid storm.

tidal volume (V_T) The volume of air moved into and out of the lungs with each respiratory cycle.

time-cycled ventilator A type of positive-pressure ventilator in which the ventilator ends inspiration after a selected inspiratory time has been achieved.

time of useful consciousness The time between a person's sudden deprivation of oxygen at a given altitude to the point at which deliberate function is lost. With the loss of effective performance during flight, a person is no longer capable of taking proper corrective or protective actions.

timing In the context of IABP therapy, a method for coordinating the IAB inflation-deflation cycle with the cardiac cycle (inflation during diastole and deflation synchronous with systole).

Title 14 Code of Federal Regulations (CFR) Part 135 Established by the FAA, this guideline governs the operations of all commuter or on-demand commercial operations.

Title 14 Code of Federal Regulations (CFR) Part 91 Established by the FAA, this guideline governs the operation of all aircraft within the United States, including the waters within 3 nautical miles of the US coast.

tocolytics A group of medications used to suppress preterm labor.

tolerance Physiologic adaptation to the effects of a drug such that increasingly larger doses of the drug are required to achieve the same effect.

TORCH screen A set of lab values taken to detect congenital infections, including toxoplasma IgG and IgM (1:1,024), rubella titers, urine cytomegalovirus titer, viral culture (for herpes), and culture of scrapings from any vesicular lesions.

torsade de pointes An undulating sinusoidal rhythm in which the axis of the QRS complexes changes from positive to negative and back in a haphazard manner.

tort A wrongful act that gives rise to a civil suit.

total lung capacity (TLC) The maximal amount of air that can fill the lungs; the sum of tidal volume, inspiratory reserve volume, expiratory reserve volume, and residual volume.

total parenteral nutrition (TPN) The IV administration of all necessary nutrients in a patient whose GI tract does not function.

total protein The total quantity of protein in a blood sample.

toxic epidermal necrolysis syndrome A severe skin reaction to certain medications, environmental allergies, and other unknown toxins, in which epidermal detachment is greater than 30%.

toxidrome A group of symptoms, or syndrome, associated with toxicity of a given substance.

tracheal breath sounds Breath sounds heard by placing the stethoscope diaphragm over the trachea or over the sternum; also called bronchial breath sounds.

tracheal deviation The late sign of a tension pneumothorax in which the trachea is tugged to one side of the neck, usually opposite the side of the pneumothorax.

transcranial Doppler (TCD) ultrasound A noninvasive method of assessing the state of intracranial perfusion and monitoring of cerebral blood flow velocity through thinner areas of the skull; used in patients following rupture of an intracranial aneurysm to assess for vasospasm, used to identify intracranial lesions following a stroke, and used for the detection of cerebral blood flow changes associated with increased ICP.

transfusion reaction A reaction resulting from an endogenous or exogenous factor related to transfused blood.

transient ischemic attack (TIA) A temporary disruption in the blood flow to the brain that lasts less than 24 hours with temporary side effects.

transient tachypnea of the newborn Transient respiratory distress, lasting a few hours, in the newborn and which is secondary to retained fetal lung fluid.

transillumination The act of using a light source placed within the trachea to visualize through the thin tissue that covers the trachea.

transjugular intrahepatic portosystemic shunt The placement of a shunt in the abdomen that bypasses much of the hepatic portal system, intended to decease portal hypertension and its effects.

translational force An injury-producing force in which the head's center of gravity moves along a linear path.

transposition of the great arteries (TGA) A defect in which the great vessels are reversed; the aorta is connected to the right ventricle, and the pulmonary artery is connected to the left ventricle.

trauma injury severity score A calculated scoring system that uses the results of the injury severity score, the revised trauma score, and the patient's age to determine survivability rate; rarely used in the transport setting.

trauma score A score from 1 to 16 that takes into account the Glasgow Coma Scale score, respiratory rate, respiratory expansion, systolic blood pressure, and capillary refill, and relates to the likelihood of patient survival; not accurate for patients with severe head injuries.

traumatic asphyxia A condition resulting from severe, sudden crushing injury to the chest and abdomen, forcing blood backward out of the right side of the heart; engorging the veins of the chest, neck, and head; and giving the chest, neck, and head an extremely cyanotic appearance.

trifascicular block The combination of bifascicular block (right bundle branch block with a block in the left anterior fascicle or left posterior fascicle) that occurs with a first-degree heart block (prolonged PR interval).

triple-lumen catheter A type of catheter consisting of three distinct continuous tubes that allow for pressure monitoring, blood sampling, and fluid and drug administration.

troponin A key protein involved in muscle contraction that is present in the serum only after cellular necrosis releases the cellular contents of cardiac muscle (such as after myocardial infarction).

tropopause The space between the troposphere and the stratosphere. It rises to 60,000′ at the equator owing to heated air masses that expand and sinks to about 30,000′ at the poles owing to contracting cold air masses.

troposphere A portion of the earth's atmosphere that extends from the surface of the earth to 5 to 10 miles

high depending on the relation to the equator and the poles. This layer is characterized by the presence of water vapors, a constant decrease in temperature with increasing altitude, and large-scale vertical currents.

true anaphylaxis An anaphylactic reaction that results when the allergen binds to IgE on the cell membranes of basophils and when mast cells stimulate the release of histamine from the cell.

true fracture The most common type of closed skull fracture, in which the depressed segment has contact with the cranial vault.

T-tube A T-shaped tube used to drain bile from the gallbladder.

tumor necrosis factor (TNF) A protein mediator released primarily by macrophages and T lymphocytes that help regulate the immune response.

turbid Cloudy or opaque.

turgor The elasticity of the skin.

two-point discrimination A test of sensation discrimination that measures the shortest distance at which the sides of two separate points of a compass or calipers can be distinguished from one another.

tympany A loud, high-pitched, abnormal lung sound.

type 1 diabetes The type of diabetic disease that usually starts in childhood and requires daily injections of supplemental synthetic insulin to control blood glucose; sometimes called juvenile or juvenile-onset diabetes.

type 2 diabetes The type of diabetic disease that usually starts later in life and often can be controlled through diet and oral medications; sometimes called adult-onset diabetes.

type A gastritis A form of gastritis in which the intrinsic factor secreted by mucosal parietal cells is attacked by autoantibodies, ultimately resulting in pernicious anemia.

type B gastritis A form of gastritis caused by *Helicobacter pylori* and in which the degree of inflammation varies in severity.

Type I spatial disorientation The pilot is unaware of becoming disoriented.

Type II spatial disorientation The pilot realizes that a problem exists but does not recognize it as disorientation.

Type III spatial disorientation A sudden incapacitating form of loss of positional awareness.

type-and-crossmatch The test to determine compatibility between patient serum and donor red blood cells prior to transfusion.

ulcerative colitis An inflammation of the rectal mucosal and submucosal tissues.

ultrasound An imaging technique that uses sound, which is transmitted through an area of the body, then denser material reflects waves back to a transducer that produces an image; typically used for obstetrics, gynecology, and abdominal diagnostics.

unassisted systole Pressure waveform reflecting what would normally occur without IABP assistance.

uncal herniation The most common type of brain herniation, in which a portion of the temporal lobe is displaced, resulting in compression of cranial nerve III, the midbrain, and the posterior cerebral artery.

uncus The medially curved anterior part of the hippocampal gyrus.

undertriage Considering a patient to be in less serious condition than he or she actually is.

unipolar lead A lead in which one of the electrodes is placed in the heart and the other lead is placed in an area of zero potential.

unipolar system A type of pacemaker system in which contact between the pacemaker itself and the body tissue actually forms the ground lead for the implantable pulse generator.

universal donors Persons who have type O blood.

universal gas law States how a hypothetical gas should act if there are no variables affecting it, such as temperature or pressure. Also known as the ideal gas law.

universal recipients Persons who have type AB blood.

urinalysis (UA) Laboratory tests performed on a patient's urine.

urinary tract infection (UTI) A common type of infection of the urinary tract that can progress to major conditions if not treated.

vacuum caput In a newborn, an accumulation of fluid at the site where a vacuum extractor was used to assist in delivering the newborn.

vasospastic disease Vascular constriction or spasm.

Vaughan-Williams classification scheme Classification system for antiarrhythmic medications.

vector An organism that carries a disease-causing microorganism from one organism to another.

ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch A state of inadequate ventilation, perfusion, or both, in which there is inadequate gas exchange.

ventilator-associated pneumonia (VAP) A type of hospital-acquired pneumonia that occurs in ventilated patients and that appears more than 48 hours after endotracheal intubation.

ventral Toward the abdomen; anterior.

ventricular-assist device (VAD) Sometimes an alternative to heart transplantation, this mechanical pump is surgically implanted and helps maintain a heart that can no longer function properly. It has primarily been used to support patients who are waiting for a heart transplant.

ventricular septal defect A hole in the septum separating the ventricles, allowing blood from the left ventricle to flow into the right ventricle.

vertebral body The anterior weight-bearing structure within the spine.

vertex A fetal position in which the head is the lowest part of the fetus, resulting in the head presenting first during delivery; this is the normal delivery presentation.

vertical compression Forces transmitted through vertebral bodies and directed either inferiorly through the skull or superiorly through the pelvis or feet (eg, from a direct blow to the parietal region of the skull or rapid deceleration from a fall through the feet, legs, and pelvis).

vesicular breath sounds Softer, muffled sounds in which the expiratory phase is barely audible.

vestibular Describes organs of equilibrium located in the inner ear.

viremia The presence of viruses in the blood.

virulence Physical or biochemical properties of a disease agent that determine its pathogenicity.

viscerocranium The bones making up the facial skeleton.

visual flight rules (VFR) A mode of flight used when weather conditions are good, meaning there is generally very good visibility and minimal cloud cover; the pilot is responsible for maintaining separation from other aircraft.

vital capacity (V_c) The maximal amount of air that can be exhaled following a maximal inspiration; the sum of tidal volume, inspiratory reserve volume, and expiratory reserve volume; approximately 80% total lung capacity.

volatile Easily changed from liquid to gas form.

voltage The force that causes current to flow in a circuit, measured in volts; also called *amplitude* in a pacing system.

volume ventilator A type of positive-pressure ventilator in which the breath ends when the predetermined tidal volume is achieved.

voluntary (somatic) nervous system The nervous system fibers that connect the structures of the CNS with skeletal muscles and the integument.

volvulus Twisting of the stomach or intestine, which often has the effect of cutting off its blood supply.

$\dot{V}/\dot{Q}$ ratio The relationship between alveolar ventilation and alveolar capillary perfusion. The normal value is 0.8.

warm zone Area surrounding the hot zone at a hazmat incident.

water ditching Crashing into water.

water intoxication A condition that occurs when the normal balance of electrolytes in the body is pushed outside safe limits by the overconsumption of water.

weather decline A situation in which an air medical provider is requested, but the pilot and the flight team determine that the weather does not meet established FAA guidelines and therefore decline to make the flight.

weight The downward force due to the weight (gravity) of the airplane and its load; directly opposed to lift.

Wernicke's area Part of the temporal lobe that is responsible for comprehension of both written and spoken words.

West Nile virus (WNV) An arthropod-borne flavivirus that is spread to birds and humans by mosquitoes, which may be characterized by sudden onset of fever with malaise, anorexia, nausea, vomiting, eye pain, headache, myalgia, rash, lymphadenopathy, fatigue, and arthralgias.

wheeze A high-pitched musical sound caused by airflow through a narrowed or constricted airway.

white blood cell (WBC) count A measure of the total number of leukocytes in the blood.

whole-bowel irrigation (WBI) A gastric decontamination method that involves the patient consuming large quantities of a nonabsorbable, electrolyte-balanced liquid that propels stomach and intestinal contents through the digestive system.

wide complex tachycardia A cardiac rhythm of greater than 100 beats/min with a QRS duration of 120 milliseconds or greater; can be of ventricular or supraventricular origin.

Wolff-Parkinson-White (WPW) syndrome A syndrome characterized by short PR intervals, delta waves, nonspecific ST-T wave changes, and paroxysmal episodes of tachycardia caused by the presence of an accessory pathway.

zeroing A process of calibrating a pressure transducer to eliminate extraneous atmospheric and hydrostatic pressures from the data being measured.

zero-order elimination Medications or chemicals are eliminated from the body at a constant rate, regardless of plasma concentration.

Zollinger-Ellison syndrome A condition in which a tumor in the GI tract secretes large amounts of acids.

zone of coagulation The center of a burn, usually the deepest and most severe area.

zone of erythema The outermost area of a burn, representing the least severe burned area, usually an area of first-degree burn.

zone of stasis The area found just outside the zone of coagulation, representing a burned area that is less severe.

Index

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