# CHAPTER 11 Nursing Care of Clients Experiencing Trauma and Shock

## LEARNING OUTCOMES

- Describe the components and types of trauma.
- Discuss causes, effects, and initial management of trauma.
- Describe steps of the primary survey to diagnose and manage life-threatening injuries.
- Discuss diagnostic tests used in assessing clients experiencing trauma and shock.
- Describe collaborative interventions for clients experiencing trauma and shock, including medications, blood transfusion, and intravenous fluids.
- Explain organ donation and forensic implications of traumatic injury or death.
- Discuss the risk factors, etiologies, and pathophysiologies of hypovolemic shock, cardiogenic shock, obstructive shock, and distributive shock.
- Use the nursing process as a framework for providing individualized care to clients experiencing trauma and shock.
- Describe the role of the nurse in trauma prevention education and evaluate a plan of care to restore the functional health status of trauma clients.
- Understand and comply with guidelines related to the Uniform Anatomical Gift Act.

## CLINICAL COMPETENCIES

- Obtain initial data about the trauma client to include history taking, assessment, review of past medical history, and communication with prehospital and other healthcare providers and family members.
- Evaluate client response to medical and surgical interventions for clients sustaining multiple trauma and shock.
- Communicate significant data and changes in the condition of a client who has sustained trauma.
- Formulate nursing diagnoses based on manifestations recognized during the nursing assessment.
- Develop a plan of care for the trauma client based on scientific knowledge and client diversity.
- Advocate for clients' rights as indicated by documents that address end-of-life issues.

***MEDIALINK***

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone

KEY TERMS

**abrasions,** *259*
**anaphylactic shock,** *276*
**blunt trauma,** *256*
**brain death criteria,** *265*
**cardiac output (CO),** *269*
**cardiogenic shock,** *274*
**contusions,** *259*
**distributive shock,** *274*
**full-thickness avulsion injuries,** *259*
**hypovolemic shock,** *273*
**lacerations,** *259*
**mean arterial pressure (MAP),** *269*
**minor trauma,** *256*
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**obstructive shock,** *274*
**penetrating trauma,** *256*
**pneumothorax,** *258*
**puncture wounds,** *259*
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**shock,** *268*
**stroke volume (SV),** *269*
**tension pneumothorax,** *258*
**transfusion,** *262*
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# THE CLIENT EXPERIENCING TRAUMA

**Trauma** is defined as injury to human tissues and organs resulting from the transfer of energy from the environment. In the past the term *trauma* has been associated with the word *accident*. *Accident* means that the injury occurred without intent, a result of random chance. We now know that a considerable number of injuries are preventable and not of random chance. Intentional and nonintentional trauma encompass a variety of injuries resulting from motor vehicle crashes, pedestrian injuries, gunshot wounds, falls, violence toward others, or self-inflicted violence. The injuries, disabilities, and deaths resulting from these acts constitute a major healthcare challenge.

## FAST FACTS

- Trauma kills more people between the ages of 1 and 44 than any other disease or illness.
- 62 percent of all deaths from ages 15 to 24 are due to trauma. (NIGMS, 2006)

Trauma usually occurs suddenly, leaving the client and family with little time to prepare for its consequences. Nurses provide a vital link in both the physical and psychosocial care for the injured client and family. In caring for the client who has experienced trauma, nurses must consider not only the initial physical injury, but also its long-term consequences, including rehabilitation. Trauma may alter the client's previous way of life, potentially affecting independence, mobility, cognitive thinking, and appearance.

## Components of Trauma

Trauma results from an abnormal exchange of energy between a host and a mechanism in a predisposing environment. The *host* is the person or group at risk of injury. Multiple factors influence the host's potential for injury: age, sex, race, economic status, preexisting illnesses, and use of substances such as street drugs and alcohol.

The *mechanism* is the source of the energy transmitted to the host. The energy exchanged can be mechanical, gravitational, thermal, electrical, physical, or chemical. Table 11–1 lists the most common mechanisms for each type of energy. Mechanical energy is the most common type of energy transferred to a host in trauma. The most common mechanical source of injury in all adult age groups is the motor vehicle.

TABLE 11–1 **Common Mechanisms of Injury by Energy Source**

| ENERGY SOURCE | COMMON MECHANISMS OF INJURY |
|---|---|
| Mechanical | Motor vehicles<br>Firearms<br>Machines |
| Gravitational | Falls |
| Thermal | Heating appliances<br>Fire<br>Freezing temperatures |
| Electrical | Wires, sockets, and other electrical objects<br>Lightning |
| Physical | Fists, feet, and other body parts (as in physical assault)<br>Sharp objects, such as knives<br>Ultraviolet radiation<br>Ionizing radiation<br>Water (drowning)<br>Other submersion agents (e.g., grain)<br>Explosions |
| Chemical | Drugs<br>Poisons<br>Industrial chemicals |

Guns are another common mechanical source of injury. Trauma from gunshot wounds has steadily increased during the past 20 years and remains a major reason for emergency department and trauma center admissions, especially in large cities.

When describing a traumatic injury, *intention* is included as a component. Most gunshot and stab wounds are examples of intentional injuries. It is important to remember, however, that some gunshot wounds are unintentional, such as those that occur when children play with their parents' guns. Other common unintentional injuries result from motor vehicle crashes, falls, drowning, and fires. Although hunting accidents are rare in comparison to the number of people participating in the sport, hunting-related deaths and injuries have decreased with the implementation of mandatory "hunting safety" courses in some states.

The final component of trauma is the *environment*. For example, a road that has become slippery after a snowstorm is a

physical environment that may contribute to an injury. Occupation is an important environmental factor to consider. Those in certain occupations face a high risk of trauma; examples include police officers, firefighters, professional athletes, race car drivers, and taxi cab drivers. One's social environment also influences risk for injury; see the Meeting Individualized Needs box below for one example, domestic violence.

## Types of Trauma

**Minor trauma** causes injury to a single part or system of the body and is usually treated in a physician's office or in the hospital emergency department. A fracture of the clavicle, a small second-degree burn, and a laceration requiring sutures are examples of minor trauma. Major or **multiple trauma** involves serious single-system injury (such as the traumatic amputation of a leg) or multiple-system injuries. Multiple trauma is most often the result of a motor vehicle crash.

Trauma is further classified as either blunt or penetrating. **Blunt trauma** occurs when there is no communication between the damaged tissues and the outside environment. It is caused by various forces including *deceleration* (a decrease in the speed of a moving object), *acceleration* (an increase in the speed of a moving object), *shearing* (forces occurring across a plane, with structures slipping across each other), *compression*, and *crushing*. Blunt forces often cause multiple injuries that can affect the head, spinal cord, bones, thorax, and abdomen. Blunt trauma is frequently caused by motor vehicle crashes, falls, assaults, and sports activities.

**Penetrating trauma** occurs when a foreign object enters the body, causing damage to body structures. Structures commonly affected include the brain, lungs, heart, liver, spleen, the intestines, and the vascular system. Examples of penetrating trauma are gunshot or stab wounds and impalement.

Other types of trauma include inhalation injuries from gases, smoke, or steam; burn or freezing injuries; and blast injuries from explosions. Blast injuries result from the temperature and velocity of air movement and the force of projectiles from the explosion. Blast injuries are more severe in water than in air because blast waves travel farther and faster in water. Trauma from blast injuries includes pulmonary edema and hemorrhage, damage to abdominal organs, burns, penetrating injuries, and ruptured tympanic membranes.

Outcome studies show a correlation between survival rates of multiple trauma victims and rapid response times by prehospital providers coupled with appropriate decision making with regard to transporting the victim to a facility capable of treating their injuries (Han et al., 2003). As a result, a system was devised to assist prehospital providers to make the appropriate decisions. Trauma clients are classified as class 1, 2, or 3 based on factors including mechanism of injury, vehicle speed, height of falls, and location of penetrating injuries. Class 3 trauma is the least severe. An example would be a same-level fall without loss of consciousness or significant injury. Class 1 trauma involves life-threatening injuries likely to require medical specialists or immediate surgical intervention. While any hospital emergency department should be capable of caring for class 3 trauma clients, clients meeting class 1 or 2 criteria should be transported to a designated trauma center when possible. Facilities designated as trauma centers have medical specialists and surgical coverage available or on call 24 hours a day.

### MEETING INDIVIDUALIZED NEEDS Assessing Elder Abuse and Intimate Partner Violence (IPV)

#### IPV

Most IPV incidents are not reported, thus it is believed that the available data greatly underestimate the true magnitude of the problem. It is estimated that more than 5.3 million women are beaten by male partners every year, resulting in 1300 deaths annually. IPV is the single largest cause of injury to women in the United States. Among men, 3.2 million IPV episodes occur annually, accounting for approximately 800,000 men raped or physically assaulted by an intimate partner.

IPV is a widespread problem that occurs regardless of age, sex, race, socioeconomic status, or education. IPV is also referred to as partner abuse or spousal abuse (CDC, 2003). In 2001, intimate partner violence made up 20% of violent crime against women. The same year, intimate partners committed 3% of all violent crimes against men.

#### VIOLENCE AND THE ELDERLY

Elder abuse is defined as anything that endangers the life of an elderly person. This can range from physical or emotional assault to intimidation, neglect, or financial exploitation. Willful deprivation of food or medical care is also included. The National Elder Abuse Incidence study found that approximately 551,000 persons aged 60 or older were abused or neglected in a 1-year period. Persons 80 years of age and older experienced abuse and neglect at two to three times their proportion in the older population. The perpetrator is a family member in 90% of the cases.

The general approach to diagnosis in abuse situations is challenging and many times hidden. As with spousal, elder, or child abuse, the task of identification is complex. The following are clues to identify violence-related injuries:

- Injuries that do not correlate with the history
- Injuries that suggest a defensive posture
- Injuries during pregnancy
- Pattern injuries
- Pattern burns
- Sexual abuse/rape
- Unusual or unexplained fractures
- Signs of confinement
- Unusual interaction between client and caregiver
- Lack of medical attention; immunizations not up to date; poor dental health
- Unexplained dehydration or malnutrition

*Source:* From Bureau of Justice Statistics Crime Data, *Intimate Partner Violence, 1993–2001*, February 2003; Retrieved March 30, 2006, from http://www.aoa.gov/eldfam/Elder_Rights/Elder_Abuse/Elder_Abuse_pf.asp

## Effects of Traumatic Injury

Death is a common result of serious traumatic injury, and may be immediate, early, or late. Immediate death happens at the scene from such injuries as a torn thoracic aorta or decapitation. Early death occurs within several hours of the injury from, for example, shock or delay in recognizing injuries. Late death generally occurs 1 or more days after the injury and results from multiple organ failure, sepsis, and coagulopathies.

Because of the serious consequences of trauma, it is important to rapidly identify the client's injuries and institute appropriate interventions quickly. Following are common results of trauma and interventions necessary for good outcomes.

### Airway Obstruction

Assessment of the airway is the highest priority in the trauma client. Other distracting injuries may take the inexperienced practitioner away from the airway, but if the airway is not patent and the client is unable to deliver oxygen to vital organs, all other interventions are futile.

Assessment includes determining airway patency. If the client is unresponsive, manual opening of the airway using a jaw-thrust or chin-lift maneuver is necessary. Once the airway is opened, the practitioner must identify any potential obstruction from the tongue, loose teeth, foreign bodies, bleeding, secretions, vomitus, or edema. If the client is responsive and can vocalize, that is a good indication that the airway is clear.

Any time the nurse performs an intervention it is important to reassess the effectiveness of the intervention. For example, if the nurse suctions the airway to remove vomitus, he or she would reassess the airway after suctioning to determine if that intervention was successful or if the airway needs to be suctioned a second time.

All trauma clients should receive high-flow oxygen until stabilized. Assessment of breathing effectiveness is paramount. Assessment should include whether the client has spontaneous breathing, good rise and fall of the chest, determination of skin color, general rate and depth of respirations, use of abdominal or accessory muscles, position of the trachea, observation of chest wall integrity and presence of jugular vein distention, bilateral breath sounds, and the presence of any surface trauma.

In addition to suctioning, other available airway adjuncts include oral or nasal pharyngeal airways, oxygen delivery devices, laryngeal mask airways, Combitubes, and endotracheal intubation (Figure 11–1 ■). Intubation is the preferred method of airway management if the client is unable to maintain oxygenation or an open airway.

Trauma clients may exhibit several aspects of airway management that are unique and require special preparation and precautions, as discussed below.

***CLOSED HEAD INJURY*** Changes in hemodynamics, oxygenation, and ventilation should be minimized in order to maintain adequate cerebral perfusion pressure. Laryngoscopy causes a marked increase in intracranial pressure (ICP).

The goal is to maintain a $Paco_2$ of 30 to 35 mmHg. Lidocaine administered 3 to 5 minutes prior to intubation can blunt an increase in ICP that is secondary to laryngeal stimulation. In a normotensive client, beta-blockers are given 2 to 3 minutes prior to intubation to attenuate the sympathetic response. Effective induction agents such as Etomidate or thiopental have not been shown to increase ICP.

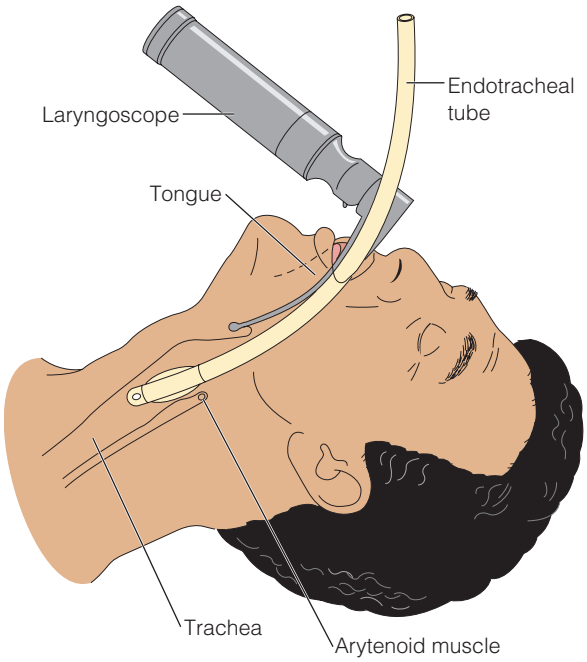



**Figure 11–1** ■ Placement of an oral endotracheal tube (ETT) for intubation. When the ETT is in place, air or oxygen can be blown into the external opening of the tube and enter the trachea.

***MAXILLOFACIAL TRAUMA*** Significant distortion of normal anatomy occurs in facial trauma, and respiratory compromise is not uncommon. Even in clients who present with mild respiratory compromise, rapid deterioration from edema or hemorrhage can occur. A surgical airway may be the only alternative.

***DIRECT AIRWAY TRAUMA*** Penetrating trauma to the neck is associated with a high degree of morbidity and mortality. Airway involvement includes dyspnea, cyanosis, subcutaneous emphysema, hoarseness, or air bubbling from the wound. Orotracheal intubation with rapid sequence intubation is the technique of choice. The key is early identification of the need for intubation before the client has no airway at all. Tracheobronchial injury occurs in approximately 10% to 20% of clients with penetrating neck injuries.

***CERVICAL SPINE INJURY*** Securing an airway in the presence of a presumed C-spine injury is recommended. Approximately 3% to 6% of major trauma victims have clinically significant C-spine injuries. Oral intubation with manual in-line axial head and neck stabilization is a safe method. The probability of C-spine injury is decreased if the following criteria are met:

- Absence of midline cervical spine tenderness
- Normal alertness
- Absence of intoxication
- Absence of a painful distracting injury
- No focal neurologic defects.

***BURNS*** Burn clients with airway compromise require aggressive management. Upper airway edema associated with inhalation or enclosed-space fires can progress during the postburn phase. Securing an airway sooner rather than later is the goal. See Chapter 17 ∞ for nursing care of the client with burns.

## Tension Pneumothorax

A **pneumothorax** results when air enters the potential space between the parietal and visceral pleura. The thorax is completely filled by the lungs. Surface tension between the pleural surfaces holds the lungs to the chest wall. Air present in the pleural space will eventually collapse the lungs. A **tension pneumothorax** is a special type of pneumothorax that is life threatening and requires immediate intervention. On inspiration, air enters the pleural space, does not escape on expiration, and increases the intrapleural pressure. This pressure collapses the injured lung and shifts the mediastinal contents, compressing the heart, great vessels, trachea, and eventually the uninjured lung. In turn, this causes the following signs and symptoms:

- Severe respiratory distress
- Hypotension
- Jugular vein distention
- Tracheal deviation toward the uninjured side
- Cyanosis.

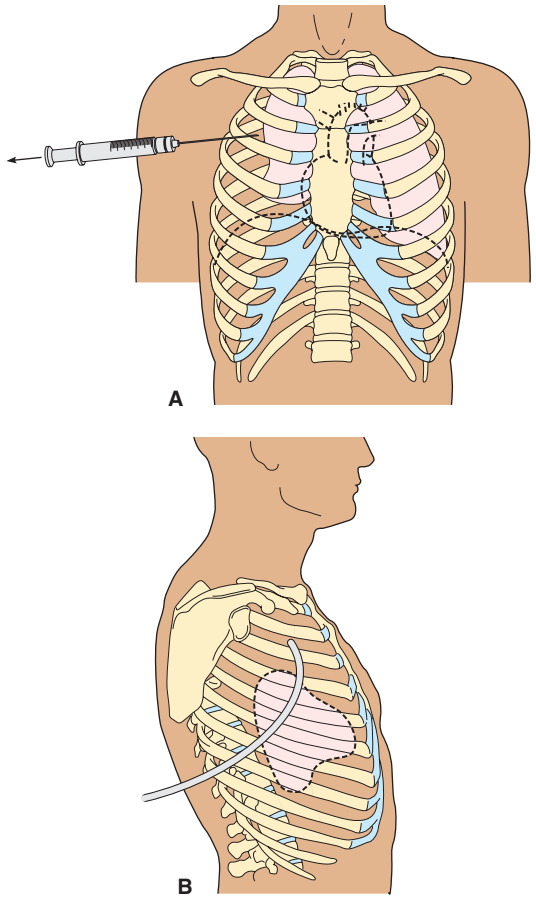



**Figure 11–2** ■ A needle thoracostomy may be used in the emergency treatment of a tension pneumothorax. *A*, A large gauge needle is introduced, and air and fluid are aspirated. *B*, Alternatively, a chest tube may be inserted and connected to a chest drainage system.

The immediate short-term lifesaving intervention is a needle thoracostomy, in which a large-bore needle is inserted into the second intercostal space at the midclavicular line. See Figure 11–2 ■.

## Hemorrhage

When the client has suffered an injury that causes external hemorrhage, such as severing of an artery, the bleeding must be controlled immediately. This may be done by applying direct pressure over the wound and applying pressure over arterial pressure points (Figure 11–3 ■).

Internal hemorrhage may result from either blunt or penetrating traumatic injury. Discovering the cause and location of the injury, as well as the extent of related blood loss, are the most important concerns. Several potential spaces in the body can accommodate large amounts of blood that may accumulate (called *third spacing*) following injury. For example, bleeding into the pleural space may occur with chest trauma, and bleeding into the abdominal cavity may occur with abdominal trauma. A pelvic fracture may cause massive hemorrhaging into the retroperitoneal region. Once the source of internal hemorrhage has been recognized, interventions are initiated, including operative control of bleeding and continual assessment of the client. Hemorrhage may result in hypovolemic shock (discussed later in the chapter).

## Integumentary Effects

Injuries to the integument generally are not as serious as other injuries, with the exception of burns (see Chapter 17 ∞). The primary organ involved in integumentary trauma is the skin;

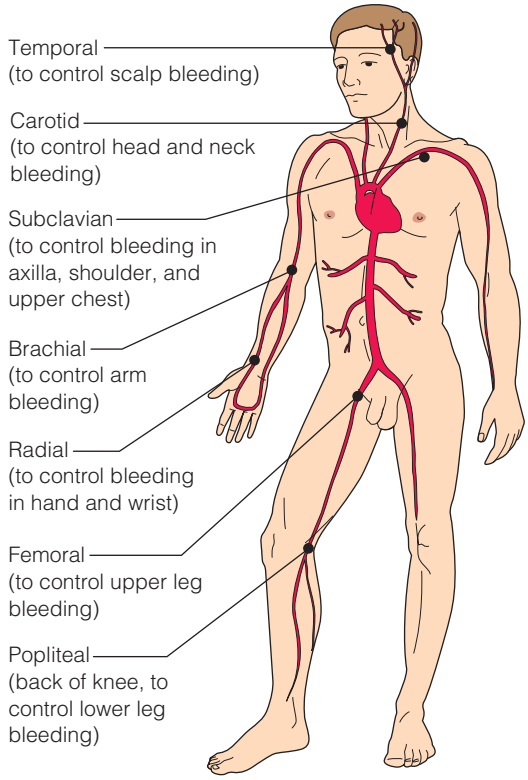



**Figure 11–3** ■ The major pressure points used for the control of bleeding.

however, underlying structures may also be injured. Injuries may result from either blunt or penetrating sources. It is important to evaluate all injuries to the integument, because they may indicate a more serious injury such as an open fracture. Additionally, large wounds may contribute to significant blood loss.

Five specific injuries to the integument are contusions, abrasions, puncture wounds, lacerations, and full-thickness avulsion injuries (Figure 11–4 ■). **Contusions**, or superficial tissue injuries, result from blunt trauma that causes the breakage of small blood vessels and bleeding into the surrounding tissue. **Abrasions**, or partial-thickness denudations of an area of integument, generally result from falls or scrapes. **Puncture wounds** occur when a sharp or blunt object penetrates the integument. **Lacerations** are open wounds that result from sharp cutting or tearing. Injuries to the integument are at risk for contamination from dirt, debris, or foreign objects. Infection may cause further physical stress to the client with multiple injuries. **Full-thickness avulsion injuries** are injuries that result in loss of all of the layers of the skin, causing fat and muscle to be exposed. The size of the wound impacts both the length of time necessary for healing to take place as well as the risk for infection. These types of injuries are treated by suturing the wound together, reattaching avulsed skin, or by skingrafting.

## Abdominal Effects

The abdomen contains both solid organs (liver, spleen, and pancreas) and hollow organs (stomach and intestines). Direct trauma to the abdomen can lacerate and compress the solid organs and cause burst injuries to the hollow organs. Blood vessels may be torn and organs may be displaced from their blood supply, producing life-threatening hemorrhage. Damage to the mesenteric vessels supplying the bowel can result in bowel ischemia and infarction. Injury to the stomach, pancreas, and small bowel may allow digestive enzymes to leak into the abdominal cavity. Rupture of the large bowel results in escape of feces, which causes peritonitis. Blunt or penetrating trauma to the abdomen may also cause rupture of the diaphragm with herniation of the abdominal organs into the thoracic cavity. The immediate threat following abdominal trauma is hemorrhage; the later threat is peritonitis.

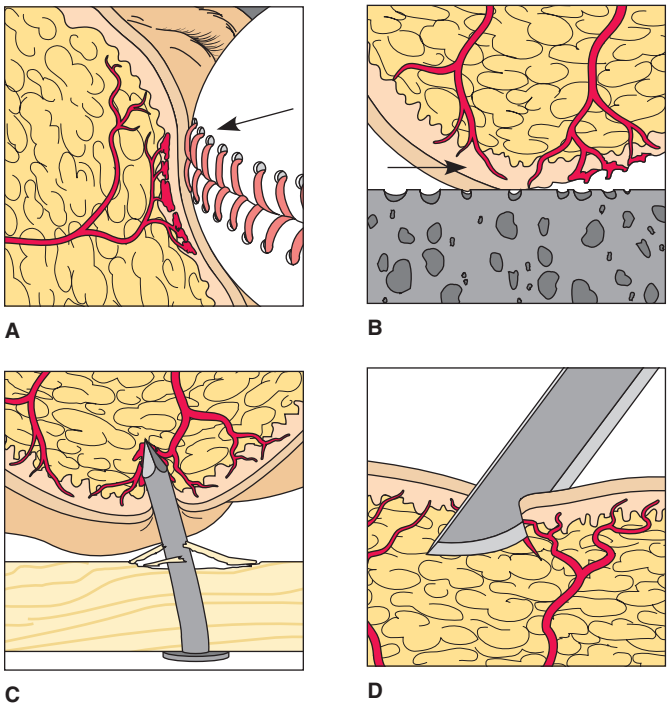



**Figure 11–4** ■ Traumatic injuries to the skin include *A,* contusion; *B,* abrasion; *C,* puncture wound; *D,* laceration.

## Musculoskeletal Effects

Musculoskeletal injuries may occur alone or with multiple injuries as the result of blunt or penetrating trauma. Musculoskeletal injuries usually are not considered a high priority in the care of the client with multiple injuries. Exceptions are the life- or limb-threatening musculoskeletal injury, such as a dislocated hip, pulseless extremity, or significant blood loss such as from a femur or pelvic fracture. Musculoskeletal injuries may provide clues to the presence of other serious injuries; for example, a fractured clavicle may indicate an associated thoracic injury. Care of the client who has suffered a musculoskeletal injury is discussed in Chapter 41 ∞.

## Neurologic Effects

Head injuries are a common type of injury sustained as the result of trauma. Injuries to the spinal cord resulting in loss of neurologic function are devastating outcomes of trauma, but they are much less common than head injuries. Most head and spinal cord injuries result from blunt trauma and are sustained in motor vehicle crashes. Falls, sports injuries, and assault are other sources of neurologic injury. Care of the client with a neurologic injury is discussed in Chapters 44, 45, and 46 ∞.

## Multiple Organ Dysfunction Syndrome

Multiple organ dysfunction syndrome (MODS) is a common complication of severe injury and a frequent cause of death in intensive care units. MODS is a progressive impairment of two or more organ systems. This is the result of an uncontrolled inflammatory response to severe injury or illness.

Clients at risk for MODS are those with a disturbance in homeostasis resulting from one or a combination of the following conditions:

- Infection
- Injury
- Inflammation
- Ischemia
- Immune response
- Intoxication of substances
- Iatrogenic factors.

The primary organ systems involved in MODS are the respiratory, renal, hepatic, hematologic, cardiovascular, gastrointestinal, and neurologic systems. Supportive therapy depends on the identification of correctable causes. It may be one or a combination of several therapies. Surgical intervention, antibiotic administration, corticosteroid administration, and correction coagulopathies are some of the therapies used for this condition. MODS following injuries produce more than half of the late mortality following trauma.

### Effects on the Family

Trauma usually occurs suddenly and with little warning. It may result in death or cause injury serious enough to alter both the client's and the family's lives. The suddenness and seriousness of the event are precipitating factors in the development of a psychologic crisis. During the past decade, some emergency departments have instituted care plans that allow families to be present during resuscitation. This type of care is not without controversy, but it should be considered when appropriate.

## INTERDISCIPLINARY CARE

Interdisciplinary care of the trauma client depends on a team approach. Providing trauma care with a team focus helps each team member know his or her role. Prompt delegation of tasks and responsibilities improves the client's chances for survival and decreases the morbidity that may result from traumatic injuries.

### Prehospital Care

The major functions of prehospital care include injury identification, critical interventions, and rapid transport.

***INJURY IDENTIFICATION*** Emergency care of the client experiencing trauma is based on rapid assessment to identify injuries and begin appropriate interventions. Injuries that indicate the need for trauma center care include:

- Penetrating injuries to the abdomen, pelvis, chest, neck, or head
- Spinal cord injuries with deficit
- Crushing injuries to the abdomen, chest, or head
- Major burns
- Injuries leading to airway compromise or obstruction.

Many methods help healthcare providers determine the seriousness of the client's injuries and the potential for survival. Scoring systems such as the Champion Revised Trauma Scoring System can be helpful (Table 11–2). A rapid but comprehensive trauma assessment, completed on the scene, includes:

- *Airway and breathing assessments* to determine if the airway is patent, maintainable, or nonmaintainable, and if ventilations are impeded, such as by rib fractures or a collapsed lung
- *Circulation assessment* to palpate peripheral and central pulses; to assess capillary refill, skin color, and temperature; and to identify any external sources of bleeding
- *Level of consciousness* and *pupillary function assessment*
- Assessment for any obvious injuries.

The Glasgow Coma Scale is another scoring system that is used to quantify the level of consciousness following traumatic brain injury. See Chapter 43 ∞.

***CRITICAL INTERVENTIONS*** As life-threatening problems are identified during the primary assessment, appropriate on-the-scene interventions must be performed immediately. These include providing life support, immobilizing the cervical spine, managing the airway, and treating hemorrhage and shock.

Immobilization of the client's cervical spine is a primary intervention. The client is placed on a spine board, and a cervical collar and a head immobilizer are applied (Figure 11–5 ■). The cervical spine may also be immobilized by logrolling the client onto a board, placing towel rolls or a head immobilizer along the sides of the client's head, and securing the client to the board. If the client was wearing a helmet at the time of injury, the helmet should remain on until the client arrives at the hospital, unless the client's airway is at risk. If necessary, healthcare personnel at the scene will remove the helmet by

**TABLE 11–2 Champion Revised Trauma Scoring System**

| TEST | SCORE | CODED VALUE |
|---|---|---|
| Glasgow Coma Scale* | 13 to 15 | 4 |
| | 9 to 12 | 3 |
| | 6 to 8 | 2 |
| | 4 to 5 | 1 |
| | 3 | 0 |
| Systolic blood pressure | > 89 | 4 |
| | 76 to 89 | 3 |
| | 50 to 75 | 2 |
| | 1 to 49 | 1 |
| | 0 | 0 |
| Respiratory rate | 10 to 29 | 4 |
| | > 29 | 3 |
| | 6 to 9 | 2 |
| | 1 to 5 | 1 |
| | 0 | 0 |
| | Total score: | _____ |

The highest possible total score is 12. The lowest possible score is 0. The higher the total score, the greater the chance of survival.

**See Chapter 43 for instructions for using the Glasgow Coma Scale.*
*Source: From "A Revision of the Trauma Score" by H. Champion et al., 1989,* Journal of Trauma, *29(5), 624. Used with permission.*

**Figure 11–5** ■ Immobilization of the cervical spine at the scene of the accident is essential to prevent further injury to the spinal cord. The combined use of a hard cervical collar, head blocks, and tape best restricts flexion, extension, rotation, and lateral bending of the neck.

*Source:* Spencer Grant/Photo Researchers, Inc.

manipulating it over the client's nose and ears while holding the client's head and neck immobile; safe removal requires at least two people. Improper removal risks injury or additional injury to the spinal cord.

If the client's airway is patent, oxygen is administered. Ventilations may be assisted with a bag-valve-mask resuscitator until airway management is achieved. Active external bleeding is controlled by direct pressure. Measures to reverse shock (discussed later in the chapter) are initiated.

***RAPID TRANSPORT*** Clients who have multiple injuries must be transported as soon as possible to a regional trauma center. The most common modes of rapid transport are ground ambulance and air ambulance, which includes helicopters specially staffed and equipped to care for trauma victims. Figure 11–6 ■ shows a flight nurse assessing a client. Stable clients within access of a ground ambulance are best transported by ground. Unstable clients and those injured in the wilderness or other areas in which ground access is difficult may best be transported by air. When these transport systems are unavailable, the client is transported by any possible means. Morbidity and mortality rates are reduced when the client is transported to a facility that can manage their injuries within an hour of the injury. In trauma, this time is referred to as the "Golden Hour."

**Figure 11–6** ■ Flight nurses provide initial assessment, stabilization, and support for clients with trauma.

Courtesy of University of Air Care/University of Cincinnati Hospital.

## Emergency Department Care

***DIAGNOSIS*** The diagnostic tests ordered once the client reaches the hospital depend on the type of injury the client has sustained. Tests that may be ordered for victims of trauma include the following:

- *Blood type and crossmatch* involves typing the client's blood for ABO antigens and Rh factor, screening the blood for antibodies, and crossmatching the client's serum and donor red blood cells.
- *Blood alcohol level* measures the amount of alcohol in a client's blood. Studies have found that between 20% and 50% of people who are injured may be intoxicated. Alcohol alters the client's level of consciousness and response to pain (NHTSA, 2006).
- *Urine drug screen* may also be ordered. Like alcohol, such drugs as cocaine alter the client's level of consciousness and overall response to the primary survey.
- *Pregnancy test* for any woman of childbearing age rules out the potential for pregnancy and fetal injury.
- The primary goal of the *focused assessment by sonography in trauma (FAST)* exam is to evaluate the presence of blood in body cavities where it is not supposed to be. Primary focus is on the peritoneum. It is also helpful in identification of blood in the pleura and pericardium.
- *Diagnostic peritoneal lavage* determines the presence of blood in the peritoneal cavity, which may indicate abdominal injury. This test is generally done in the emergency department. A local anesthetic (such as lidocaine) is injected subcutaneously, and a small incision is made in the lower abdomen. A catheter is placed into the peritoneal cavity, and any free blood is aspirated. If 10 mL of blood is found, the client is taken to the operating room for exploratory surgery. If no free blood is aspirated, 1 L of a warm isotonic solution (Ringer's solution or normal saline) is rapidly infused into the peritoneal cavity and then allowed to drain by gravity. If the solution returns pink and is found to have a red blood cell count of 100,000 $mm^3$, a white blood cell count of >500, or bile, food, or feces, the test is considered positive and the client is taken to the operating room for exploratory surgery. This procedure has been used less since the inception of the FAST exam.
- *Computerized tomography (CT) scans* can discover injuries to the brain, skull, spine, spinal cord, chest, and abdomen.
- *Magnetic resonance imaging (MRI) scans* can discover injuries to the brain and spinal cord.

***MEDICATIONS*** Medications used to treat the client who has experienced trauma depend on the type and severity of the injuries, as well as the degree of traumatic shock that is present. The following general categories of medications may be used. (Fluid

administration and the drugs listed are covered later in the chapter in discussion of the collaborative care of the client in shock.)

- Blood components and crystalloids are administered intravenously in the initial treatment of traumatic shock to replace intravascular volume.
- Inotropic drugs (drugs that increase myocardial contractility) are given to increase cardiac output and improve tissue perfusion. These drugs, administered only after fluid volume restoration, include dopamine (Dopastat, Intropin), dobutamine (Dobutrex), and isoproterenol (Isuprel).
- Vasopressors may be administered in conjunction with fluid replacement to treat neurogenic, septic, or anaphylactic shock. Examples of vasopressors include dopamine, epinephrine, norepinephrine, and phenylephrine.
- Opioids, administered by bolus or continuous infusion, are used to treat pain as soon as possible. However, the effects of the pain medications may alter client responses to injury and mask potential injuries. If pain medications are administered, they must be carefully regulated, and the client must be closely monitored.
- If the client has penetrating and open wounds, tetanus immunization status must be determined. If the client is unable to remember when the last tetanus immunization was given or is unable to answer, tetanus prophylaxis is given.

***BLOOD TRANSFUSIONS*** Blood and blood components are initially produced in the body and then donated for use by another person through a **transfusion** (an infusion of blood or blood components). A client may be given whole blood, packed red blood cells (RBCs), platelets, plasma, albumin, clotting factors, prothrombin, or cryoprecipitate (Table 11–3). Blood and blood components increase the amount of hemoglobin available to carry oxygen to the cells, improve hemoglobin and hematocrit levels during active bleeding, increase intravascular volume, and replace deficient substances such as platelets and clotting factors.

Each person has one of four blood types: A, B, AB, or O. The blood group antigens A and B, present on RBC membranes, form the basis for the ABO blood categorization. The presence or absence of these inherited antigens determines one's blood type. People with blood type A have A antigens, those with type B have B antigens, those with type AB have both antigens, and those with neither antigen have blood type O (called a universal donor).

## FAST FACTS

- Type AB blood is the "universal recipient."
- Type O blood is the "universal donor."

ABO antibodies develop in the serum of people whose RBCs lack the corresponding antigen; these antibodies are called anti-A and anti-B. The person with blood type B has A antibodies, the person with type A has B antibodies, the person with type O has both types of antibodies, and the person with blood type AB has no antibodies (called a universal recipient).

A third antigen on the RBC membrane is D. People who are Rh positive have the D antigen, whereas people who are Rh negative do not. These antigens and antibodies may cause ABO and Rh incompatibilities.

A transfusion of incompatible blood causes hemolysis (breakdown) of the RBCs and agglutination of erythrocytes. (*Agglutination* is the clumping of cells that results from their interaction with specific antibodies.) The ABO blood group names and compatibilities are listed in Table 11–4.

Before RBCs or whole blood can be administered, a series of procedures determine donor and recipient ABO types and Rh groups. These procedures, called a *type and crossmatch*, are performed by mixing the donor cells with the recipient's serum and watching for agglutination. If none occurs, the blood is considered compatible.

Despite meticulous procedures for matching blood types and antigens, blood transfusion reactions may still occur. The most common is a *febrile reaction.* Antibodies within the client receiving the blood are directed against the donor's white blood cells, causing fever and chills. Febrile reactions typically begin during the first 15 minutes of the transfusion. Using leukocyte-poor blood avoids future febrile reactions.

*Hypersensitivity reactions* result when antibodies in the client's blood react against proteins, such as immunoglobulin A, in the donor blood. Hypersensitivity reactions may appear during or after the transfusion. The manifestations of hypersensitivity reaction include *urticaria* (the appearance of reddened wheals of various sizes on the skin) and itching.

*Hemolytic reactions,* the most dangerous transfusion reactions, usually result from an ABO incompatibility. Clumping RBCs block capillaries, decreasing blood flow to vital organs. In addition, macrophages engulf the clumped RBCs, releasing free hemoglobin into the circulating blood; the hemoglobin is then filtered by the kidneys and may block the renal tubules, causing renal failure. Hemolytic reactions usually begin after infusion of 100 to 200 mL of the incompatible blood. Manifestations of a hemolytic reaction include flushing of the face, a burning sensation along the vein, headache, urticaria, chills, fever, lumbar pain, abdominal pain, chest pain, nausea and vomiting, tachycardia, hypotension, and dyspnea. If any of these manifestations appear, the blood transfusion must be immediately discontinued.

Other risks to clients receiving blood include circulatory overload, electrolyte imbalances, and infectious diseases such as hepatitis or cytomegalovirus.

Clients who have experienced trauma of any severity have had substantial blood loss and are usually in hypovolemic shock. Blood replacement is the treatment of choice to restore oxygen-carrying capacity. Clients in severe shock with active bleeding are given universal, type O red blood cells immediately. Clients with less severe injuries or bleeding may be stabilized with other types of fluids until type-specific or crossmatched blood is available.

Some emergency departments and trauma centers use autotransfusion to provide blood for transfusions for the client with multiple injuries and/or severe shock. Autotransfusion is a method of blood administration in which special equipment collects and returns the client's own blood. The chest cavity is the typical source of blood to be autotransfused.

TABLE 11–3 **Volume Resuscitation Therapies**

| COMPONENT | INDICATIONS | ADVANTAGES | DISADVANTAGES |
|---|---|---|---|
| Ringer's lactate | Restoration of circulating volume<br>Replacement of electrolyte deficits | Good availability<br>Safe to use<br>Low cost<br>Aids in buffering acidosis | Rapid movement from the intravascular to the extravascular space, leading to three or more times requirement for replacement |
| Normal saline | Restoration of circulating volume<br>Vehicle compatible with administration of blood | Good availability<br>Low cost<br>Safe to use | Hyperchloremic acidosis associated with prolonged use of sodium solutions |
| Whole blood | Replaces blood volume and oxygen-carrying capacity in hemorrhage and shock | Contains RBCs, plasma proteins, clotting factors, and plasma | Contains few platelets or granulocytes; deficient in clotting factors V and VII<br>Greatest risks are for incompatibility or circulatory overload |
| Packed RBCs | Restoration of intravascular volume<br>Replacement of oxygen-carrying capacity | One unit of RBCs should increase the hemoglobin of a 70-kg adult by approximately 1 g/dL in the absence of volume overload or continuing blood loss | Red cells require compatibility testing<br>Risk of transmitting bloodborne pathogens<br>Should be warmed to prevent hypothermia |
| Platelets | Significant thrombocytopenia (platelet count less than 20,000–50,000/$mm^3$)<br>Continued hemorrhage | Compatibility testing is not required<br>Typical platelet transfusion should raise the platelets of a 70-kg adult approximately 30,000–50,000/UL | Postexposure prophylaxis with anti-Rh immune globulin should be considered following $Rh^+$ platelet transfusion to an $Rh^-$ woman |
| Albumin | Expends blood volume in shock and trauma | Good availability | Is not a substitute for whole blood<br>Hypersensitivity reactions can occur |
| Fresh frozen plasma (FFP) | Documented coagulopathy<br>Restoration of clotting factors<br>Supplies plasma proteins | Crossmatching and Rh compatibility not required | Must be thawed in a 37°C water bath for approximately 30 minutes<br>Should be ABO compatible |
| Cryoprecipitate | Coagulopathy with low fibrinogen<br>Restoration of fibrinogen | Rh type not important | Risk of transmitting bloodborne pathogens<br>Contains hemagglutinins<br>If large volume of ABO incompatible cryoprecipitate are administered, intravascular hemolysis can occur |

Nursing considerations for blood transfusion therapy are described in the Medication Administration box on the following page.

## Emergency Surgery

Immediate surgical intervention is indicated when the client remains in shock despite resuscitation and there is no obvious external sign of blood loss. Abdominal and chest x-ray, ultrasound studies, diagnostic peritoneal lavage, or CT scan may be performed to help identify the potential source of the blood loss. It is important for the emergency or trauma nurse to speak with the family as soon as possible and keep them informed about what is happening to their family member. Unfortunately, the need for emergency surgery may not allow time for family members or significant others to see their loved one before transfer to the operating room.

## Organ Donation

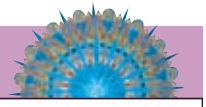
The Uniform Anatomical Gift Act (1968, 1987) requires that people be informed about their options for organ donation.

TABLE 11–4 **Blood Group Types and Compatibilities**

| BLOOD GROUP | RBC AGGLUTINOGENS | SERUM AGGLUTINOGENS | COMPATIBLE DONOR BLOOD GROUPS | INCOMPATIBLE DONOR BLOOD GROUPS |
|---|---|---|---|---|
| A | A | Anti-B | A, O | B, AB |
| B | B | Anti-A | B, O | A, AB |
| AB | A, B | None | A, B, AB, O | None |
| O | None | Anti-A, anti-B | O | A, B, AB |

*Note:* Group O is often called the universal donor, and group AB is called the universal recipient.

## MEDICATION ADMINISTRATION Blood Transfusion



The risk for and seriousness of blood transfusion reactions require that extreme caution be taken when blood is administered. Most fatal transfusion reactions are the result of human error. Although general guidelines are provided here, each institution has specific policies and procedures that must be followed. Prior to beginning the transfusion, the nurse must determine that typed and crossmatched blood is available and collect the needed equipment: a Y-tubing blood administration set with a filter, a large-bore intravenous catheter (usually 18 or 19 gauge), and normal saline solution. Only normal saline is used with a blood transfusion. Dextrose causes clumping of RBCs, and distilled water causes hemolysis.

### Nursing Responsibilities

- Obtain client consent.
- Assess for any previous reactions to blood.
- Explain the procedure to the client, and answer any questions.
- Prepare the intravenous equipment. Shut off one side of the Y tubing, and attach the other side to the saline solution. Flush the tubing and filter with the saline.
- If venous access is not already in place, insert the intravenous needle (following body substance precautions), and begin administering the saline.
- Using institutional procedure, obtain the blood from the blood bank or laboratory. Administer the blood immediately; if this is not possible, return it to the blood bank or laboratory.
- Check and document that the donor and recipient blood have been tested and are compatible. This usually involves two nurses, each verifying that:
  a. An order for blood has been written.
  b. Type and crossmatch have been done.
  c. The name of the client and the name on the blood bag are identical.
  d. The number assigned to the unit of blood is identical to the one on the requisition for the blood.
  e. Blood type and Rh factor are compatible.
  f. The blood has not exceeded its expiration date.
  g. The unit of blood is intact and has no bubbles or discoloration.
- Identify the client by reading the armband and, if conscious, asking the client to tell you his or her name. Check the armband against the unit of blood.
- Gently invert the blood bag several times to mix the plasma and RBCs.
- Take and record vital signs as a baseline.
- Attach the open side of the Y tubing to the blood unit, and begin the transfusion at a slow rate of about 2 mL/min. (Some trauma clients may have blood infused at a rapid rate. If blood is infused rapidly, it may need to be warmed prior to administration to prevent hypothermia.) Stay with the client for at least the first 15 minutes of the transfusion, monitoring for manifestations of a reaction and taking the client's vital signs.
- Continue to monitor the client during the transfusion, assessing for manifestations of hypersensitivity or hemolytic reactions and taking and recording vital signs as directed by institutional policy.
- After the first 15 minutes, the rate of infusion is increased. If there is no danger of fluid volume overload, most clients can tolerate an infusion of a unit of blood (ranging from 250 to 500 mL, depending on the blood component administered) in 2 hours. The unit of blood must be administered within 3 to 4 hours; after this time, it has warmed and begins to deteriorate.
- Take the following actions if manifestations of a reaction occur:
  a. Stop the infusion of blood immediately, and notify the physician. Continue to infuse the saline.
  b. Take vital signs and assess manifestations.
  c. Compare the blood slip with the unit of blood to ensure that an identification error was not made.
  d. Save the blood bag and any remaining blood for return to the laboratory for further tests to determine the cause of the reaction.
  e. Follow institutional policy for collecting urine and venous blood samples.
  f. Continue to monitor the client and provide prescribed interventions to treat hypersensitivity or hemolytic manifestations.

### Health Education for the Client and Family

- The possible risks of blood transfusions include transmission of infectious diseases and acquired immune deficiency syndrome (AIDS). However, because of careful handling and storage of blood, bacterial contamination is rare. Although hepatitis may be transmitted by contaminated blood, new tests for hepatitis antibodies in donor blood are reducing this risk. Many people are afraid of contracting AIDS from blood; however, donor screening and HIV-antibody testing of donor blood has virtually eliminated the transmission of HIV by blood transfusion. A new risk that has been identified is the transmission of West Nile Virus through blood transfusions. Screening for this risk is asking potential donors about the presence of symptoms indicative of West Nile Virus.
- During the transfusion, immediately report any warm feelings, chills, itching, feelings of weakness or fainting, or difficulty breathing.
- Report any signs of a delayed transfusion reaction: chills, fever, cough, difficulty breathing, hives, itching, or changes in circulation, and seek medical care immediately.

Under this act, consent for organ donation may be given not only by the donor but also by a spouse, adult children, parents, adult siblings, guardian, or any adult authorized to do so. The act also encourages people to carry donor cards.

The increased success of organ transplant has made it a more common and valuable method of prolonging and improving life; however, many people are still waiting for organs, and many people who may be suitable organ donors die each year from trauma. Organs and tissues that may be transplanted include bones, eyes, liver, lungs, skin, muscles and tendons, pancreas, intestines, kidneys, heart, and heart valves.

The organ donation process begins with identification of the potential organ donor. Most people are potential organ donors. Exceptions include those who:

- Currently abuse intravenous drugs
- Have preexisting untreated infections, such as septicemia
- Have any malignancy other than a primary brain tumor
- Have active tuberculosis.

In the past HIV-positive clients were excluded as donors. Recent legislative changes now allow HIV-positive clients to donate to HIV-positive recipients.

The family needs to be made aware of the client's prognosis and presented with the option of donating the client's organs. Both the family's and the client's feelings about organ donation must be explored. Even if the client carries an organ donation card, many institutions will not remove any organs without a signature from a family member or other authorized person. The nurse must always respect the family's concerns and feelings during this process. Some members of certain cultural groups may have religious constraints or issues of mistrust that may interfere with the donation process.

Box 11–1 lists **brain death criteria**. Once brain death has been confirmed, the family must also understand the diagnosis and be allowed time to accept the client's death.

When caring for an adult client who is an organ donor, the nurse carries out the following:

- Maintain systolic blood pressure of 90 mmHg to keep the client's organs perfused until removal.
- Maintain urine output at >30 mL/h. This is usually accomplished by administering fluids and/or inotropic agents such as dopamine.
- Maintain oxygen saturation at 90% or greater.

### Forensic Considerations

Injuries often happen under circumstances that require legal investigation. Many injuries, particularly penetrating trauma, may involve criminal activity. Therefore, the nurse must recognize the need to identify, store, and properly transfer potential evidence for medicolegal investigations.

Each item of clothing removed from the client must be placed in a breathable container, such as a paper bag, and documented appropriately. Bullets or knives should be labeled, with their source specified, and given to the proper authorities. Holes found in clothing should not be disturbed. When it is necessary to cut off clothing, these areas should be avoided and never cut through if at all possible.

**BOX 11–1 Brain Death Criteria**

**Clinical Signs**

- Irreversible condition
- Apnea with a $Paco_2$ greater than 60 mmHg
- No response to deep stimuli
- No spontaneous movement (some spinal cord reflexes may be present)
- No gag or corneal reflex
- No oculocephalic or oculovestibular reflex
- Absence of toxic or metabolic disorders

**Confirmatory Tests**

- Cerebral blood flow study
- Electroencephalogram

The client's hands may yield important evidence, such as powder burns or residue on the skin or tissue or hair samples beneath the fingernails. In the case of death, it is recommended that paper bags be placed over the client's hands if the presence of evidence is suspected; otherwise, the evidence should be collected from nail clippings.

Identify all wounds and document these findings with pictures, diagrams, or written descriptions. Once the evidence has been collected, identified, and properly stored, ensure that it is given to the appropriate authorities. A chain of custody needs to be maintained throughout the entire process. All evidence must be identified and labeled, and documentation procedures must chronicle where and in whose possession the evidence has been. For the chain of custody to remain intact, the evidence must remain in the continuous possession of identified people and be marked and sealed in tamper-proof containers.

## NURSING CARE

Nursing care of the client who has been injured begins with a primary assessment and the initiation of collaborative interventions for any life-threatening injuries. Nursing care is directed toward the client's specific responses to trauma.

### Health Promotion

Prevention efforts can reduce the incidence and severity of trauma. Areas of health promotion and trauma prevention interventions for individuals and communities include the following:

- *Motor vehicle safety:* seat belts, air bags, helmets, driving under the influence of alcohol or drugs, reckless driving, visual or cognitive deficits in the older adult, cell phone use, driver fatigue
- *Home safety:* snow and ice removal, electrical wiring, falls, burns, drowning
- *Farm safety:* operating heavy equipment, safe storage of chemicals such as fertilizers
- *Work safety:* operating work equipment, wearing safety equipment, removal of jewelry
- *Relationships:* domestic violence, child abuse, elder abuse, or neglect
- *Communities:* gun control, gangs, condition of streets, neighborhood safety.

(In providing information about trauma prevention to members of the community, the nurse serves as a healthcare educator, political activist, and safety advocate.)

### Assessment

See Interdisciplinary Care for assessment of the client experiencing trauma.

### Nursing Diagnoses and Interventions

The trauma client has many complex and interrelated actual or potential alterations in health. The nursing care in this section focuses on client and family problems with respirations, infection, immobility, spirituality, and stress. Nursing interventions for decreased cardiac output and altered perfusion are discussed in the section of the chapter on nursing care of the client in shock. See the accompanying Nursing Care Plan on page 266.

MEDIALINK Case Study: A Client Experiencing Trauma

## NURSING CARE PLAN A Client with Multiple Injuries

Jane Souza is a 25-year-old married woman with two children who provides day care for preschool children in her home. As she is driving the interstate at 65 miles per hour, a car crosses the median and strikes her vehicle head-on. Jane, who is not wearing a seat belt, is thrown forward against the steering wheel. The front of her car is pushed up against her by the car that struck her, entrapping her lower extremities.

After extensive efforts to extricate her from the car, Jane is transported to the local trauma center. She is still conscious, is receiving high-flow oxygen by mask, and has one intravenous line in place. Her vital signs are a palpable systolic blood pressure of 80, a pulse rate of 120, and a respiratory rate of 36. On arrival, she states that she is having difficulty breathing.

### ASSESSMENT

- *Airway:* Maintainable with high-flow oxygen in place.
- *Breathing:* Respiratory rate of 36, multiple bruising and abrasions on right side of her chest, decreased breath sounds on the right side.
- *Circulation:* No palpable radial pulses; palpable brachial pulses. Monitor shows sinus tachycardia. No active external bleeding noted. Skin color pale, cool to the touch, and diaphoretic.
- *Neurologic:* Moved her fingers when asked; complains of difficulty breathing; denies that she is hurt. Pupils 4 mm, equal, and react to light. Has a broken right arm and an open fracture of the left ankle; because of these injuries, extremity movement is limited.

Because of Jane's respiratory distress, she is intubated and ventilated with 100% oxygen. Another intravenous line is inserted and O-negative blood administered.

### DIAGNOSES

- *Ineffective Breathing Pattern* related to multiple bruises and abrasions on the right side of the chest, and respiratory difficulty
- *Deficient Fluid Volume* related to acute internal blood loss (presumed because no active bleeding can be found)
- *Risk for Injury* related to trauma resuscitation

### EXPECTED OUTCOMES

- Maintain adequate oxygenation.
- Maintain adequate circulating blood volume.

### PLANNING AND IMPLEMENTATION

- Monitor airway and assist in any needed airway management.
- Explain all procedures.
- Monitor the effects of fluid and blood administration, including any changes in blood pressure and pulse.
- Prepare for transfer to the operating room for emergency surgery.
- Keep family informed about her condition.

### EVALUATION

Jane is transferred to the operating room, where it is determined that she has a ruptured spleen and a serious pelvic fracture. Jane's treatment continues in the operating room.

### CRITICAL THINKING IN THE NURSING PROCESS

1. Is the nursing diagnosis *Deficient Fluid Volume* appropriate for Jane Souza? Why or why not?
2. The assessment of a client who has experienced trauma is, in order: A = airway, B = breathing, and C = circulation. What is the rationale for this sequence?
3. Following surgery, Jane is moved to the surgical intensive care unit. She is very anxious and restless. What assessments would you make to identify the cause of her restlessness?
4. Infection is a common complication for the trauma client. Describe five risks for infection that are present from the time of injury to the time of hospital discharge.

***See Evaluating Your Response in Appendix C.***

## Ineffective Airway Clearance

The client with multiple injuries is at great risk for developing airway obstruction and apnea. Facial injuries, loose teeth, blood, and vomitus increase the risk for aspiration and obstruction. Neurologic injuries and cerebral edema alter the client's respiratory drive and ability to keep the airway clear.

- Assess if airway is patent, maintainable, or nonmaintainable. Assess for manifestations of airway obstruction: stridor, tachypnea, bradypnea, cough, cyanosis, dyspnea, decreased or absent breath sounds, changes in oxygen levels, and changes in level of consciousness. *Assessing the airway and initiating interventions are the first steps in managing the client with multiple injuries.*
- Monitor oxygen saturation by applying a pulse oximeter. Adjust oxygen flow to maintain oxygen saturation from 94% to 100%. *Changes in oxygen saturation as measured by the pulse oximeter indicate the effectiveness of the client's airway. Pulse oximetry in clients who have been exposed to carbon monoxide (i.e., house fires) is unreliable since it cannot differentiate carboxyhemoglobin from oxyhemoglobin.*
- Monitor level of consciousness. *An early sign of an ineffective airway is change in the client's behavior. If the client becomes restless, anxious, combative, or unresponsive, the effectiveness of the airway needs to be immediately evaluated and appropriate interventions initiated.*

## Risk for Infection

Traumatic injuries are considered dirty wounds. Projectiles enter the body through dirty surfaces and clothing, carrying dirt and debris into the wound. Open fractures provide a portal for the entry of bacteria and dirt. Even with surgical intervention, the wounds often remain contaminated.

- Use careful hand washing practices. *Hand washing remains the single most important factor in preventing the spread of infection.*
- Use strict standard precautions and aseptic technique when caring for wounds. *Standard precautions are essential to protect the client and the nurse from infection.* In addition:
  - Monitor wounds for odor, redness, heat, swelling, and copious or purulent drainage.

- Monitor hidden wounds, such as those under casts, by asking the client whether the pain has increased and observing for increased drainage and heat over the area of the wound.
- Ensure that cross-contamination between wounds does not occur. Collect drainage in ostomy bags if it is copious. *The skin is the first line of defense against infection. Wounds provide a portal of entry for organisms. Risk factors for wound infection include contamination, inadequate wound care, and the condition of the wound at the time of closure. Aseptic techniques used in applying and changing dressings reduce the entry of organisms.*

- Take and record vital signs, including temperature, every 2 to 4 hours. *Vital signs, particularly an elevated body temperature, indicate the presence of an infection.*
- Provide adequate fluids and nutrition. *Adequate fluids, calories, and protein are essential to wound healing.*
- Assess for manifestations of gas gangrene: fever, pain, and swelling in traumatized tissues; drainage with a foul odor. *Gas gangrene is usually caused by the organism* Clostridium perfringens. *This bacterium is found in the soil and can be introduced into the body during a traumatic injury. The organism grows in the tissues, causing necrosis; hydrogen and carbon dioxide are released, with resultant swelling of tissues. If the infection continues, tissues are progressively destroyed, and sepsis and death may result.*
- Assess status of tetanus immunization and administer tetanus toxoid or human toxin-antitoxin as prescribed. *Tetanus is caused by an exotoxin produced by* Clostridium tetani, *usually introduced through an open wound. The organism is commonly found in the soil.*
- Use strict aseptic technique when inserting catheters, suctioning, administering parenteral medications, or performing any other invasive procedure. *Using aseptic technique during invasive procedures reduces the risk of entry of organisms.*

## Impaired Physical Mobility

The client with trauma injuries is often unable to change positions independently and is at risk for complications of the integumentary, cardiovascular, gastrointestinal, respiratory, musculoskeletal, and renal systems. Clients at greatest risk are those who have had multiple injuries, spinal cord injuries, peripheral nerve injuries, and traumatic amputations. Collaborate with the physical therapist and occupational therapist (if available) to determine the most effective types and schedule of exercises and assistive devices.

- If active bleeding or edema is not present, provide active or passive exercises to affected and unaffected extremities at least once every 8 hours. *Exercise improves muscle tone, maintains joint mobility, improves circulation, and prevents contractures.*
- Help the client turn, cough, and deep breathe and use the incentive spirometer at least every 2 hours. *Changing positions, coughing, deep breathing, and incentive spirometry reduce the risk of integumentary and respiratory complications.*
- If the client is unable to be moved and positioned, consider a specialty bed, such as the kinetic continuous rotation bed (Figure 11–7 ■). *The kinetic continuous rotation bed allows continuous turning of the client; the motion decreases pulmonary complications, venous stasis, postural hypotension, urinary stasis, muscle wasting, and bone demineralization.*
- Monitor the lower extremities each day for manifestations of deep venous thrombosis: heat, swelling, and pain. Measure and record the circumference of the thigh and calf each day. If antiemboli stockings or intermittent compression stockings are used, remove them for 1 hour during each shift and assess the skin. *Venous stasis results when surrounding muscles are unable to contract and help move the blood through the veins. Thrombus (clot) formation in deep veins is a major risk for pulmonary embolism.*

## Spiritual Distress

Trauma generally strikes without warning and carries potentially devastating consequences, including severe alterations in the lives of the victim and family, and death. The traumatic death of a loved one may be the most difficult event a family may ever experience. The decision to cease life support systems or to donate organs challenges the family's belief systems and psychologic stability. Nursing care of the family (or client) experiencing spiritual distress includes the following:

- Give the family information about the option to donate the client's organs. *The decision to donate organs needs to be based on information about the client's condition, prognosis, and criteria by which brain death is determined. It is*

**Figure 11–7** ■ A kinetic continuous rotation bed provides a means of turning the client with multiple injuries to decrease the hazards of immobility.

Courtesy of Kinetic Concepts, Inc.

*important to convey to family members that organ donation is only an option and that they should not feel they are obligated to consent or are doing something wrong if they do not consent.*

- Encourage the family to ask questions and express their feelings about the traumatic event and/or organ donation. *Allowing families to express their feelings may help prevent long-term consequences such as guilt.*
- Refer the family for follow-up care. Long-term follow-up is important for the family facing the sudden death of a loved one. *Grieving is not an overnight process, and providing the family with resources that may be used in the future may help prevent future crises and dysfunction.* (For more information, see Chapter 5 ∞.)

### Post-Trauma Syndrome

Post-trauma syndrome is an intense, sustained emotional response to a disastrous event. It is characterized by emotions that range from anger to fear and by flashbacks or psychic numbing. In the initial stage, the client may be calm or may express feelings of anger, disbelief, terror, and shock. In the long-term phase, which begins anywhere from a few days to several months after the event, the client often experiences flashbacks and nightmares of the traumatic event. The client may call on ineffective coping mechanisms, such as alcohol or drugs, and withdraw from relationships.

- Assess emotional responses while providing physical care. Observe for crying, sleep problems, suspiciousness, and fear during the initial phase of treatment. If the client is unconscious, encourage family members and friends to express their feelings. *These assessments provide valuable information about the client's ability to cope with the trauma.*
- Be available if the client wishes to talk about the trauma, and encourage expression of feelings. *The client may initially deny negative feelings; this denial is a coping mechanism in the initial phase of recovery.*
- Teach relaxation techniques, such as deep breathing, progressive muscle relaxation, or imagery (see Chapter 9 ∞). *These techniques are often useful in coping when thoughts of the trauma recur.*
- Refer the client and family members for counseling, psychotherapy, or support groups as appropriate. *Continued therapy may be necessary in assisting the client and family to resolve the acute and long-term effects of trauma.*

## Using NANDA, NIC, and NOC

Chart 11–1 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with multiple injuries.

## Community-Based Care

Address the following topics to prepare the client and family for home care:

- The type of home environment to which the client will be returning, including any changes that will be required to let the client function in that environment
- Medications, dressings, wound care, equipment, and supplies
- Special diet, if needed
- Rehabilitation plan and its effect on the client's family

NANDA, NIC, AND NOC LINKAGES

CHART 11–1 **The Client Experiencing Trauma**

**NANDA**

Post-Trauma Syndrome

↓

**NIC**

Counseling

Coping Enhancement

Simple Relaxation Therapy

Support System Enhancement

↓

**NOC**

Anxiety Level

Coping

Stress Level

Social Support

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Follow-up appointments with the physician or at the trauma clinic
- Emotional changes that the client may undergo as a result of the trauma
- Helpful resources:
  - Home health care
  - Community support groups
  - National Institute of Neurological Disorders and Stroke.

# THE CLIENT EXPERIENCING SHOCK

**Shock** is a clinical syndrome characterized by a systemic imbalance between oxygen supply and demand. This imbalance results in a state of inadequate blood flow to body organs and tissues, causing life-threatening cellular dysfunction.

## Overview of Cellular Homeostasis and Hemodynamics

To maintain cellular metabolism, cells of all body organs and tissues require a regular and consistent supply of oxygen and the removal of metabolic wastes. This homeostatic regulation is maintained primarily by the cardiovascular system and depends on four physiologic components:

1. A cardiac output sufficient to meet bodily requirements
2. An uncompromised vascular system, in which the vessels have a diameter sufficient to allow unimpeded blood flow

and have good tone (the ability to constrict or dilate to maintain normal pressure)
3. A volume of blood sufficient to fill the circulatory system, and a blood pressure adequate to maintain blood flow
4. Tissues that are able to extract and use the oxygen delivered through the capillaries.

In a healthy person, these components function as a system to maintain tissue perfusion. During shock, however, one or more of these components are disrupted. An understanding of basic hemodynamics is necessary to understand the pathophysiology of shock:

- **Stroke volume (SV)** is the amount of blood pumped into the aorta with each contraction of the left ventricle.
- **Cardiac output (CO)** is the amount of blood pumped per minute into the aorta by the left ventricle. CO is determined by multiplying the stroke volume (SV) by the heart rate (HR): CO = SV × HR.

**PRACTICE ALERT**
Cardiac output (CO) = stroke volume (SV) × heart rate (HR).

- **Mean arterial pressure (MAP)** is the product of cardiac output and systemic vascular resistance (SVR): MAP = CO × SVR. When CO, SVR, or total blood volume rises, MAP and tissue perfusion increase. Conversely, when CO, SVR, or total blood volume falls, MAP and tissue perfusion decrease.
- The sympathetic nervous system maintains the smooth muscle surrounding the arteries and arterioles in a state of partial contraction called *sympathetic tone*. Increased sympathetic stimulation increases vasoconstriction and SVR; decreased sympathetic stimulation allows vasodilatation, which decreases SVR.

## Pathophysiology

When one or more cardiovascular components do not function properly, the body's hemodynamic properties are altered. Consequently, tissue perfusion may be inadequate to sustain normal cellular metabolism. The result is the clinical syndrome known as shock. The manifestations of shock result from the body's attempts to maintain vital organs (heart and brain) and to preserve life following a drop in cellular perfusion. However, if the injury or condition triggering shock is severe enough or of long enough duration, then cellular hypoxia and cellular death occur.

Shock is triggered by a sustained drop in mean arterial pressure. This drop can occur after a decrease in cardiac output, a decrease in the circulating blood volume, or an increase in the size of the vascular bed due to peripheral vasodilatation. If intervention is timely and effective, the physiologic events that characterize shock may be stopped; if not, shock may lead to death. See Table 11–5 for classifications of shock.

### Stage I: Early, Reversible, and Compensatory Shock

The initial stage of shock begins when baroreceptors in the aortic arch and the carotid sinus detect a sustained drop in MAP of less than 10 mmHg from normal levels. The circulating blood volume may decrease (usually to less than 500 mL), but not enough to cause serious effects.

The body reacts to the decrease in arterial pressure as it would to any physical stressor. The cerebral integration center initiates the body's response systems, causing the sympathetic nervous system to increase the heart rate and the force of cardiac contraction, thus increasing cardiac output. Sympathetic stimulation also causes peripheral vasoconstriction, resulting in increased systemic vascular resistance and a rise in arterial pressure. The net result is that the perfusion of cells, tissues, and organs is maintained.

Symptoms are almost imperceptible during the early stage of shock. The pulse rate may be slightly elevated. If the injury is minor or of short duration, arterial pressure is usually maintained, and no further symptoms occur.

Compensatory shock begins after the MAP falls 10 to 15 mmHg below normal levels. The circulating blood volume is reduced by 25% to 35% (1000 mL or more), but compensatory mechanisms are able to maintain blood pressure and tissue perfusion to vital organs, thereby preventing cell damage.

- Stimulation of the sympathetic nervous system results in the release of epinephrine from the adrenal medulla and the release of norepinephrine from the adrenal medulla and the sympathetic fibers. Both hormones rapidly stimulate the alpha- and

TABLE 11–5 **Classification of Hemorrhagic Shock and Client Presentation**

| | COMPENSATED/ CLASS I | MILD/CLASS II | MODERATE/CLASS III | SEVERE/CLASS IV |
|---|---|---|---|---|
| Blood loss | Up to 750 mL | 750–1500 mL | 1500–2000 mL | >2000 mL |
| Percent of blood volume loss | Up to 15% | 15–30% | 30–40% | >40% |
| Heart rate | <100 | >100 | >120 | >140 |
| Blood pressure | Normal or increased | Normal | Decreased | Markedly decreased |
| Pulse pressure | Normal or increased | Decreased | Decreased | Decreased |
| Capillary refill | Normal | Mild increase | Usually delayed | Delayed |
| Respiratory rate | Normal | Mild increase | Moderate tachypnea | Markedly tachypnea |
| Urine output (mL/h) | >30 mL | 20–30 mL | 5–15 mL | Anuria |
| Mental status | Normal–slightly anxious | Mildly anxious–agitated | Anxious–confused | Lethargic–obtunded |

beta-adrenergic fibers. Stimulated alpha-adrenergic fibers cause vasoconstriction in the blood vessels supplying the skin and most of the abdominal viscera. Perfusion of these areas decreases. Stimulated beta-adrenergic fibers cause vasodilatation in vessels supplying the heart and skeletal muscles ($beta_1$ response), and increase the heart rate and force of cardiac contraction ($beta_2$ response). Further, blood vessels in the respiratory system dilate, and the respiratory rate increases ($beta_2$ response). Thus, stimulation of the sympathetic nervous system results in increased cardiac output and oxygenation of these tissues.

- The renin–angiotensin response occurs as the blood flow to the kidneys decreases. Renin released from the kidneys converts a plasma protein to angiotensin II, which causes vasoconstriction and stimulates the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to reabsorb water and sodium and to lose potassium. The absorption of water maintains circulating blood volume while increased vasoconstriction increases SVR, maintaining central vascular volume and raising blood pressure.
- The hypothalamus releases adrenocorticotropic hormone, causing the adrenal glands to secrete aldosterone. Aldosterone promotes the reabsorption of water and sodium by the kidneys, preserving blood volume and pressure.
- The posterior pituitary gland releases antidiuretic hormone, which increases renal reabsorption of water to increase intravascular volume. The combined effects of hormones released by the hypothalamus and posterior pituitary glands work to conserve central vascular volume.
- As MAP falls in the compensatory stage of shock, decreased capillary hydrostatic pressure causes a fluid shift from the interstitial space into the capillaries. The net gain of fluid raises the blood volume.

Working together, these compensatory mechanisms can maintain MAP for only a short period of time. During this period, the perfusion and oxygenation of the heart and brain are adequate. If effective treatment is provided, the process is arrested, and no permanent damage occurs. However, unless the underlying cause of shock is reversed, these compensatory mechanisms soon become harmful, and shock perpetuates shock.

## Stage II: Intermediate or Progressive Shock

The progressive stage of shock occurs after a sustained decrease in MAP of 20 mmHg or more below normal levels and a fluid loss of 35% to 50% (1800 to 2500 mL of fluid). Although the compensatory mechanisms in the previous state remain activated, they are no longer able to maintain MAP at a level sufficient to ensure perfusion of vital organs.

The vasoconstriction response that first helped sustain MAP eventually limits blood flow to the point that cells become oxygen deficient. To remain alive, the affected cells switch from aerobic to anaerobic metabolism. The lactic acid formed as a by-product of anaerobic metabolism contributes to an acidotic state at the cellular level. As a result, adenosine triphosphate, the source of cellular energy, is produced inefficiently. Lacking energy, the sodium-potassium pump fails. Potassium moves out of the cell, while sodium and water move inward. As this process continues, the cell swells, cell membrane integrity is lost, and cell organelles are damaged. Lysosomes within the cell spill out their digestive enzymes, which disintegrate any remaining organelles. Some enzymes spread to adjacent cells, where they erode and rupture cell membranes.

The acid by-products of anaerobic metabolism dilate the precapillary arterioles and constrict the postcapillary venules. This causes increased hydrostatic pressure within the capillary, and fluid shifts back into the interstitial space. The capillaries also become increasingly permeable, allowing serum proteins to shift from the vascular space into the interstitium. The buildup of plasma proteins increases the osmotic pressure in the interstitium, further accelerating the fluid shift out of the capillaries.

Throughout this period, the heart rate and vasoconstriction increase; however, perfusion of the skin, skeletal muscles, kidneys, and gastrointestinal organs is greatly diminished. Cells in the heart and brain become hypoxic while other body cells and tissues become ischemic and anoxic. A generalized state of acidosis and hyperkalemia ensues (see Chapter 10 ∞). Unless this stage of shock is treated rapidly, the client's chances of survival are poor.

## Stage III: Refractory or Irreversible Shock

If shock progresses to the irreversible stage, tissue anoxia becomes so generalized and cellular death so widespread that no treatment can reverse the damage. Even if MAP is temporarily restored, too much cellular damage has occurred to maintain life. Death of cells is followed by death of tissues, which results in death of organs. Death of vital organs contributes to subsequent death of the body.

## Effects of Shock on Body Systems

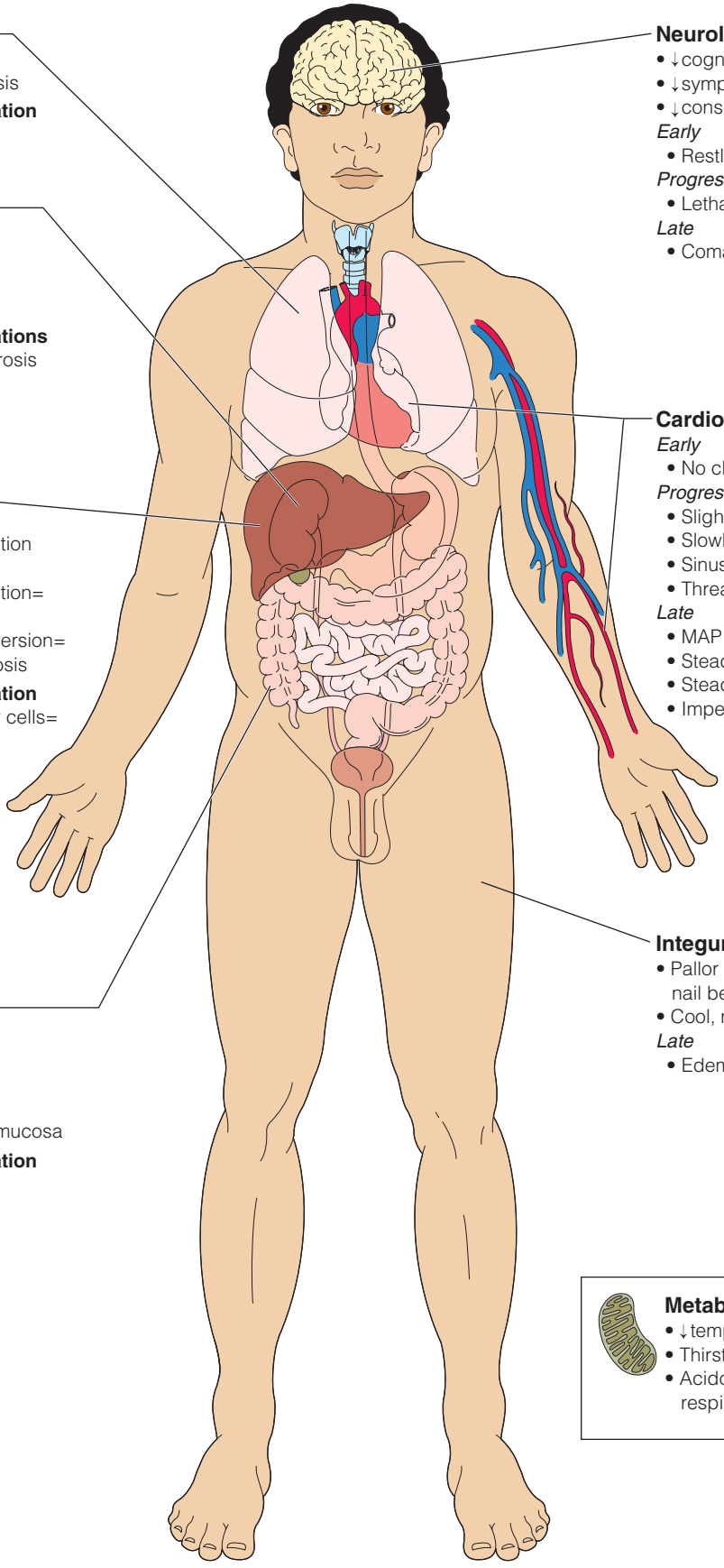
Whatever its causes, shock produces predictable effects on the body's organ systems. (See *Multisystem Effects of Shock* on the following page.)

***CARDIOVASCULAR SYSTEM*** The perfusion and oxygenation of the heart are adequate in the early stages of shock. As shock progresses, myocardial cells become hypoxic, and myocardial muscle function diminishes. Initially, the blood pressure may be normal or even slightly elevated (as a result of compensatory mechanisms) and the heart rate only slightly increased. Sympathetic stimulation increases the heart rate (a sinus tachycardia of 120 beats per minute is common) in an effort to increase cardiac output. As a result of vasoconstriction and decreased blood volume, the palpated pulse is rapid, weak, and thready; as shock progresses, peripheral pulses are usually nonpalpable.

Tachycardia reduces the time available for left ventricular filling and coronary artery perfusion, further reducing cardiac output. With progressive shock, altered acid–base balance, hypoxia, and hyperkalemia damage the heart's electrical systems and contractility. Consequently, cardiac dysrhythmias may develop. Decreased blood volume with decreased venous return also decreases cardiac output, and blood pressure falls.

The blood pressure changes produced by shock are characterized by a progressive decrease in both systolic and diastolic pressures and a narrowing pulse pressure. Auscultation of

# MULTISYSTEM EFFECTS **of Shock**



**Respiratory**
- ↑ respiratory rate
- Respiratory acidosis

**Potential Complication**
- ARDS

**Urinary**
- ↓ renal perfusion
- ↓GFR

*Late*
- Oliguria

**Potential Complications**
- Acute tubular necrosis
- Kidney failure

**Hepatic**

*Early*
- ↑ glucose production

*Progressive*
- ↓glucose production= hypoglycemia
- ↓lactic acid conversion= metabolic acidosis

**Potential Complication**
- Destroyed Kupffer cells= systemic bacterial infections

**Gastrointestinal**

*Early*
- ↓GI motility

*Late*
- Paralytic ileus
- Ulceration of GI mucosa

**Potential Complication**
- Bowel necrosis

**Neurologic**
- ↓cognition
- ↓sympathetic activity
- ↓consciousness

*Early*
- Restlessness, apathy

*Progressive*
- Lethargy

*Late*
- Coma

**Cardiovascular**

*Early*
- No change

*Progressive*
- Slightly ↑BP
- Slowly ↑HR
- Sinus tachycardia
- Thready pulse

*Late*
- MAP <60 mmHg
- Steadily ↓BP
- Steadily ↓CO
- Imperceptible pulses

**Integumentary**
- Pallor (skin, lips, oral mucosa, nail beds, conjunctiva)
- Cool, moist skin

*Late*
- Edema

**Metabolic Processes**
- ↓temperature
- Thirst
- Acidosis (metabolic and respiratory)

blood pressure is often difficult or impossible and is an inaccurate reflection of blood pressure status. For this reason, hemodynamic monitoring is usually instituted to follow the client's cardiovascular status accurately.

***RESPIRATORY SYSTEM*** During shock, oxygen delivery to cells may be impaired by a drop in circulating blood volume or, in the case of blood loss, by an insufficient number of red blood cells that carry oxygen. Although the respiratory rate increases because of compensatory mechanisms that promote oxygenation, the number of alveoli that are perfused decreases, and gas exchange is impaired. As a result, oxygen levels in the blood decrease, and carbon dioxide levels increase. As perfusion of the lungs diminishes, carbon dioxide is retained, and respiratory acidosis occurs.

A complication of decreased perfusion of the lungs is acute respiratory distress syndrome (ARDS), or "shock lung." The exact mechanism that produces ARDS is unknown, but some contributing factors have been identified. The pulmonary capillaries become increasingly permeable to proteins and water, resulting in noncardiogenic pulmonary edema. Production of surfactant (which controls surface tension within alveoli) is impaired, and the alveoli collapse or fill with fluid. This potentially lethal form of respiratory failure may result from any condition that causes hypoperfusion of the lungs, but it is more common in shock caused by hemorrhage, severe allergic responses, trauma, and infection. (ARDS is discussed further in Chapter 39 ∞.)

***GASTROINTESTINAL AND HEPATIC SYSTEMS*** The gastrointestinal organs normally receive 25% of the cardiac output through the splanchnic circulation. Shock constricts the splanchnic arterioles and redirects arterial blood flow to the heart and brain. Consequently, gastrointestinal organs become ischemic and may be irreversibly damaged.

Gastric mucosa tends to ulcerate when it becomes ischemic. Lesions of the gastric and duodenal mucosa (called *stress ulcers*) can develop within hours of severe trauma, sepsis, or burns (Porth, 2005). Gastrointestinal ulcers may hemorrhage within 2 to 10 days following the original cause of shock. In addition, the permeability of damaged mucosa increases, allowing enteric bacteria or their toxins to enter the abdominal cavity and then progress to the circulation, resulting in sepsis.

Gastric and intestinal motility is impaired during shock, and paralytic ileus may result. If the episode of shock is prolonged, necrosis of the bowel may occur. In many cases, alterations in the structure and function of the gastrointestinal tract impair absorption of nutrients, such as protein and glucose.

Shock also alters the metabolic functions of the liver. Initially, *gluconeogenesis* (the process of forming glucose from noncarbohydrate sources) and *glycogenolysis* (the breakdown of glycogen into glucose) increase. This process allows blood glucose levels to increase as the body attempts to respond to the stressor; however, as shock progresses, liver functions are impaired, and hypoglycemia develops. Metabolism of fats and protein is impaired, and the liver can no longer effectively remove lactic acid, contributing to the development of metabolic acidosis.

The destruction of the liver's reticuloendothelial Kupffer cells (phagocytes that destroy bacteria) causes a further problem. Bacteria may proliferate within the circulatory system, causing overwhelming bacterial infection and toxicity.

***NEUROLOGIC SYSTEM*** The primary effects of shock on the neurologic system involve changes in mental status and orientation. Cerebral hypoxia produces altered levels of consciousness, beginning with apathy and lethargy and progressing to coma. A common early symptom of cerebral hypoxia is restlessness. Continued ischemia of brain cells eventually causes swelling, resulting in cerebral edema, neurotransmitter failure, and irreversible brain cell damage.

As cerebral ischemia worsens, the sympathetic activity and vasomotor centers are depressed. This leads to a loss of sympathetic tone, causing systemic vasodilatation and pooling of blood in the periphery. As a result, venous return and cardiac output further decrease.

**PRACTICE ALERT**

An early sign of shock is a change in the level of consciousness. Late signs of shock include mental status changes, hypotension, and marked tachycardia.

***RENAL SYSTEM*** Blood that normally perfuses the kidneys is shunted to the heart and brain during the progressive stage of shock, resulting in renal hypoperfusion. The drop in renal perfusion is reflected in a corresponding decrease in the glomerular filtration rate. Urine output is reduced, and the urine that is produced is highly concentrated. Oliguria of <20 mL/h indicates progressive shock.

Healthy kidneys can tolerate a drop in perfusion for only about 20 minutes; thereafter, acute tubular necrosis develops (Porth, 2005). As tubular necrosis occurs, epithelial cells slough off and block the tubules, disrupting nephron function. The accumulating loss of functional nephrons eventually causes renal failure. Without normal renal function, metabolic waste products are retained in the plasma.

If treatment restores renal perfusion, the kidneys can regenerate the lost epithelial cells in the tubules, and renal function usually returns to normal. However, in a client who is older or chronically ill or in sustained shock, loss of renal function may become permanent.

***EFFECTS ON SKIN, TEMPERATURE, AND THIRST*** In most types of shock, blood vessels supplying the skin are vasoconstricted, and the sweat glands are activated. As a result, changes in skin color occur. The skin of Caucasian clients becomes pale. In people with darker skin (such as those of African, Hispanic, or Mediterranean descent), shock-related skin color changes may be assessed as paleness of the lips, oral mucous membranes, nail beds, and conjunctiva. The skin is usually cool and moist and, in the later stages of shock, often edematous.

The body temperature decreases as shock progresses, the result of a decrease in overall body metabolism. Some people in shock become thirsty, probably a response to decreased blood volume and increased serum osmolality (Porth, 2005).

## Types of Shock

Shock is identified according to its underlying cause. All types of shock progress through the same stages and exert similar effects on body systems. Any differences are noted in the following discussion.

### Hypovolemic Shock

**Hypovolemic shock** is caused by a decrease in intravascular volume of 15% or more (Porth, 2005). In hypovolemic shock, the venous blood returning to the heart decreases, and ventricular filling drops. As a result, stroke volume, cardiac output, and blood pressure decrease. Hypovolemic shock is the most common type of shock, and it often occurs simultaneously with other types.

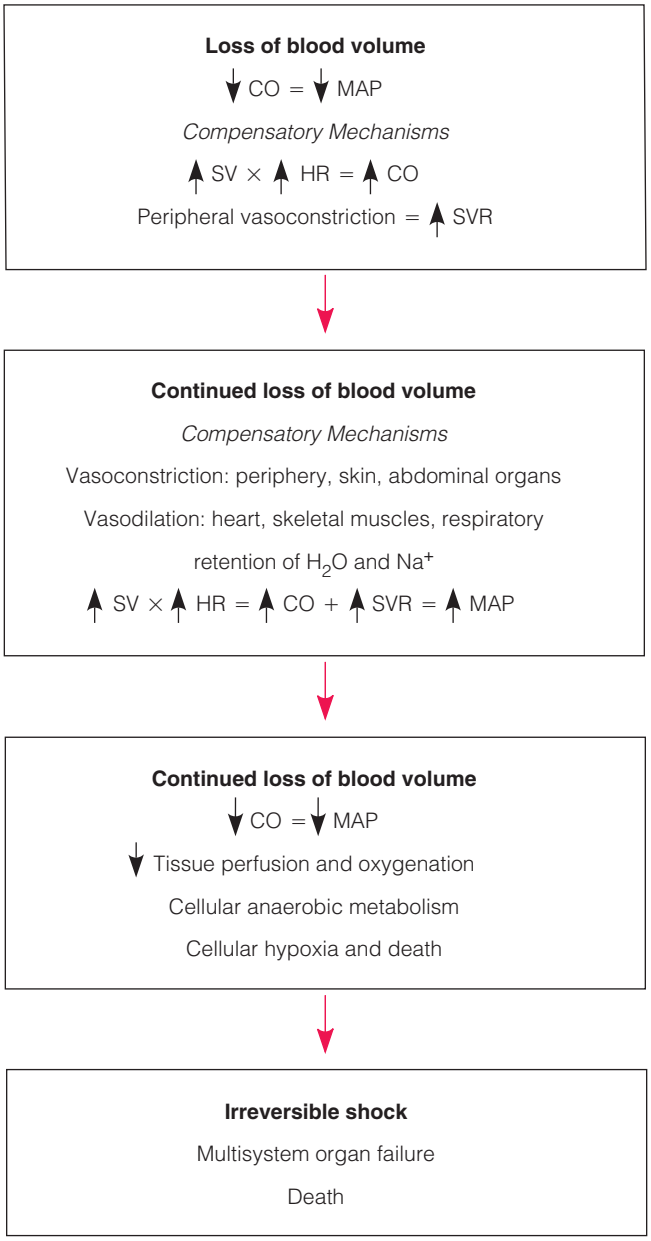
The decrease in circulating blood volume that triggers hypovolemic shock may result from:

- Loss of blood volume from hemorrhage (from surgery, trauma, gastrointestinal bleeding, blood coagulation disorders, ruptured esophageal varices)
- Loss of intravascular fluid from the skin due to injuries such as burns (see Chapter 17 ∞)
- Loss of blood volume from severe dehydration
- Loss of body fluid from the gastrointestinal system due to persistent and severe vomiting or diarrhea, or continuous nasogastric suctioning
- Renal losses of fluid due to the use of diuretics or to endocrine disorders such as diabetes insipidus
- Conditions causing fluid shifts from the intravascular compartment to the interstitial space
- Third spacing due to such disorders as liver diseases with ascites, pleural effusion, or intestinal obstruction

Hypovolemic shock affects all body systems. Its effects vary depending on the client's age, general state of health, extent of injury or severity of illness, length of time before treatment is provided, and the rate of volume loss.

The manifestations of hypovolemic shock result directly from the decrease in circulating blood volume and the initiation of compensatory mechanisms (Figure 11–8 ■). The loss of circulating blood volume reduces cardiac output by decreasing venous return to the heart. As a result, blood pressure drops. The carotid and cardiac baroreceptors sense the decrease in blood pressure and communicate it to the vasomotor centers in the brainstem. The vasomotor centers then induce the sympathetic compensatory responses. If the fluid loss is less than 500 mL, activation of the sympathetic response is generally adequate to restore cardiac output and blood pressure to near normal, although the heart rate may remain elevated.

With a sustained loss of blood volume (1000 mL or more), the shock stage progresses. Heart rate and vasoconstriction increase, and blood flow to the skin, skeletal muscles, kidneys, and abdominal organs decreases. Several renal mechanisms and a decline in capillary pressure help conserve blood volume. Eventually, the amount of blood flowing to cells is too low to oxygenate them and sustain production of cellular energy. Anaerobic metabolism begins, producing an acidotic environment for cells. As a result, cells lose their physical integrity. If untreated, shock causes multiple organ failure, and death results. Manifestations of various stages of hypovolemic shock are listed in the box on the following page.




**Key** CO: Cardiac output
HR: Heart rate
MAP: Mean arterial pressure
SV: Stroke volume
SVR: Systemic vascular resistance

**Figure 11–8** ■ The stages of hypovolemic shock.

***OLDER ADULTS EXPERIENCING HYPOVOLEMIA*** With aging comes a relative decrease in sympathetic activity in relation to the cardiovascular system. Cardiac compliance also decreases with age. Atherosclerosis affects many vital organs' sensitivity to even the slightest reduction in blood flow. Many elderly people experience secondary volume depletion due to chronic diuretic

MEDIALINK Hypovolemic Shock Animation

### MANIFESTATIONS of Hypovolemic Shock

**INITIAL STAGE**

- Blood pressure: normal to slightly decreased
- Pulse: slightly increased from baseline
- Respirations: normal (baseline)
- Skin: cool, pale (in periphery), moist
- Mental status: alert and oriented
- Urine output: slight decrease
- Other: thirst, decreased capillary refill time

**COMPENSATORY AND PROGRESSIVE STAGES**

- Blood pressure: hypotension
- Pulse: rapid, thready
- Respirations: increased
- Skin: cool, pale (includes trunk); poor turgor with fluid loss, edematous with fluid shift
- Mental status: restless, anxious, confused, or agitated
- Urine output: oliguria (less than 30 mL/h)
- Other: marked thirst, acidosis, hyperkalemia, decreased capillary refill time, decreased or absent peripheral pulses

**IRREVERSIBLE STAGE**

- Blood pressure: severe hypotension (often, systolic pressure is below 80 mmHg)
- Pulse: very rapid, weak
- Respirations: rapid, shallow; crackles and wheezes
- Skin: cool, pale, mottled with cyanosis
- Mental status: disoriented, lethargic, comatose
- Urine output: anuria
- Other: loss of reflexes, decreased or absent peripheral pulses

use or malnutrition. Also, clients prescribed beta-blockers may not present with tachycardia as an early indicator of shock. This important sign can be masked due to beta-adrenergic blockade. These clients will require early invasive monitoring in order to avoid excessive or inadequate volume restoration. This should be considered early in the treatment phase.

## Cardiogenic Shock

**Cardiogenic shock** occurs when the heart's pumping ability is compromised to the point that it cannot maintain cardiac output and adequate tissue perfusion. Cardiac disorders are discussed in Chapters 30 and 32 ∞; this section focuses only on the effects of shock caused by these disorders.

The loss of the pumping action of the heart may be caused by the following conditions:

- Myocardial infarction
- Cardiac tamponade
- Restrictive pericarditis
- Cardiac arrest
- Dysrhythmias, such as fibrillation or ventricular tachycardia
- Pathologic changes in the valves
- Cardiomyopathies from hypertension, alcohol, bacterial or viral infections, or ischemia
- Complications of cardiac surgery
- Electrolyte imbalances (especially changes in normal potassium and calcium levels)
- Drugs affecting cardiac muscle contractility
- Head injuries causing damage to the cardioregulatory center.

Myocardial infarction is the most common cause of cardiogenic shock. Clients admitted to the hospital for treatment of myocardial infarction or cardiac surgery are at risk for cardiogenic shock. The severity and progression of shock are related to the amount of myocardial damage.

Whatever the cardiogenic cause, the decrease in cardiac output causes a decrease in MAP. Heart rate may increase in response to compensatory mechanisms. However, tachycardia increases myocardial oxygen consumption and decreases coronary perfusion. The myocardium becomes progressively depleted of oxygen, causing further myocardial ischemia and necrosis. The typical sequence of shock is essentially unchanged in cardiogenic shock.

Cyanosis, however, is more common in cardiogenic shock, because stagnating blood increases extraction of oxygen from the hemoglobin at the capillary beds. As a result, the skin, lips, and nail beds may appear cyanotic. As cardiac failure and cardiogenic shock progress, left ventricular end-diastolic pressure increases. The increase is transmitted to the pulmonary capillary bed, and pulmonary edema may occur. Retention of blood in the right side of the heart increases right atrial pressure, which leads to jugular venous distention as a result of backflow through the vena cava. Manifestations of cardiogenic shock are listed in the box below.

## Obstructive Shock

**Obstructive shock** is caused by an obstruction in the heart or great vessels that either impedes venous return or prevents effective cardiac pumping action. The causes of obstructive shock are impaired diastolic filling (e.g., pericardial tamponade or pneumothorax), increased right ventricular afterload (e.g., pulmonary emboli), and increased left ventricular afterload (e.g., aortic stenosis, abdominal distention). The manifestations are the result of decreased cardiac output and blood pressure, with reduced tissue perfusion and cellular metabolism.

## Distributive Shock

**Distributive shock** (also called **vasogenic shock**) includes several types of shock that result from widespread vasodilatation and decreased peripheral resistance. Because the blood volume does not change, relative hypovolemia results.

### MANIFESTATIONS of Cardiogenic Shock

- Blood pressure: hypotension
- Pulse: rapid, thready; distention of veins of hands and neck
- Respirations: increased, labored; crackles and wheezes; pulmonary edema
- Skin: pale, cyanotic, cold, moist
- Mental status: restless, anxious, lethargic progressing to comatose
- Urine output: oliguria to anuria
- Other: dependent edema; elevated central venous pressure (CVP); elevated pulmonary capillary wedge pressure; arrhythmias

MEDIALINK Case Study: Identifying Types of Shock

## Septic Shock

**Septic shock**, the leading cause of death for clients in intensive care units, is one part of a progressive syndrome called *systemic inflammatory response syndrome* (SIRS). This condition is most often the result of gram-negative bacterial infections (i.e., *Pseudomonas, E. coli, Klebsiella*), but may also follow gram-positive infections from *Staphylococcus* and *Streptococcus* bacteria. Gram-negative sepsis has greatly increased in the past 10 years, with a 60% mortality rate despite treatment. The pathophysiology of septic shock is complex and not completely understood.

Clients at risk for developing infections leading to septic shock include those who are hospitalized, have debilitating chronic illnesses, or have poor nutritional status. The risk is heightened after invasive procedures or surgery. Other clients at risk of septic shock include older adults and those who are immunocompromised. Portals of entry for infection that may lead to septic shock are as follows:

- *Urinary system:* catheterizations, suprapubic tubes, cystoscopy
- *Respiratory system:* suctioning, aspiration, tracheostomy, endotracheal tubes, respiratory therapy, mechanical ventilators
- *Gastrointestinal system:* peptic ulcers, ruptured appendix, peritonitis
- *Integumentary system:* surgical wounds, intravenous catheters, intra-arterial catheters, invasive monitoring, decubitus ulcers, burns, trauma
- *Female reproductive system:* elective surgical abortion, ascending infections from transmission of bacteria during the intrapartal and postpartal periods, tampon use, sexually transmitted infections.

Septic shock begins with *septicemia* (the presence of pathogens and their toxins in the blood). As pathogens are destroyed, their ruptured cell membranes allow endotoxins to leak into the plasma. The endotoxins disrupt the vascular system, coagulation mechanism, and immune system and trigger an immune and inflammatory response (see Chapter 12 ∞ for more information). For this reason, the initial effects of septic shock differ from those of hypovolemic and cardiogenic shock; cardiac output is high and systemic vascular resistance is low.

Endotoxins directly damage the endothelial lining of small blood vessels first; the small blood vessels of the kidneys and lungs are most susceptible. Cellular damage stimulates the release of vasoactive proteins and activates coagulation factor XII. The vasoactive proteins stimulate peripheral vasodilatation and increase capillary permeability; the activation of coagulation factors results in the production of multiple intravascular blood clots.

As a result of the increased capillary permeability and vasodilatation, fluid shifts from the intravascular space to the interstitial space. Hypovolemia results as fluid volume is lost from the circulating blood. Hypovolemia and intravascular coagulation alter oxygenation and cellular metabolism, leading to anaerobic metabolism, lactic acidosis, and cellular death.

Septic shock has an early phase and a late phase. In early septic shock (sometimes called the *warm phase*), vasodilatation results in weakness and warm, flushed skin, and the septicemia often causes high fever and chills. In late septic shock (sometimes called the *cold phase*), hypovolemia and activity of the compensatory mechanisms result in typical shock manifestations, including cold, moist skin; oliguria; and changes in mental status. Death may result from respiratory failure, cardiac failure, or renal failure. Manifestations of septic shock are listed in the box below.

*Toxic shock syndrome* is an especially virulent form of septic shock, occurring most frequently in menstruating women who use tampons. It is thought that bacterial toxins diffuse from the site of infection in the vagina into the circulation. The toxins then trigger a widespread inflammatory response and septic shock. The manifestations of toxic shock syndrome include extreme hypotension, hyperpyrexia, headache, myalgia, confusion, skin rash, vomiting, and diarrhea (Porth, 2005).

Disseminated intravascular coagulation (DIC), a generalized response to injury, is a potential risk in septic shock. This condition is characterized by simultaneous bleeding and clotting throughout the vasculature. Sepsis injures blood cells, causing platelet aggregation and decreased blood flow. As a result, blood clots form throughout the microcirculation. The clotting slows circulation further while stimulating excess fibrinolysis. As the body's stores of clotting factors are depleted, generalized bleeding begins. DIC is further discussed in Chapter 34 ∞.

## Neurogenic Shock

**Neurogenic shock** is the result of an imbalance between parasympathetic and sympathetic stimulation of vascular smooth muscle. If parasympathetic overstimulation or sympathetic understimulation persists, sustained vasodilatation occurs, and blood pools in the venous and capillary beds.

Neurogenic shock causes dramatic reduction in systemic vascular resistance as the size of the vascular compartment increases. As systemic vascular resistance decreases, pressure in the blood vessels becomes too low to drive nutrients across capillary membranes, and cellular metabolism is impaired.

### MANIFESTATIONS of Septic Shock

**EARLY (WARM) SEPTIC SHOCK**

- Blood pressure: normal to hypotension
- Pulse: increased, thready
- Respirations: rapid and deep
- Skin: warm, flushed
- Mental status: alert, oriented, anxious
- Urine output: normal
- Other: increased body temperature; chills; weakness; nausea, vomiting, diarrhea; decreased CVP

**LATE (COLD) SEPTIC SHOCK**

- Blood pressure: hypotension
- Pulse: tachycardia, arrhythmias
- Respirations: rapid, shallow, dyspneic
- Skin: cool, pale, edematous
- Mental status: lethargic to comatose
- Urine output: oliguria to anuria
- Other: normal to decreased body temperature; decreased CVP

The following conditions can cause neurogenic shock by increasing parasympathetic stimulation or inhibiting sympathetic stimulation of the smooth muscle of blood vessels:

- Head injury
- Trauma to the spinal cord (spinal shock, a form of neurogenic shock, is described in Chapter 45 ∞)
- Insulin reactions (which cause hypoglycemia, decreasing glucose to the medulla)
- Central nervous system depressant drugs (such as sedatives, barbiturates, or narcotics)
- Anesthesia (spinal and general)
- Severe pain
- Prolonged exposure to heat.

Bradycardia occurs early, but tachycardia begins as compensatory mechanisms are initiated. Central venous pressure drops as veins dilate, venous return to the heart decreases, stroke volume decreases, and MAP falls. In early stages, the extremities are warm and pink (from the pooling of blood), but as shock progresses, the skin becomes pale and cool. Manifestations of neurogenic shock are listed in the box below.

### Anaphylactic Shock

**Anaphylactic shock** is the result of a widespread hypersensitivity reaction (called *anaphylaxis*). The pathophysiology in this type of shock includes vasodilatation, pooling of blood in the periphery, and hypovolemia with altered cellular metabolism. These physiologic alterations occur when a sensitized person has contact with an *allergen* (a foreign substance to which an individual is hypersensitive). Many different allergens can cause anaphylactic shock, including medications, blood administration, latex, foods, snake venom, and insect stings.

Anaphylactic shock does not occur with the first exposure to an allergen. With the first exposure to a foreign substance (the *antigen*), the body produces specific immunoglobulin E (IgE) antibodies against this antigen. The person is thus sensitized to that specific antigen. With subsequent exposure, the antigen reacts with the already formed IgE antibodies, disrupting cellular integrity. In addition, large amounts of histamine and other vasoactive amines are released and distributed through the circulatory system. These substances cause increased capillary permeability and massive vasodilatation, resulting in profound hypotension and eventual vascular collapse.

Histamine also causes constriction of smooth muscles in the bladder, uterus, intestines, and bronchioles. Respiratory distress, bronchospasm, laryngospasm, and severe abdominal cramping result. Serotonin (a neurotransmitter with vasoconstrictive properties) is released, further affecting respiratory status by increasing capillary permeability in the lungs. As a result, plasma leaks into the alveoli, gas exchange is impaired, and pulmonary edema may occur.

Anaphylactic shock begins and progresses rapidly. Manifestations may begin within 20 minutes of contact with an antigen. Unless appropriate intervention is provided, death can occur within a matter of minutes. Because anaphylaxis is rapid and potentially lethal, people with known allergies should carry some form of warning (such as a Medic-Alert bracelet) informing others of their susceptibility. Healthcare providers should be extremely careful to assess and document allergies or previous drug reactions. Manifestations of anaphylactic shock are listed in the box below.

**MANIFESTATIONS of Neurogenic Shock**

- Blood pressure: hypotension
- Pulse: slow and bounding
- Respirations: vary
- Skin: warm, dry
- Mental status: anxious, restless, lethargic progressing to comatose
- Urine output: oliguria to anuria
- Other: lowered body temperature

## INTERDISCIPLINARY CARE

Medical care for the client in shock focuses on treating the underlying cause, increasing arterial oxygenation, and improving tissue perfusion. Depending on the cause and type of shock, interventions include emergency care measures, oxygen therapy, fluid replacement, and medications. Emergency care is often the first course of collaborative action taken to arrest shock, as discussed earlier in this chapter.

### Diagnosis

The following diagnostic tests can help identify the type of shock and assess the client's physical status. Measurements include:

- *Blood hemoglobin* and *hematocrit.* Changes in hemoglobin and hematocrit concentrations usually occur in hypovolemic shock. These changes reflect the underlying etiology. In hypovolemic shock resulting from hemorrhage, the hemoglobin and hematocrit concentrations are lower than normal. In hypovolemic shock resulting from intravascular fluid loss, by contrast, the hemoglobin and hematocrit concentrations are higher than normal.
- *Arterial blood gases (ABGs),* to determine oxygen and carbon dioxide levels and pH. The effects of shock and of the body's compensatory mechanisms cause a decrease in pH (indicating acidosis), a decrease in the partial pressure of

**MANIFESTATIONS of Anaphylactic Shock**

- Blood pressure: hypotension
- Pulse: increased, dysrhythmias
- Respirations: dyspnea, stridor, wheezes, laryngospasm, bronchospasm, pulmonary edema
- Skin: warm, edematous (lips, eyelids, tongue, hands, feet, genitals)
- Mental status: restless, anxious, lethargic to comatose
- Urine output: oliguria to anuria
- Other: paresthesias; pruritus; abdominal cramps, vomiting, diarrhea

oxygen ($Pa_{O_2}$) and in total oxygen saturation, and an increase in the partial pressure of carbon dioxide ($Pa_{CO_2}$).
- *Serum electrolytes,* to monitor the severity and progression of shock. As shock progresses, glucose levels decrease, sodium levels decrease, and potassium levels increase.
- *Blood urea nitrogen (BUN), serum creatinine levels, urine specific gravity,* and *osmolality,* to check renal function. As perfusion of the kidneys is decreased and renal function is reduced, the BUN and creatinine levels increase as does urine specific gravity and osmolality.
- *Blood cultures,* to identify the causative organism in septic shock.
- *White blood cell (WBC) count* and *differential,* in the client with septic or anaphylactic shock. The total WBC count is increased in septic shock. Elevated neutrophils indicate acute infection, increased monocytes indicate a bacterial infection, and increased eosinophils indicate an allergic response.
- *Serum cardiac enzymes,* which are elevated in cardiogenic shock: lactate dehydrogenase (LDH), creatine phosphokinase (CPK), and serum glutamic-oxaloacetic transaminase (SGOT).
- *Central venous catheter,* to aid in the differential diagnosis of shock and to provide information about the preload of the heart. A pulmonary artery catheter may be inserted to monitor cardiac dynamics, fluid balance, and the effects of vasoactive medications.

Other diagnostic tests may be ordered to determine the extent of injury or damage or to locate the site of internal hemorrhage. These tests might include x-ray studies, CT scans, MRI, endoscopic examinations, and echocardiograms. Newer diagnostic methods for hypoperfusion include gastric tonometry and sublingual $Pa_{CO_2}$. Gastric tonometry measures the partial pressure of carbon dioxide in the gastric lumen. The measurement of sublingual carbon dioxide correlates well with decreased MAP (Sole et al., 2001).

## Medications

When fluid replacement alone is not sufficient to reverse shock, vasoactive drugs (drugs causing vasoconstriction or vasodilatation) and inotropic drugs (drugs improving cardiac contractility) may be administered. When used to treat shock, these drugs increase venous return through vasoconstriction of peripheral vessels; they also improve the pumping ability of the heart by facilitating myocardial contractility and by dilating coronary arteries to increase perfusion of the myocardium.

Drugs used to treat shock are discussed in the Medication Administration box on page 278. Other drugs that may be administered to the client in shock include:
- Diuretics to increase urine output after fluid replacement has been initiated
- Sodium bicarbonate to treat acidosis
- Calcium to replace calcium lost as a result of blood transfusions
- Antiarrhythmic agents to stabilize heart rhythm
- Broad-spectrum antibiotics to suppress organisms responsible for septic shock
- A cardiotonic glycoside (such as digitalis) to treat cardiac failure
- Corticosteroids to treat anaphylactic shock
- Morphine to dilate veins and decrease anxiety.

## Oxygen Therapy

Establishing and maintaining a patent airway and ensuring adequate oxygenation are critical interventions in reversing shock. All clients in shock (even those with adequate respirations) should receive oxygen therapy (usually by mask or nasal cannula) to maintain the $Pa_{O_2}$ at greater than 80 mmHg during the first 4 to 6 hours of care. If the client's unassisted respiration cannot maintain $Pa_{O_2}$ at this level, ventilatory assistance may be necessary. Care of the client requiring ventilatory assistance is discussed in Chapter 38 ∞.

## Fluid Replacement

The most effective treatment for the client in hypovolemic shock is the administration of intravenous fluids or blood. Fluids also treat septic and neurogenic shock. However, the client with cardiogenic shock may require either fluid replacement or restriction, depending on pulmonary artery pressure.

Various fluids may be administered alone or in combination as part of fluid replacement therapy in treating shock. Whole blood or blood products increase the oxygen-carrying capacity of the blood and thus increase oxygenation of cells. Fluid replacements, such as crystalloid and colloid solutions, increase circulating blood volume and tissue perfusion. Fluid replacements are administered in massive amounts through two large-bore peripheral lines or through a central line.

***CRYSTALLOID SOLUTIONS*** Crystalloid solutions contain dextrose or electrolytes dissolved in water; they are either isotonic or hypotonic. Isotonic solutions include normal saline (0.9%), lactated Ringer's solution, and Ringer's solution. Hypotonic solutions include one-half normal saline (0.45%) and 5% dextrose in water ($D_5W$).

All crystalloid solutions increase fluid volume in both the intravascular and the interstitial space. Of the total amount infused, only about 25% remains in the intravascular system; the remaining 75% moves into the interstitial space. Consequently, fluid volume is only minimally expanded and the potential for peripheral edema is increased when crystalloid solutions are used. However, Ringer's lactate (an electrolyte solution) and 0.9% saline are the fluids of choice in treating hypovolemic shock, especially in the emergency phase of care while blood is being typed and crossmatched. Large amounts of these solutions may be infused rapidly, increasing blood volume and tissue perfusion.

***COLLOID SOLUTIONS*** Colloid solutions contain substances (colloids) that should not diffuse through capillary walls. Hence, colloids tend to remain in the vascular system and increase the osmotic pressure of the serum, causing fluid to move into the vascular compartment from the interstitial space. As a result, plasma volume expands. Colloid solutions used to treat shock include 5% albumin, 25% albumin, hetastarch, plasma protein fraction, and dextran.

## MEDICATION ADMINISTRATION The Client in Shock

### ADRENERGICS (SYMPATHOMIMETICS)

**Vasoconstrictors**
- **Norepinephrine (Levophed)**
- **Metaraminol (Aramine)**

**Inotropes**
- **Dopamine (Inotropin)***
- **Dobutamine (Dobutrex)**
- **Isoproterenol (Isuprel)**

Adrenergic drugs (also called sympathomimetics) mimic the fight-or-flight response of the sympathetic nervous system, selectively stimulating alpha-adrenergic and beta-adrenergic receptors. Many of these drugs have both vasopressor (vasoconstricting) effects and positive inotropic effects (Table 11–6). Stimulation of alpha-adrenergic receptors results in vasoconstriction and increased systemic blood pressure. Stimulation of beta-adrenergic receptors increases the force and rate of myocardial contraction.

The physiologic effect of these drugs includes improved perfusion and oxygenation of the heart, with increased stroke volume and heart rate, and increased cardiac output. Increased cardiac output in turn increases tissue perfusion and oxygenation. The major disadvantage is that increases in stroke volume and heart rate also increase the oxygen requirements of the myocardium. These drugs may be used in the early stages of shock, especially in types of shock characterized by vasodilation.

#### Nursing Responsibilities

- Carefully monitor responses in the older adult, who may be especially sensitive to sympathomimetics and require lower doses.
- When administering these drugs by the subcutaneous route, carefully aspirate the injection site to avoid injecting the drug directly into a blood vessel.
- Use the intravenous route only with continuous infusion pumps. Carefully adjust the dose to accommodate the client's cardiovascular status (as ordered by the physician or by written protocol).
- Document lung sounds, vital signs, and hemodynamic parameters before starting the medication, and then according to institutional policy (usually every 5 to 15 minutes).

TABLE 11–6 **Adrenergic Drugs Used to Treat Shock**

| ACTION | DRUG | RECEPTOR |
|---|---|---|
| Vasoconstrictors | Norepinephrine (Levophed) | A |
| | Metaraminol (Aramine) | A |
| Inotropes | Dopamine (Inotropin)* | A, $B^1$ |
| | Dobutamine (Dobutrex) | $B^1$ |
| | Isoproterenol (Isuprel) | $B^1$, $B^2$ |

**Receptors are dose dependent.*

- Record and monitor urine output. Report output of less than 30 mL/h.
- Be aware that the sympathomimetics are incompatible with sodium bicarbonate or alkaline solutions.
- When administering drugs that cause vasoconstriction, such as norepinephrine (Levophed) and metaraminol (Aramine), monitor the intravenous insertion site for infiltration. If infiltration does occur, stop the infusion and notify the physician immediately. (Infiltration may cause ischemia and necrosis of tissue.)

#### Health Education for the Client and Family

- Because these drugs mimic a physiologic reaction to stress, they may cause feelings of anxiety.
- Close monitoring to adjust the dose will be carried out by qualified nurses using written protocols.
- Report heart palpitations or chest pain immediately.

### VASODILATORS

**Nitroglycerin (Tridil)**
**Nitroprusside (Nipride)**

Drugs that cause vasodilation act directly on smooth muscle, affecting both arterioles and veins. Peripheral resistance, cardiac output, and pulmonary wedge pressure are all reduced as a result of the vasodilation. These effects decrease the oxygen need of the heart and decrease pulmonary congestion. Vasodilators are used primarily in the treatment of cardiogenic shock and may be combined with a sympathomimetic (e.g., dopamine).

#### Nursing Responsibilities

- Protect these drugs from light by wrapping the intravenous bag in the package that is provided.
- Mix with $D_5W$ only.
- Infuse with an infusion pump, and use within 4 hours of reconstitution.
- Do not add other medications to the solution.
- Assess mental status, blood pressure, and pulse prior to initiating medication. Thereafter, assess blood pressure and pulse according to institutional policy (usually every 5 minutes initially, then every 15 minutes until stable, and then every hour).
- Monitor for confusion, dizziness, tachycardia, arrhythmias, hypotension, and adventitious breath sounds. Report these immediately if they occur, and slow infusion to a keep-open rate.
- Monitor for signs of thiocyanate poisoning (nausea, disorientation, muscle spasms, decreased or absent reflexes) if infusion lasts longer than 72 hours.
- Keep client in bed with side rails up.

#### Health Education for the Client and Family

- It is important to stay in bed and change positions slowly to avoid dizziness.
- The blood pressure and pulse are taken frequently to adjust the dose of medication.
- Headache is a common side effect.

Colloid products reduce platelet adhesiveness and have been associated with reductions in blood coagulation. Consequently, the client's prothrombin time (PT), International Normalized Ratio (INR), platelet count, and activated partial thromboplastin time (APTT) should be monitored when these solutions are administered. Normal values are as follows:

| | |
|---|---|
| PT | 10–15 seconds |
| INR | 1–1.2 seconds |
| Platelets | 150,000–400,000 |
| APTT | <35 seconds |

MEDICATION ADMINISTRATION **Colloid Solutions**

**COLLOID SOLUTIONS (PLASMA EXPANDERS)**
**Albumin 5% (Albuminar-5, Buminate 5%)**
**Albumin 25% (Albuminar-25, Buminate 25%)**
**Dextran 40 (Gentran 40)**
**Dextran 70 (Gentran 70, Macrodex)**
**Dextran 75 (Gentran 75)**
**Hetastarch (Hespan [HES])**
**Plasma protein fraction (Plasmanate, Plasma-Plex, Plasmatein, Protenate)**

These solutions are blood volume expanders and are used to treat hypovolemic shock due to surgery, hemorrhage, burns, or other trauma. Albumin and plasma protein fraction are prepared from healthy blood donors. Dextran and hetastarch are synthetically prepared large molecules. The solutions promote circulatory volume and tissue perfusion by rapidly expanding plasma volume. Dextran solutions are infrequently used.

**Nursing Responsibilities**

- Before infusion begins, establish baseline of vital signs, lung sounds, heart sounds, and (if possible) CVP and pulmonary artery wedge pressure.
- Start administration of ordered intravenous fluids, using a large-gauge (18- or 19-gauge) infusion needle.
- Take and record vital signs as required by institutional policy (usually every 15 to 60 minutes) and client status.
- Take and record intake and output every 1 to 2 hours.
- Monitor for manifestations of congestive heart failure or pulmonary edema (dyspnea, cyanosis, cough, crackles, wheezes). If these manifestations appear, stop the fluids and notify the physician immediately.
- Monitor for bleeding from new sites; an increase in blood pressure may cause bleeding in severed vessels that did not bleed with decreased blood pressure.
- Monitor for manifestations of dehydration (dry lips; scant, dark-colored urine; loss of skin turgor). Increased intravenous fluids are usually ordered if the client becomes dehydrated.
- Monitor for manifestations of circulatory overload (jugular vein distention, increase in CVP, increase in pulmonary artery wedge pressure). If these manifestations occur, slow rate of infusion and notify physician.
- Monitor prothrombin time, partial thromboplastin time, and platelet counts.
- If administering dextran or plasma protein fraction, have epinephrine and antihistamines readily available for any manifestations of a hypersensitivity reaction (fever, chills, rash, headache, wheezing, flushing).
- Maintain client on bed rest with side rails elevated.

**Health Education for the Client and Family**

- The solutions are given to replace lost serum protein, which helps maintain the volume of blood.
- The vital signs are taken frequently to ensure the safety of the client.

See the Medication Administration box above for further information about colloid solutions and associated nursing responsibilities and client teaching.

***BLOOD AND BLOOD PRODUCTS*** If hypovolemic shock is due to hemorrhage, the infusion of blood and blood products may be indicated. The goal of blood administration is to keep the hematocrit at 30% to 35% and the hemoglobin level between 12.5 and 14.5 g/100 mL. Available blood and blood products include fresh whole blood, stored whole blood, packed RBCs, platelet concentrate, fresh-frozen plasma, and cryoprecipitate. Often, packed RBCs are given to provide hemoglobin concentration and are supplemented with crystalloids to maintain an adequate circulatory volume. (See discussion of blood administration earlier in the chapter.)

## NURSING CARE

Nursing assessments and interventions to prevent shock are an essential part of the nursing care of every client. The primary nursing interventions to prevent shock are assessment and monitoring.

### Health Promotion and Assessment

Nursing assessments are critical in preventing shock. Identifying clients at risk and making focused assessments are essential. Although shock may occur at any age, physiologic changes with aging make the older adult a high-risk population. (See the Nursing Care of the Older Adult box on page 280.)

- *Hypovolemic shock:* Clients who have undergone surgery, have sustained multiple traumatic injuries, or have been seriously burned are most likely to develop hypovolemic shock. Monitoring fluid status is essential in preventing shock and includes daily assessments of weight, fluid intake by all routes, measurable fluid loss (e.g., urine, vomitus, wound drainage, gastric drainage, and chest tube drainage), and fluid loss that must be estimated, such as fluid lost via profuse perspiration and wound drainage. Assessments for the critically ill client are ongoing and include fluid balance, hemodynamic values, and vital signs.
- *Cardiogenic shock:* Clients with left anterior wall myocardial infarctions are at risk for developing cardiogenic shock. Nursing care to prevent the development of cardiogenic shock focuses on maintaining or improving myocardial oxygen supply by providing immediate pain relief, maintaining rest, and administering supplemental oxygen.
- *Neurogenic shock:* The risk of neurogenic shock is increased in clients who have spinal cord injuries and those who have received spinal anesthesia. Preventive nursing care includes maintaining immobility of clients with spinal cord trauma and elevating the head of the bed 15 to 20 degrees following spinal anesthesia. Elevations of more than 20 degrees, however, can potentiate headaches following spinal anesthesia and should be avoided.
- *Anaphylactic shock:* Prevent anaphylactic shock by collecting information about allergies and drug reactions during the

### NURSING CARE OF THE OLDER ADULT — Variations In Assessment Findings—Shock

- Cardiac changes may include a thickened left ventricular wall, decreased elasticity of the myocardium, and more rigid valves. These changes result in a decreased stroke volume and cardiac output, thus decreasing responses to shock in general and increasing the risk of cardiogenic shock.
- Decreased arterial wall elasticity and vasomotor tone reduce the older adult's ability to respond to a decrease in oxygenation.
- Decreased elasticity and turgor of the skin make assessments of skin turgor more difficult.
- Previous medication and blood administration increase the risk of anaphylactic shock.
- Decreased immune system response increases the risk of septic shock.

health history. Note these allergies clearly on all documents and place a special armband on the client. Careful and frequent assessments during blood administration may prevent serious reactions to blood or blood products.

- *Septic shock:* Clients who are hospitalized, are debilitated, are chronically ill, or have undergone invasive procedures or tube insertions are at high risk for septic shock. Nursing care to prevent septic shock includes careful and consistent hand washing, the use of aseptic techniques for procedures (e.g., catheterizations, suctioning, changing dressings, starting and maintaining intravenous fluids or medications), and monitoring for local and systemic manifestations (e.g., WBC and differential counts) of infection.

## Nursing Diagnoses and Interventions

Nursing care for the client in shock focuses on assessing and monitoring overall tissue perfusion and on meeting psychosocial needs of the client and the family. This section discusses nursing diagnoses that are appropriate for the client with hypovolemic shock. See accompanying Nursing Care Plan on the following page.

### Decreased Cardiac Output

Decreased cardiac output is the primary problem for the client in shock. Although much of the care related to this diagnosis is collaborative, many independent nursing interventions are critical to the care of the client in shock.

### NURSING RESEARCH — Evidence-Based Practice for Care of ICU Clients Sustaining Multiple Trauma

Ventilator-associated pneumonia (VAP) is an important client safety issue in critically injured clients. The purpose of this study (Dodek, 2005) was to develop an evidence-based guideline for prevention of ventilator-assisted pneumonia.

Data extraction consisted of gathering physical, positional, and pharmacologic interventions that may influence the development of VAP. The authors searched for pertinent randomized trials and case reviews that involved clients on mechanical ventilation. The study was isolated to include only adults, and only studies published before April 1, 2003, were considered.

Physical strategies included:

- Route of endotracheal intubations
- Systematic search for maxillary sinusitis
- Frequency of ventilator circuit changes
- Airway humidification
- Endotracheal suctioning system
- Sublette secretion drainage
- Chest physiology
- Timing of tracheotomy.

Positional strategies included:

- Kinetic bed therapy
- Semirecumbent positioning
- Prone positioning.

Pharmacologic strategies included:

- Stress ulcer prophylaxis
- Prophylactic antibiotics, including selective decontamination of the digestive track.

**IMPLICATIONS FOR NURSING**

The following section identifies pertinent findings to support the conclusion that effectively implemented guidelines may decrease the morbidity, mortality, and costs of VAP in mechanically ventilated clients.

Based on direct evidence the following recommendations are encouraged:

- Orotracheal intubation is the route of choice when intubation is necessary.
- Recommend that new circuits for each client be instituted and changed if the circuits become soiled.
- Weekly changes of heat and moisture exchangers on equipment providing mechanical ventilation.
- Recommend the use of closed endotracheal suction systems that are changed for each new client and as clinically indicated.
- Recommend that clinicians consider the use of subglottic secretion drainage.
- Recommend that clinicians consider the use of kinetic beds.
- Recommend the use of semirecumbent positioning, with a goal of 45 degrees, in clients without contraindications.

The pharmacologic strategies that were studied were unfounded and not recommended for the VAP guideline.

**CRITICAL THINKING IN CLIENT CARE**

1. Considering the information from this study, how would you communicate the recommendations to the medical staff for the client with mechanical ventilation?
2. What is the rationale behind the above recommendations?

## NURSING CARE PLAN A Client with Septic Shock

Huang Mei Lan is a 43-year-old unmarried female who lives alone in a major West Coast city. Ms. Huang came to America 15 years ago from China and now speaks English well. Her family still lives in China. She worked in a neighborhood sewing shop until 3 years ago, when she was diagnosed with breast cancer. Her treatment included mastectomy of the affected breast and follow-up chemotherapy.

Last month, Ms. Huang experienced a recurrence of cancer in the lymph glands of the affected side. Surgery to remove the glands was performed and chemotherapy started. Ms. Huang has a central line, a urinary catheter, and a surgical incision. She is underweight, weak, and depressed. Although she has multiple physical problems, she never complains or asks for any kind of medication.

### ASSESSMENT

Ms. Huang's primary nurse, Robert O'Brien, enters her room early in the morning to make an initial assessment. He finds Ms. Huang huddled in the middle of the bed, shivering violently. Her vital signs are T 104°F, P 110, R 30, and BP 106/66. Her skin is hot, dry, and flushed with poor turgor. She is alert and oriented, but is restless and appears anxious. Ms. Huang states she is nauseated and suddenly begins vomiting and is incontinent of liquid stool. Laboratory data indicate leukocytosis, respiratory alkalosis, and reduced platelet count. Blood cultures, as well as cultures of Ms. Huang's sputum, urine, and wound drainage, are conducted. She is diagnosed as having septic shock.

Hetastarch is ordered per intravenous line, and intravenous broad-spectrum antibiotics are begun until the organism and its portal of entry can be determined. Despite treatment, Ms. Huang's condition worsens. Her blood pressure continues to drop, her skin becomes cool and cyanotic, and she begins to have periods of disorientation. She is transferred to the critical care unit. As she is being prepared for the transfer, she begins to cry and asks, "Am I going to die?"

### DIAGNOSES

- *Deficient Fluid Volume* related to vomiting, diarrhea, high fever, and shift of intravascular volume to interstitial spaces
- *Ineffective Breathing Pattern* related to rapid respirations and progression of septic shock
- *Ineffective Tissue Perfusion* related to progression of septic shock with decreased cardiac output, hypotension, and massive vasodilatation
- *Anxiety* related to feelings that illness is worsening and is potentially life threatening, and the transfer to the critical care unit

### EXPECTED OUTCOMES

- Maintain adequate circulating blood volume.
- Regain and maintain blood gas parameters within normal limits.
- Regain and maintain stable hemodynamic levels.
- Verbalize increased ability to cope with stressors.

### PLANNING AND IMPLEMENTATION

- Monitor neurologic status, including mental status and level of consciousness.
- Monitor cardiovascular status, including arterial blood pressure; rate, rhythm, and quality of pulses; central venous pressure; pulmonary artery pressure; and cardiac output.
- Monitor color and character of skin.
- Monitor results of ABGS, blood counts, clotting times, and platelet counts.
- Monitor respiratory status, including respiratory rate, rhythm, and breath sounds.
- Monitor body temperature every 2 hours.
- Monitor urinary output hourly, reporting any output of <30 mL/h.
- Explain procedures and provide comfort measures (oral care, skin care, turning, positioning).

### EVALUATION

Despite intensive nursing and medical care, Ms. Huang's condition remains critical. The interventions are continued.

### CRITICAL THINKING IN THE NURSING PROCESS

1. Vasopressors may be used in the treatment of septic shock. Explain the rationale for their use.
2. While monitoring Ms. Huang's ABGS, the nurse notes that her $Pa_{O_2}$ is <60 mmHg and her $Pa_{CO_2}$ is >50. What do these findings indicate, and why have they occurred?
3. Ms. Huang has been given large amounts of colloids intravenously. Hemodynamic monitoring indicates a higher than normal CVP and pulmonary artery pressure. What do these findings indicate? What physical assessments would you make to confirm the changes?

*See Evaluating Your Response in Appendix C.*

- Assess and monitor cardiovascular function via the following:
  - Blood pressure
  - Heart rate and rhythm
  - Pulse oximetry
  - Peripheral pulses
  - Hemodynamic monitoring of arterial pressures, pulmonary artery pressures, and central venous pressures (CVPs).

  *A baseline assessment is necessary to establish the stage of shock. If palpable peripheral pulses and audible (to auscultation) blood pressure are lost, inserting central arterial, venous, and pulmonary artery catheters is essential to establish progression of shock accurately and to evaluate the client's response to therapy.*
- Measure and record intake and output (total output and urinary output) hourly. *A decrease in circulating blood volume with hypotension and the effect of the compensatory mechanisms associated with shock can cause renal failure. Urinary output of <30 mL/h in an acutely ill adult indicates reduced renal blood flow.*
- Monitor bowel sounds, abdominal distention, and abdominal pain. *Decreased splanchnic blood flow reduces bowel motility and peristalsis; paralytic ileus may result.*
- Monitor for sudden sharp chest pain, dyspnea, cyanosis, anxiety, and restlessness. *Hemoconcentration and increased platelet aggregation may result in pulmonary emboli.*
- Maintain bed rest and provide (to the extent possible) a calm, quiet environment. Place in a supine position with the legs

elevated to about 20 degrees, trunk flat, and head and shoulders elevated higher than the chest (about 10 degrees) (Figure 11–9 ■). *Limiting activity and ensuring rest decreases the workload of the heart. The supine position with legs elevated increases venous return; however, this position should not be used for clients in cardiogenic shock. The Trendelenburg position is no longer recommended, because it causes the abdominal organs to press against the diaphragm (limiting respirations), decreases filling of the coronary arteries, and initiates aortic and carotid sinus reflexes.*

## Ineffective Tissue Perfusion

As shock progresses, diminished tissue perfusion causes ischemia and hypoxia of major organ systems. As shock worsens, blood flow and oxygenation of the lungs, heart, and brain are also impaired. Hypoxia and ischemia result from decreased tissue perfusion in the kidneys, brain, heart, lungs, gastrointestinal tract, and the periphery.

- Monitor skin color, temperature, turgor, and moisture. *Decreased tissue perfusion is evidenced by the skin becoming pale, cool, and moist; as hemoglobin concentrations decrease, cyanosis occurs.*
- Monitor cardiopulmonary function by assessing/monitoring the following:
  - Blood pressure (by auscultation or by hemodynamic monitoring)
  - Rate and depth of respirations
  - Lung sounds
  - Pulse oximetry
  - Peripheral pulses (brachial, radial, dorsalis pedis, and posterior tibial); include presence, equality, rate, rhythm, and quality (If unable to palpate pulses, use a device such as a Doppler ultrasound flowmeter to assess peripheral arterial blood flow.)
  - Jugular vein distention
  - CVP measurements.

  *Baseline vital signs are necessary to determine trends in subsequent findings. As shock progresses, the blood pressure decreases, and the pulse becomes rapid, weak, and thready. As perfusion of the lungs decreases, crackles, wheezes, and dyspnea are commonly assessed. Capillary refill is prolonged, and peripheral pulses are weak or nonpalpable. Neck veins that cannot be seen when the client is in the supine position indicate decreased intravascular volume. CVP is an accurate means of determining fluid status in the client in shock; the findings will be low (5 to 15 cm of water is normal) in hypovolemic shock because of the decreased blood volume.* (See Chapter 10 ∞ for a discussion of CVP.)
- Monitor body temperature. *An elevated body temperature increases metabolic demands, depleting reserves of bodily energy. It also increases myocardial oxygen demand and may place the client with previous cardiac problems at even greater risk for hypoperfusion.*
- Monitor urinary output per Foley catheter hourly, using a urometer. *Urine output is a reliable indicator of renal perfusion.*
- Assess mental status and level of consciousness. *The appropriateness of the client's behavior and responses reflects the adequacy of cerebral circulation. Restlessness and anxiety are common early in shock; in later stages, the client may become lethargic and progress to a comatose state. Altered levels of consciousness are the result of both cerebral hypoxia and the effects of acidosis on brain cells.*

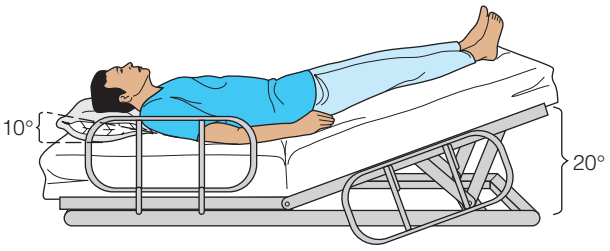



**Figure 11–9** ■ The client in shock should be positioned with the lower extremities elevated approximately 20 degrees (knees straight), trunk horizontal, and the head elevated about 10 degrees.

## Anxiety

Many clients in hypovolemic shock have experienced some form of major trauma and may have life-threatening, multiple injuries. Following on-the-scene treatment, the client is usually admitted to the healthcare setting through the emergency department. Surgery may be required to treat injuries, followed by care in a critical care unit. Throughout this sequence of crisis events, treatment is invasive, and contact with family is minimal. Client and family responses to these situations of uncertainty, instability, and change include anxiety, fear, and powerlessness. These responses are affected by age, developmental level, cultural and ethnic group, experience with illness and the healthcare system, and support systems.

- Assess the cause(s) of the anxiety, and manipulate the environment to provide periods of rest. *Reducing stimuli that cause anxiety is calming and facilitates rest, which is necessary in the client at risk for bleeding.*
- Administer prescribed pain medications on a regular basis. *Pain precipitates and/or aggravates anxiety.*
- Provide interventions to increase comfort and reduce restlessness:
  - Maintain a clean environment.
  - Provide skin and oral care.
  - Monitor the effectiveness of ventilation or oxygen therapy.
  - Eliminate all nonessential activities.
  - Remain with the client during procedures.
  - Speak slowly and calmly, using short sentences.
  - Use touch to provide support.

  *Unfamiliar sounds, sights, and odors can increase anxiety. Damp skin or a dry mouth increases discomfort. Inadequate gas exchange with a decrease in oxygen or an increase in carbon dioxide in the blood may cause the client to experience a "feeling of doom." Activity increases the body's need for oxygen. Listening and touch provide support in an environment in which the client often feels alone and abandoned. Severe anxiety interferes with the ability to understand others and to respond appropriately.*
- Provide support for the client and family:
  - Provide time, space, and privacy for family members.

- Allow family members access to the client when feasible.
- Encourage the expression of feelings and concerns. Provide anticipatory guidance to prepare for recovery or death and to support realistic hope.
- Acknowledge the beliefs, values, and expectations of the client and family.

*Allowing the family access to the client reduces anxiety and gives both the client and the family some feeling of control. If prognosis is poor, access and involvement allow the family to begin the grieving process. If recovery is expected, contact provides the client and family with a feeling of hope. Supporting the client and family facilitates concrete problem solving, promotes acceptance of the illness and its implications, and helps them begin to establish ways of managing the illness experience.*

- Provide information about the current setting to both the client and family; give the family information about available resources (such as pastoral care, social services, temporary housing, meals). *Knowing what to expect and how to control the environment to meet basic needs reduces anxiety.*

## Using NANDA, NIC, and NOC

Chart 11–2 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client who is experiencing shock.

## Community-Based Care

Home care for the client who has experienced shock is highly individualized, depending on the cause and the illness or injury that caused shock. Therefore, topics for consideration are not included in this section.

NANDA, NIC, AND NOC LINKAGES

CHART 11–2 **The Client in Shock**

**NANDA**

Ineffective Tissue Perfusion: Cerebral

↓

**NIC**

Acid-Base Monitoring

Cerebral Perfusion Monitoring

Fluid/Electrolyte Management

Hypovolemia Management

Shock Management

↓

**NOC**

Tissue Perfusion: Cerebral

Circulation Status

Vital Signs

Neurologic Status

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

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## CHAPTER HIGHLIGHTS

- Traumatic injuries affect human tissues and organs resulting from a transfer of energy from the environment. Energy sources can be mechanical, gravitational, thermal, electrical, physical, or chemical.
- Trauma can be categorized as minor trauma, which causes minimal damage to underlying tissues, or major trauma, which can involve a serious single system injury or multiple trauma. Trauma is further categorized into blunt and penetrating trauma. Blunt trauma is caused by various forces such as deceleration, acceleration, shearing, compression, or crushing. Penetrating trauma occurs when a foreign object enters the body.
- Airway assessment is the highest priority in the trauma patient and supersedes all other interventions.
- During the primary survey the nurse identifies all life-threatening injuries and performs appropriate interventions. In the secondary survey, the nurse identifies all injuries in order to prioritize care.
- Shock is a clinical condition in which there is an imbalance between oxygen supply and demand. This imbalance results in inadequate blood flow to organs and tissues causing life-threatening cellular dysfunction.
- An early sign of shock is a change in the level of consciousness with restlessness being a common symptom of cerebral hypoxia.
- There are three stages of shock: compensatory (stage I) which is an early and reversible stage; progressive (stage II) which is when fluid loss is 35–50% and affected cells switch from aerobic to anerobic metabolism in order to remain alive; and the final stage is irreversible (stage III) in which tissue anoxia and cellular death become so widespread that no treatment can reverse the damage.
- Hypovolemic shock is the most common type of shock and is caused by a decrease in the circulating blood volume by 15% or greater. Treatment consists of stopping the bleeding, getting oxygen to the cellular level, and restoring blood volume. This is accomplished by providing high-flow oxygen to the patient, surgical intervention, and transfusion of blood products.
- Cardiogenic shock is caused when the pumping ability of the heart is compromised to the point that the heart cannot maintain adequate cardiac output and tissue perfusion.
- Septic shock is a part of a progressive syndrome called systemic inflammatory response syndrome (SIRS), a condition most often caused by gram–negative infections.
- Anaphylactic shock results in vasodilatation, pooling of blood in the periphery and hypovolemia which leads to altered cellular metabolism. This condition is the result of a fulminating hypersensitivity reaction to a foreign substance.

## TEST YOURSELF NCLEX-RN® REVIEW

1 What is the most common mechanical source of injury in adults of all ages?
   **1.** gunshot wounds
   **2.** fire
   **3.** drowning
   **4.** motor vehicles

2 Severe facial injuries, such as those resulting from going through a windshield, increase the risk for all of the following. Which would you assess first?
   **1.** airway obstruction
   **2.** hemorrhage
   **3.** contusions
   **4.** fractures

3 Which on-the-scene intervention would be a priority?
   **1.** Determine cause of injury.
   **2.** Assess airway patency.
   **3.** Assess peripheral capillary refill.
   **4.** Palpate for internal hemorrhage.

4 You are monitoring blood administration to a trauma victim in shock. Which of the following assessments indicate a dangerous transfusion reaction?
   **1.** red raised areas (wheals) on the skin that itch
   **2.** an increase in body temperature by 3°
   **3.** decreasing blood pressure and dyspnea
   **4.** increasing blood pressure and pulse

5 What type of shock causes widespread vasodilatation and decreased peripheral resistance?
   **1.** cardiogenic shock
   **2.** septic shock
   **3.** hypovolemic shock
   **4.** obstructive shock

6 What is the best method to manage uncontrolled bleeding?
   **1.** apply direct pressure
   **2.** clamp a visible vessel
   **3.** apply a tourniquet
   **4.** elevate the injured part

7 Trauma is defined as:
   **1.** injury to human tissues from the transfer of energy.
   **2.** result of random chance.
   **3.** accidental injury.
   **4.** an intentional injury.

8 Which of the following is not a risk from a blood transfusion?
   **1.** AIDS
   **2.** West Nile Virus
   **3.** transfusion reaction
   **4.** weight gain

9 Distributive shock is caused by:
   **1.** blood loss.
   **2.** widespread vasodilatation.
   **3.** ineffective cardiac pumping action.
   **4.** hypersensitivity reaction.

10 Shock is defined as:
   **1.** a systemic imbalance between oxygen supply and demand.
   **2.** sufficient cardiac output.
   **3.** hemorrhage.
   **4.** abnormal blood pressure.

*See Test Yourself answers in Appendix C.*

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