chapter 15

Shock and Multiple Organ Dysfunction Syndrome

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Describe shock and its underlying pathophysiology.
2. Compare clinical findings of the compensatory, progressive, and irreversible stages of shock.
3. Describe organ dysfunction that may occur with shock.
4. Describe similarities and differences in shock due to hypovolemic, cardiogenic, neurogenic, anaphylactic, and septic shock states.
5. Identify medical and nursing management priorities in treating patients in shock.
6. Identify vasoactive medications used in treating shock, and describe nursing implications associated with their use.
7. Discuss the importance of nutritional support in all forms of shock.
8. Discuss the role of nurses in psychosocial support of patients experiencing shock and their families.
9. Discuss multiple organ dysfunction syndrome.

GLOSSARY

anaphylactic shock: circulatory shock state resulting from a severe allergic reaction producing an overwhelming systemic vasodilation and relative hypovolemia

biochemical mediators: messenger substances that may be released by a cell to create an action at that site or be carried by the bloodstream to a distant site before being activated; also called cytokines

cardiogenic shock: shock state resulting from impairment or failure of the myocardium

circulatory shock: shock state resulting from displacement of blood volume creating a relative hypovolemia and inadequate delivery of oxygen to the cells; also called distributive shock

colloids: intravenous solutions that contain molecules that are too large to pass through capillary membranes

crystalloids: intravenous electrolyte solutions that move freely between the intravascular compartment and interstitial spaces

hypovolemic shock: shock state resulting from decreased intravascular volume due to fluid loss

multiple organ dysfunction syndrome: presence of altered function of two or more organs in an acutely ill patient such that interventions are necessary to support continued organ function

neurogenic shock: shock state resulting from loss of sympathetic tone causing relative hypovolemia

septic shock: circulatory shock state resulting from overwhelming infection causing relative hypovolemia

shock: physiologic state in which there is inadequate blood flow to tissues and cells of the body

systemic inflammatory response syndrome: overwhelming inflammatory response in the absence of infection causing relative hypovolemia and decreased tissue perfusion

Shock is a life-threatening condition with a variety of underlying causes. It is characterized by inadequate perfusion that, if untreated, results in cell death. The progression of shock is neither linear nor predictable, and shock states, especially septic shock, comprise a current area of aggressive clinical research. Nurses caring for patients with shock and for those at risk for shock must understand the underlying mechanisms of shock and recognize its subtle as well as more obvious signs. Rapid assessment with early recognition and response to shock states is essential to the patient's recovery.

Overview of Shock

Shock can best be defined as a condition in which widespread perfusion to the cells is inadequate to deliver oxygen and nutrients to support vital organs and cellular function (VonRueden, Bolton & Vary, 2008). Adequate blood flow to the tissues and cells requires an adequate cardiac pump, effective vasculature or circulatory system, and sufficient blood volume. If one of these components is impaired, perfusion to the tissues is threatened or compromised. Without treatment, inadequate blood flow to the cells results in poor delivery of oxygen and nutrients, cellular hypoxia, and cell death that progresses to organ dysfunction and eventually death.

Shock affects all body systems. It may develop rapidly or slowly, depending on the underlying cause. During shock, the body struggles to survive, calling on all its homeostatic mechanisms to restore blood flow. Any insult to the body can create a cascade of events resulting in poor tissue perfusion. Therefore, almost any patient with any disease state may be at risk for developing shock. Conventionally, the primary underlying pathophysiologic process and underlying disorder are used to classify the shock state (eg, hypovolemic shock, cardiogenic shock, and circulatory shock [all discussed later in the chapter]).

Regardless of the initial cause of shock, certain physiologic responses are common to all types of shock. These physiologic responses include hypoperfusion of tissues, hypermetabolism, and activation of the inflammatory response. The body responds to shock states by activating the sympathetic nervous system and mounting a hypermetabolic and inflammatory response. Once shock develops, the patient's survival may have more to do with the body's ability to effectively respond to it than with the initial cause of shock. Failure of compensatory mechanisms to effectively restore physiologic balance is the final pathway of all shock states and results in end-organ dysfunction and death (Cocchi, Kimlin, Walsh, et al., 2007; Dellinger, Levy, Carlet, et al., 2008; King, 2007; VonRueden, et al., 2008).

Nursing care of patients with shock requires ongoing systematic assessment. Many of the interventions required in caring for patients with shock call for close collaboration with other members of the health care team and rapid implementation of prescribed therapies. Nurses must anticipate these therapies because they need to be implemented with speed and accuracy.

Normal Cellular Function

Energy metabolism occurs within the cell, where nutrients are chemically broken down and stored in the form of adenosine triphosphate (ATP). Cells use this stored energy to perform necessary functions, such as active transport, muscle contraction, and biochemical synthesis, as well as specialized cellular functions, such as the conduction of electrical impulses. ATP can be synthesized aerobically (in the presence of oxygen) or anaerobically (in the absence of oxygen). Aerobic metabolism yields far greater amounts of ATP per mole of glucose than does anaerobic metabolism; therefore, it is a more efficient and effective means of producing energy. In addition, anaerobic metabolism results in the accumulation of the toxic end product, lactic acid, which must be removed from the cell and transported to the liver for conversion into glucose and glycogen.

Pathophysiology

Cellular Changes

In shock, the cells lack an adequate blood supply and are deprived of oxygen and nutrients; therefore, they must produce energy through anaerobic metabolism. This results in low energy yields from nutrients and an acidotic intracellular environment. Because of these changes, normal cell function ceases (Fig. 15-1). The cell swells and the cell membrane becomes more permeable, allowing electrolytes and fluids to seep out of and into the cell. The sodium–potassium pump becomes impaired; cell structures, primarily the mitochondria, are damaged; and death of the cell results.

Glucose is the primary substrate required for the production of cellular energy in the form of ATP. In stress states, catecholamines, cortisol, glucagons, and inflammatory cytokines and mediators are released, causing hyperglycemia and insulin resistance to mobilize glucose for cellular metabolism. Activation of these substances promotes gluconeogenesis, which is the formation of glucose from noncarbohydrate sources such as proteins and fats. Glycogen that has been stored in the liver is converted to glucose through glycogenolysis to meet metabolic needs, increasing the blood glucose concentration (ie, hyperglycemia).

Continued activation of the stress response by shock states causes a depletion of glycogen stores, resulting in increased proteolysis and eventual organ failure (Vincent, 2007). The inability of the body to have enough nutrients and oxygen for normal cellular metabolism causes a buildup of metabolic end products in the cells and interstitial spaces. Cellular metabolism is impaired, and a negative feedback loop is initiated.

Vascular Responses

Local regulatory mechanisms, referred to as autoregulation, stimulate vasodilation or vasoconstriction in response to **biochemical mediators** (ie, cytokines) released by the cell, communicating the need for oxygen and nutrients (King, 2007). A biochemical mediator is a substance released by a cell or immune cells such as macrophages; the substance triggers an action at a cell site or travels in the bloodstream to a distant site, where it triggers action. Researchers are learning more every day about the physiologic actions of more than 100 known cytokines (VonRueden, et al., 2008).

Blood Pressure Regulation

Three major components of the circulatory system—blood volume, the cardiac pump, and the vasculature—must respond effectively to complex neural, chemical, and

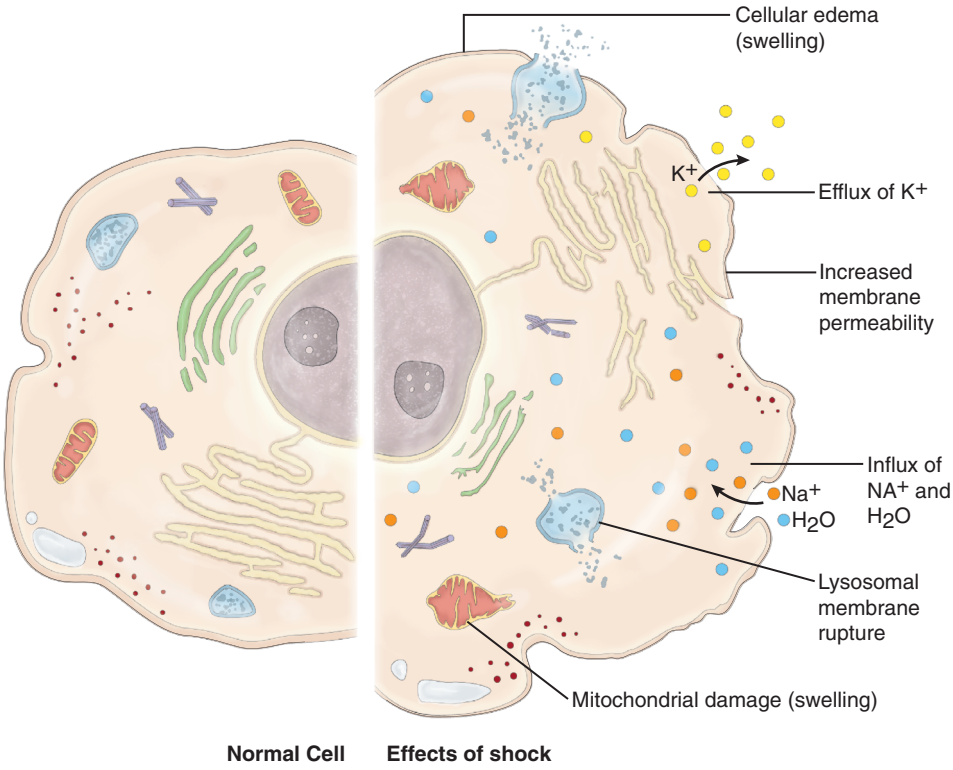

Figure 15-1 Cellular effects of shock. The cell swells and the cell membrane becomes more permeable; fluids and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged, and the cell dies.

hormonal feedback systems to maintain an adequate blood pressure (BP) and perfuse body tissues. BP is regulated through a complex interaction of neural, chemical, and hormonal feedback systems affecting both cardiac output and peripheral resistance. This relationship is expressed in the following equation:

Mean arterial BP = Cardiac output × Peripheral resistance

Cardiac output is a product of the stroke volume (the amount of blood ejected from the left ventricle during systole) and heart rate. Peripheral resistance is determined by the diameter of the arterioles.

Tissue perfusion and organ perfusion depend on mean arterial pressure (MAP), or the average pressure at which blood moves through the vasculature. MAP must exceed 65 mm Hg for cells to receive the oxygen and nutrients needed to metabolize energy in amounts sufficient to sustain life (Dellinger, et al., 2008). True MAP can be calculated only by complex methods. Frequently, MAP is calculated by automatic blood pressure machines, however, the nurse must ensure accurate blood pressure measurement is obtained before interpreting data from automated vital sign equipment.

BP is regulated by baroreceptors (pressure receptors) located in the carotid sinus and aortic arch. These pressure receptors are responsible for monitoring the circulatory volume and regulating neural and endocrine activities (see Chapter 14 for further description). When BP drops, catecholamines (epinephrine and norepinephrine) are released from the adrenal medulla. These increase heart rate and cause vasoconstriction, thus restoring BP. Chemoreceptors, also located in the aortic arch and carotid arteries, regulate BP and respiratory rate using much the same mechanism in response to changes in oxygen and carbon dioxide concentrations in the blood. These primary regulatory mechanisms can respond to changes in BP on a moment-to-moment basis.

The kidneys regulate BP by releasing renin, an enzyme needed for the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. This stimulation of the renin–angiotensin mechanism and the resulting vasoconstriction indirectly lead to the release of aldosterone from the adrenal cortex, which promotes the retention of sodium and water. The increased concentration of sodium in the blood stimulates the release of antidiuretic hormone (ADH) by the pituitary gland. ADH causes the kidneys to retain water further in an effort to raise blood volume and BP. These secondary regulatory mechanisms may take hours or days to respond to changes in BP. The relationship between the initiation of shock and the responsiveness of primary and secondary regulatory mechanisms that compensate for deficits in blood volume, the pumping effectiveness of the heart, or vascular tone, which may result because of the shock state, is noted in Figure 15-2.

Stages of Shock

Shock is believed to progress along a continuum of stages. Shock can be identified as early or late, depending on the signs and symptoms and the overall severity of organ dysfunction. A convenient way to understand the physiologic responses and subsequent clinical signs and symptoms of shock is to divide the continuum into separate stages: compensatory (stage 1), progressive (stage 2), and irreversible (stage 3). The earlier that medical and nursing

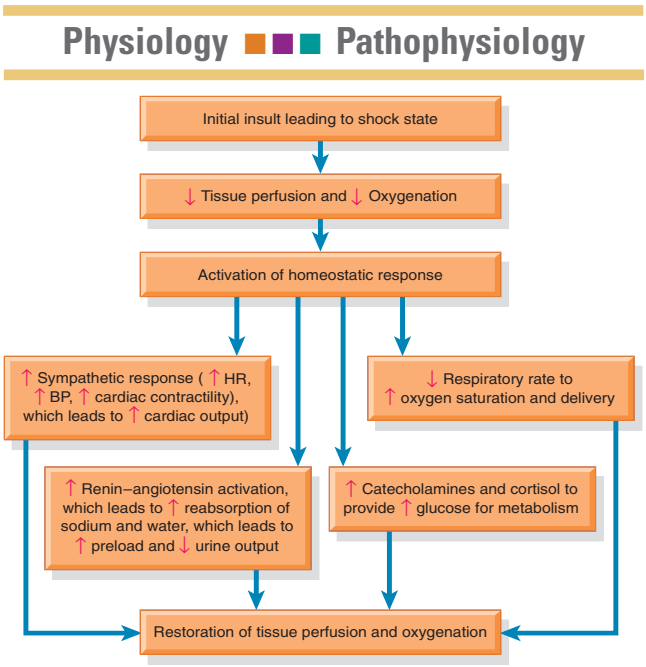

Figure 15-2 Compensatory mechanisms in shock.

interventions are initiated along this continuum, the greater the patient's chance of survival. Current research is focusing on assessing patients at greatest risk for shock and implementing early and aggressive interventions to reverse tissue hypoxia (King, 2007; Otero, Nguygen, Huang, et al., 2006). Studies suggest that the window of opportunity that increases the likelihood of patient survival occurs when aggressive therapy begins within 6 hours of identifying a shock state, especially septic shock (Otero, et al., 2006; Rivers, McIntyre, Morro, et al., 2005).

COMPENSATORY STAGE

In the compensatory stage of shock, the BP remains within normal limits. Vasoconstriction, increased heart rate, and increased contractility of the heart contribute to maintaining adequate cardiac output. This results from stimulation of the sympathetic nervous system and subsequent release of catecholamines (epinephrine and norepinephrine). Patients display the often-described "fight or flight" response. The body shunts blood from organs such as the skin, kidneys, and gastrointestinal tract to the brain, heart, and lungs to ensure adequate blood supply to these vital organs. As a result, the skin is cool and clammy, bowel sounds are hypoactive, and urine output decreases in response to the release of aldosterone and ADH.

Clinical Manifestations

Despite a normal BP, the patient shows numerous clinical signs indicating inadequate organ perfusion (Table 15-1). The result of inadequate perfusion is anaerobic metabolism and a buildup of lactic acid, producing metabolic acidosis. The respiratory rate increases in response to metabolic acidosis. This rapid respiratory rate facilitates removal of excess carbon dioxide but raises the blood pH and often causes a compensatory respiratory alkalosis. The alkalotic state causes mental status changes, such as confusion or combativeness, as well as arteriolar dilation. If treatment begins in this stage of shock, the prognosis for the patient is better than in later stages.

Medical Management

Medical treatment is directed toward identifying the cause of the shock, correcting the underlying disorder so that shock does not progress, and supporting those physiologic processes that thus far have responded successfully to the threat. Because compensation cannot be maintained indefinitely, measures such as fluid replacement and medication therapy must be initiated to maintain an adequate BP and reestablish and maintain adequate tissue perfusion (Otero, et al., 2006).

Nursing Management

As stated earlier, intervention as soon as possible along the continuum of shock is the key to improving the patient's prognosis. The nurse must systematically assess the patient at risk for shock to recognize the subtle clinical signs of the compensatory stage before the patient's BP drops. Special considerations related to recognizing early signs of shock in the elderly patient are given in Chart 15-1.

Table 15-1 CLINICAL FINDINGS IN STAGES OF SHOCK

	Stage		
Finding	**Compensatory**	**Progressive**	**Irreversible**
Blood pressure	Normal	Systolic <80–90 mm Hg Requires fluids resuscitation to support blood pressure	Requires mechanical or pharmacologic support
Heart rate	>100 bpm	>150 bpm	Erratic or asystole
Respiratory status	>20 breaths/min $PaCO_2$ <32 mm Hg	Rapid, shallow respirations; crackles PaO_2 <80 mm Hg $PaCO_2$ >45 mm Hg	Requires intubation and mechanical ventilation and oxygenation
Skin	Cold, clammy	Mottled, petechiae	Jaundice
Urinary output	Decreased	0.5 mL/kg/h	Anuric, requires dialysis
Mentation	Confusion	Lethargy	Unconscious
Acid–base balance	Respiratory alkalosis	Metabolic acidosis	Profound acidosis

CHART 15-1

Recognizing Shock in Older Patients

The physiologic changes associated with aging, coupled with pathologic and chronic disease states, place older people at increased risk for developing a state of shock and possibly multiple organ dysfunction syndrome (MODS). Elderly people can recover from shock if it is detected and treated early with aggressive and supportive therapies. Nurses play an essential role in assessing and interpreting subtle changes in older patients' responses to illness.

- Medications such as beta-blocking agents (metoprolol [Lopressor]) used to treat hypertension may mask tachycardia, a primary compensatory mechanism to increase cardiac output, during hypovolemic states.
- The aging immune system may not mount a truly febrile response (temperature more than 38°C [100.4°F]), but an increasing trend in body temperature should be addressed. The patient may also report increased fatigue and malaise in the absence of a febrile response.
- The heart does not function well in hypoxemic states, and the aging heart may respond to decreased myocardial oxygenation with dysrhythmias that may be misinterpreted as a normal part of the aging process.
- There is a progressive decline in respiratory muscle strength, maximal ventilation, and response to hypoxia. Older patients have a decreased respiratory reserve and decompensate more quickly.
- Changes in mentation may be inappropriately misinterpreted as dementia. Older people with a sudden change in mentation should be aggressively treated for the presence of infection and organ hypoperfusion.

Monitoring Tissue Perfusion

In assessing tissue perfusion, the nurse observes for changes in level of consciousness, vital signs (including pulse pressure), urinary output, skin, and laboratory values (eg, base deficit and lactic acid levels). In the compensatory stage of shock, serum sodium and blood glucose levels are elevated in response to the release of aldosterone and catecholamines.

The nurse should monitor the patient's hemodynamic status and promptly report deviations to the physician, assist in identifying and treating the underlying disorder by continuous in-depth assessment of the patient, administer prescribed fluids and medications, and promote patient safety. Vital signs are key indicators of hemodynamic status and BP is an indirect measure of tissue hypoxia. The nurse should report a systolic BP lower than 90 mm Hg or a drop in systolic BP of 40 mm Hg from baseline.

Pulse pressure correlates well with stroke volume. Pulse pressure is calculated by subtracting the diastolic measurement from the systolic measurement; the difference is the pulse pressure (Cottingham, 2006). Normally, the pulse pressure is 30 to 40 mm Hg. Narrowing or decreased pulse pressure is an earlier indicator of shock than a drop in systolic BP. Decreased or narrowing pulse pressure, an early indication of decreased stroke volume, is illustrated in the following example:

$$\text{Systolic BP} - \text{Diastolic BP} = \text{Pulse pressure}$$

Normal pulse pressure:

$$120 \text{ mg Hg} - 80 \text{ mm Hg} = 40 \text{ mm Hg}$$

Narrowing of pulse pressure:

$$90 \text{ mm Hg} - 70 \text{ mm Hg} = 20 \text{ mm Hg}$$

Elevation of the diastolic BP with release of catecholamines and attempts to increase venous return through vasoconstriction is an early compensatory mechanism in response to decreased stroke volume, BP, and overall cardiac output.

NURSING ALERT

By the time BP drops, damage has already been occurring at the cellular and tissue levels. Therefore, the patient at risk for shock must be assessed and monitored closely before the BP falls.

Continuous central venous oximetry ($Sc\bar{v}O_2$) monitoring may be used to evaluate mixed venous blood oxygen saturation and the severity of tissue hypoperfusion states. A central catheter is introduced into the superior vena cava (SVC), and a sensor on the catheter measures the oxygen saturation of the blood in the SVC as blood returns to the heart and pulmonary system for reoxygenation. A normal $Sc\bar{v}O_2$ value is 70% (Goodrich, 2006; Rivers, et al., 2005). Body tissues use approximately 25% of the oxygen delivered to them during normal metabolism. During states of stress, such as shock, more oxygen is consumed and the $Sc\bar{v}O_2$ saturation is lower, indicating that the tissues are consuming more oxygen.

Interventions focus on decreasing tissue oxygen requirements and increasing perfusion to deliver more oxygen to the tissues. For instance, sedating agents may be administered to lower metabolic demands, the patient's pain may be treated with intravenous (IV) opioid agents, or measures to prevent shivering, decrease metabolic demands for oxygen. Supplemental oxygen and mechanical ventilation may be required to increase the delivery of oxygen in the blood. Administration of IV fluids and medications supports blood pressure and cardiac output, and the transfusion of packed red blood cells enhances oxygen transport. Monitoring tissue oxygen consumption with $Sc\bar{v}O_2$ is a minimally invasive measure to more accurately assess tissue oxygenation in the compensatory stage of shock before changes in vital signs detect altered tissue perfusion (Dellinger, et al., 2008; Goodrich, 2006; Otero, et al., 2006).

New technologies allow clinicians to detect changes in tissue perfusion before changes in classic signs (BP, heart rate, and urine output) indicative of hypoperfusion occur.

Two of these technologies include sublingual capnometry and near-infrared spectroscopy. Sublingual capnometry, a noninvasive technology, provides information about the degree of hypoperfusion based on the sublingual partial pressure of carbon dioxide (PCO_2) (Goodrich, 2006). A probe is placed under the patient's tongue, and PCO_2 levels are derived from the blood flow found in the mucosal bed. During shock, an elevated PCO_2 indicates poor tissue perfusion. Near-infrared spectroscopy (NIRS), a continuous noninvasive technology, uses light transmission to measure skeletal muscle oxygenation as an indicator of shock. The NIRS probe is applied to the thenar muscle that is located on the palm of the hand near the thumb, and it measures the oxygen saturation of tissue by determining the amount of infrared light absorption. Low values of tissue oxygenation (eg, less than 80%) indicate severity of shock; the lower the value, the more severe the tissue hypoxia.

Although treatments are prescribed and initiated by the physician, the nurse usually implements them, operates and troubleshoots equipment used in treatment, monitors the patient's status during treatment, and evaluates the immediate effects of treatment. In addition, the nurse assesses the response of the patient and family to the crisis and its treatment.

Reducing Anxiety

Patients and their families often become anxious and apprehensive when they face a major threat to health and well-being and are the focus of attention of many health care providers. Providing brief explanations about the diagnostic and treatment procedures, supporting the patient during these procedures, and providing information about their outcomes are usually effective in reducing stress and anxiety and thus promoting the patient's physical and mental well-being. Speaking in a calm reassuring voice and using gentle touch also help ease the patient's concerns. These actions may provide comfort for critically ill, frightened patients (Benner, 2004; Duran, Oman, Abel, et al., 2007). Research has repeatedly shown that family members have certain needs during a health-related crisis, including needing honest, consistent, and thorough communication with health care providers; needing physical and emotional closeness to the patient; sensing that health care providers care about their patients; seeing the patient frequently; and knowing exactly what has been done for the patient (Duran, et al., 2007).

The nurse should advocate that family members be present during procedures and while patient care is provided. The presence of family provides a necessary connection and support for the patient during a time of crisis.

Promoting Safety

The nurse must be vigilant for potential threats to the patient's safety, because a high anxiety level and altered mental status impair judgment. In this stage of shock, patients who were previously cooperative and followed instructions may now disrupt IV lines and catheters and complicate their condition. Therefore, close monitoring and frequent reorientation interventions are essential.

PROGRESSIVE STAGE

In the second stage of shock, the mechanisms that regulate BP can no longer compensate, and the MAP falls below normal limits. Patients are clinically hypotensive; this is defined as a systolic BP of less than 90 mm Hg or a decrease in systolic BP of 40 mm Hg from baseline (Dellinger, et al., 2008; VonRueden, et al., 2008).

Pathophysiology

Although all organ systems suffer from hypoperfusion at this stage, several events perpetuate the shock syndrome. First, the overworked heart becomes dysfunctional, the body's inability to meet increased oxygen requirements produces ischemia, and biochemical mediators cause myocardial depression (Dellinger, et al., 2008; Otero, et al., 2006; VonRueden, et al., 2008). This leads to failure of the cardiac pump, even if the underlying cause of the shock is not of cardiac origin. Second, the autoregulatory function of the microcirculation fails in response to the numerous biochemical mediators released by the cells, resulting in increased capillary permeability, with areas of arteriolar and venous constriction further compromising cellular perfusion (King, 2007; VonRueden, et al., 2008). At this stage, the prognosis worsens. The relaxation of precapillary sphincters causes fluid to leak from the capillaries, creating interstitial edema and return of less fluid to the heart. In addition, the inflammatory response to injury is activated, and proinflammatory and anti-inflammatory mediators are released, which activate the coagulation system in an effort to reestablish homeostasis (King, 2007). The body mobilizes energy stores and increases oxygen consumption to meet the increased metabolic needs of the underperfused tissues and cells.

Even if the underlying cause of the shock is reversed, the sequence of compensatory responses to the decrease in tissue perfusion perpetuates the shock state, and a vicious circle ensues. The cellular reactions that occur during the progressive stage of shock are an active area of clinical research. It is believed that the body's response to shock or lack of response in this stage of shock may be the primary factor determining the patient's survival.

Clinical Manifestations

Chances of survival depend on the patient's general health before the shock state as well as the amount of time it takes to restore tissue perfusion. As shock progresses, organ systems decompensate.

Respiratory Effects

The lungs, which become compromised early in shock, are affected at this stage. Subsequent decompensation of the lungs increases the likelihood that mechanical ventilation will be needed. Respirations are rapid and shallow. Crackles are heard over the lung fields. Decreased pulmonary blood flow causes arterial oxygen levels to decrease and carbon dioxide levels to increase. Hypoxemia and biochemical mediators cause an intense inflammatory response and pulmonary vasoconstriction, perpetuating pulmonary capillary hypoperfusion and hypoxemia. The hypoperfused alveoli

stop producing surfactant and subsequently collapse. Pulmonary capillaries begin to leak, causing pulmonary edema, diffusion abnormalities (shunting), and additional alveolar collapse. This condition is called acute lung injury (ALI); as ALI continues, interstitial inflammation and fibrosis are common consequences, leading to acute respiratory distress syndrome (ARDS) (Cocci, et al., 2007; Girard, Kess, Fuchs, et al., 2008; Villar, Perez-Mendez, Lopez, et al., 2007). Further explanation of ALI and ARDS, as well as their nursing management, can be found in Chapter 23.

Cardiovascular Effects

A lack of adequate blood supply leads to dysrhythmias and ischemia. The heart rate is rapid, sometimes exceeding 150 bpm. The patient may complain of chest pain and even suffer a myocardial infarction. Levels of cardiac enzymes (eg, myocardial creatine kinase [CK-MB] and cardiac troponin I [cTn-I]) increase. In addition, myocardial depression and ventricular dilation may further impair the heart's ability to pump enough blood to the tissues to meet oxygen requirements.

New laboratory markers can be used to assess the function of the heart. B-type natriuretic peptide (BNP) is one of these markers. BNP is increased when the ventricle is overdistended; therefore, elevations in BNP can be used to assess ventricular function of patients in shock states (Wilson Tang & Francis, 2005).

Neurologic Effects

As blood flow to the brain becomes impaired, mental status deteriorates. Changes in mental status occur with decreased cerebral perfusion and hypoxia. Initially, the patient may exhibit subtle changes in behavior or agitation and confusion. Subsequently, lethargy increases, and the patient begins to lose consciousness.

Renal Effects

When the MAP falls below 70 mm Hg (Cottingham, 2006; Dellinger, et al., 2008; King, 2007; Pinsky, 2007), the glomerular filtration rate of the kidneys cannot be maintained, and drastic changes in renal function occur. Acute renal failure (ARF) may develop. ARF is characterized by an increase in blood urea nitrogen (BUN) and serum creatinine levels, fluid and electrolyte shifts, acid–base imbalances, and a loss of the renal-hormonal regulation of BP. Urinary output usually decreases to less than 0.5 mL/kg/h (or less than 30 mL/h) but may vary depending on the phase of ARF. For further information about ARF, see Chapter 44.

Hepatic Effects

Decreased blood flow to the liver impairs the ability of liver cells to perform metabolic and phagocytic functions. Consequently, the patient is less able to metabolize medications and metabolic waste products, such as ammonia and lactic acid. Metabolic activities of the liver, including gluconeogenesis and glycogenolysis, are impaired. The patient becomes more susceptible to infection as the liver fails to filter bacteria from the blood. Liver enzymes (aspartate aminotransferase [AST], alanine aminotransferase [ALT], lactate dehydrogenase [LDH]) and bilirubin levels are elevated, and the patient appears jaundiced.

Gastrointestinal Effects

Gastrointestinal (GI) ischemia can cause stress ulcers in the stomach, putting the patient at risk for GI bleeding. In the small intestine, the mucosa can become necrotic and slough off, causing bloody diarrhea. Beyond the local effects of impaired perfusion, GI ischemia leads to bacterial toxin translocation, in which bacterial toxins enter the bloodstream through the lymphatic system. In addition to causing infection, bacterial toxins can cause cardiac depression, vasodilation, increased capillary permeability, and an intense inflammatory response with activation of additional biochemical mediators. The net result is interference with healthy cellular functioning and their ability to metabolize nutrients (Stapleton, Jones & Heyland, 2007).

Hematologic Effects

The combination of hypotension, sluggish blood flow, metabolic acidosis, coagulation system imbalance, and generalized hypoxemia can interfere with normal hemostatic mechanisms. In shock states, the inflammatory cytokines activate the clotting cascade, causing deposition of microthrombi in multiple areas of the body and consumption of clotting factors. The alterations of the hematologic system, including imbalance of the clotting cascade, are linked to the overactivation of the inflammatory response of injury (Remick, 2007a; VonRueden, et al., 2008). Disseminated intravascular coagulation (DIC) may occur either as a cause or as a complication of shock. In this condition, widespread clotting and bleeding occur simultaneously. Bruises (ecchymoses) and bleeding (petechiae) may appear in the skin. Coagulation times (eg, prothrombin time [PT], activated partial thromboplastin time [aPTT]) are prolonged. Clotting factors and platelets are consumed and require replacement therapy to achieve hemostasis. Further discussion of DIC appears in Chapter 33.

Medical Management

Specific medical management in the progressive stage of shock depends on the type of shock and its underlying cause. It is also based on the degree of decompensation in the organ systems. Medical management specific to each type of shock is discussed later in this chapter. Although there are several differences in medical management by type of shock, some medical interventions are common to all types. These include the use of appropriate IV fluids and medications to restore tissue perfusion by the following methods:

- Supporting the respiratory system
- Optimizing intravascular volume
- Supporting the pumping action of the heart
- Improving the competence of the vascular system

Other aspects of management may include early enteral nutritional support, aggressive hyperglycemic control with IV insulin (Hafidh, Reuter, Chassels, et al., 2007; Vanhorebeek, Langouche & Van den Berghe, 2007), and use of antacids, histamine-2 (H_2) blockers, or antipeptic agents to reduce the risk of GI ulceration and bleeding.

NURSING ALERT

Tight glycemic control (blood glucose, 80 to 110 mg/dL) has been shown to reduce morbidity and mortality of acutely ill patients.

Nursing Management

Nursing care of patients in the progressive stage of shock requires expertise in assessing and understanding shock and the significance of changes in assessment data. Early interventions are essential to the survival of patients; therefore, suspecting that a patient may be in shock and reporting subtle changes in assessment are imperative. Patients in the progressive stage of shock are cared for in the intensive care setting to facilitate close monitoring (hemodynamic monitoring, electrocardiographic [ECG] monitoring, arterial blood gases, serum electrolyte levels, physical and mental status changes); rapid and frequent administration of various prescribed medications and fluids; and possibly interventions with supportive technologies, such as mechanical ventilation, dialysis, and intra-aortic balloon pump.

Working closely with other members of the health care team, the nurse carefully documents treatments, medications, and fluids that are administered, recording the time, dosage or volume, and patient response. In addition, the nurse coordinates both the scheduling of diagnostic procedures that may be carried out at the bedside and the flow of health care personnel involved in the care of patients.

Preventing Complications

The nurse helps reduce the risk of related complications and monitors the patient for early signs of complications. Monitoring includes evaluating blood levels of medications, observing invasive vascular lines for signs of infection, and checking neurovascular status if arterial lines are inserted, especially in the lower extremities. Simultaneously, the nurse promotes the patient's safety and comfort by ensuring that all procedures, including invasive procedures and arterial and venous punctures, are carried out using correct aseptic techniques and that venous and arterial puncture and infusion sites are maintained with the goal of preventing infection. Nursing interventions that reduce the incidence of ventilator-associated pneumonias must also be implemented. These include frequent oral care, aseptic suction technique, turning, and elevating the head of the bed at least 30 degrees to prevent aspiration (Carson, Tyner, Sanders, et al., 2007; Dellinger, et al., 2008). Positioning and repositioning of the patient to promote comfort and maintain skin integrity are essential.

Promoting Rest and Comfort

Efforts are made to minimize the cardiac workload by reducing the patient's physical activity and treating pain and anxiety. Promoting patient rest and comfort is a priority. To ensure that the patient obtains as much uninterrupted rest as possible, the nurse performs only essential nursing activities. To conserve the patient's energy, the nurse should protect the patient from temperature extremes (eg, excessive warmth or cold, and shivering), which can increase the metabolic rate and oxygen consumption and thus the cardiac workload. The patient should not be warmed too quickly, and warming blankets should not be applied, because they can cause vasodilation and a subsequent drop in BP.

Supporting Family Members

Because patients in shock receive intense attention by the health care team, families may be overwhelmed and frightened. Family members may be reluctant to ask questions or seek information for fear that they will be in the way or will interfere with the attention given to the patient. The nurse should make sure that the family is comfortably situated and kept informed about the patient's status. Often, families need advice from the health care team to get some rest; family members are more likely to take this advice if they feel that the patient is being well cared for and that they will be notified of any significant changes in the patient's status. A visit from the hospital chaplain may be comforting and provides some attention to the family while the nurse concentrates on the patient.

IRREVERSIBLE STAGE

The irreversible (or refractory) stage of shock represents the point along the shock continuum at which organ damage is so severe that the patient does not respond to treatment and cannot survive. Despite treatment, BP remains low. Renal and liver failure, compounded by the release of necrotic tissue toxins, creates an overwhelming metabolic acidosis. Anaerobic metabolism contributes to a worsening lactic acidosis. Reserves of ATP are almost totally depleted, and mechanisms for storing new supplies of energy have been destroyed. Respiratory system failure prevents adequate oxygenation and ventilation despite mechanical ventilatory support, and the cardiovascular system is ineffective in maintaining an adequate MAP for perfusion. Multiple organ dysfunction progressing to complete organ failure has occurred, and death is imminent. Multiple organ dysfunction can occur as a progression along the shock continuum or as a syndrome unto itself and is described in more detail later in this chapter.

Medical Management

Medical management during the irreversible stage of shock is usually the same as for the progressive stage. Although the patient may have progressed to the irreversible stage, the judgment that the shock is irreversible can be made only retrospectively on the basis of the patient's failure to respond to treatment. Strategies that may be experimental (eg, investigational medications, such as antibiotic agents and immunomodulation therapy) may be tried to reduce or reverse the severity of shock.

Nursing Management

As in the progressive stage of shock, the nurse focuses on carrying out prescribed treatments, monitoring the patient, preventing complications, protecting the patient from injury, and providing comfort. Offering brief explanations to the patient

about what is happening is essential even if there is no certainty that the patient hears or understands what is being said. Simple comfort measures, including reassuring touches, should continue to be provided despite the patient's nonresponsiveness to verbal stimuli (Benner, 2004, Duran, et al., 2007).

As it becomes obvious that the patient is unlikely to survive, the family must be informed about the prognosis and likely outcome. Opportunities should be provided throughout the patient's care for the family to see, touch, and talk to the patient. Close family friends or spiritual advisors may be of comfort to the family members in dealing with the inevitable death of their loved one. Whenever possible and appropriate, the patient's family should be approached regarding any living wills, advance directives, or other written or verbal wishes the patient may have shared in the event that he or she became unable to participate in end-of-life decisions. In some cases, ethics committees may assist families and health care teams in making difficult decisions.

During this stage of shock, the family may misinterpret the actions of the health care team. They have been told that nothing has been effective in reversing the shock and that the patient's survival is very unlikely, yet they find physicians and nurses continuing to work feverishly on the patient. Distraught, grieving families may interpret this as a chance for recovery when none exists, and family members may become angry when the patient dies. Conferences with all members of the health care team and the family promote better understanding by the family of the patient's prognosis and the purpose for management measures. During these conferences, it is essential to explain that the equipment and treatments being provided are intended for patient comfort and do not suggest that the patient will recover. Family members should be encouraged to express their wishes concerning the use of life-support measures.

General Management Strategies in Shock

As described previously and in the discussion of types of shock to follow, management in all types and all phases of shock includes the following:

- Support of the respiratory system with supplemental oxygen and/or mechanical ventilation to provide optimal oxygenation (see Chapter 25)
- Fluid replacement to restore intravascular volume
- Vasoactive medications to restore vasomotor tone and improve cardiac function
- Nutritional support to address the metabolic requirements that are often dramatically increased in shock

Therapies described in this section require collaboration among all members of the health care team to ensure that the manifestations of shock are quickly identified and that adequate and timely treatment is instituted to achieve the best outcome possible.

Fluid Replacement

Fluid replacement, also referred to as fluid resuscitation, is administered in all types of shock. The type of fluids administered and the speed of delivery vary, but fluids are administered to improve cardiac and tissue oxygenation, which in part depends on flow. The fluids administered may include **crystalloids** (electrolyte solutions that move freely between intravascular and interstitial spaces), **colloids** (large-molecule IV solutions), and blood components (packed red blood cells, fresh frozen plasma, and platelets).

Crystalloid and Colloid Solutions

The best fluid to treat shock remains controversial. In emergencies, the "best" fluid is often the fluid that is readily available. Fluid resuscitation should be initiated early in shock to maximize intravascular volume. There is no consensus regarding whether crystalloids or colloids should be used; however, with crystalloids, more fluid is necessary to restore intravascular volume (Roberts, Alderson, Bunn, et al., 2007).

Crystalloids are electrolyte solutions that move freely between the intravascular compartment and the interstitial spaces. Isotonic crystalloid solutions are often selected because they contain the same concentration of electrolytes as the extracellular fluid and therefore can be given without altering the concentrations of electrolytes in the plasma. IV crystalloids commonly used for resuscitation in hypovolemic shock include 0.9% sodium chloride solution (normal saline) and lactated Ringer's solution (Boswell & Scalea, 2008; Cottingham, 2006). Ringer's lactate is an electrolyte solution containing the lactate ion, which should not be confused with lactic acid. The lactate ion is converted to bicarbonate, which helps buffer the overall acidosis that occurs in shock. A disadvantage of using isotonic crystalloid solutions is that some of the volume administered is lost to the interstitial compartment and some remains in the intravascular compartment. This occurs as a consequence of cellular permeability that occurs during shock. Diffusion of crystalloids into the interstitial space means that more fluid must be administered than the amount lost (Cottingham, 2006; Roberts, et al., 2007).

Care must be taken when rapidly administering isotonic crystalloids to avoid both underresuscitating and overresuscitating the patient in shock. Insufficient fluid replacement is associated with a higher incidence of morbidity and mortality from lack of tissue perfusion, whereas excessive fluid administration can cause systemic and pulmonary edema that progresses to ARDS, abdominal compartment syndrome, and multiple organ dysfunction syndrome (MODS).

Depending on the cause of the hypovolemia, a hypertonic crystalloid solution, such as 3% sodium chloride, is sometimes administered in hypovolemic shock. These solutions exert a large osmotic force that pulls fluid from the intracellular space to the extracellular space to achieve a fluid balance (Cottingham, 2006). This osmotic effect results in fewer fluids being administered to restore intravascular volume. Complications associated with use of hypertonic solutions include excessive serum osmolality, which can cause rapid fluid shifts overwhelming the heart, and hypernatremia.

Generally, IV colloidal solutions are similar to plasma proteins, in that they contain molecules that are too large to pass through capillary membranes. Colloids expand intravascular volume by exerting oncotic pressure, thereby pulling fluid into the intravascular space. Colloidal solutions

have the same effect as hypertonic solutions in increasing intravascular volume, but less volume of fluid is required than with crystalloids. In addition, colloids have a longer duration of action than crystalloids, because the molecules remain within the intravascular compartment longer.

Typically, if colloids are used to treat tissue hypoperfusion, albumin is the agent prescribed. Albumin is a plasma protein; an albumin solution is prepared from human plasma and is heated during production to reduce its potential to transmit disease. The disadvantage of albumin is its high cost compared to crystalloid solutions. Synthetic colloid preparations, such as hetastarch and dextran solution, may also be used for colloid infusions; however, dextran may interfere with platelet aggregation and, therefore, is not indicated if hemorrhage is the cause of the hypovolemic shock or if the patient has a coagulation disorder.

NURSING ALERT

With all colloidal solutions, side effects include the rare occurrence of anaphylactic reactions. Nurses must monitor patients closely.

Complications of Fluid Administration

Close monitoring of the patient during fluid replacement is necessary to identify side effects and complications. The most common and serious side effects of fluid replacement are cardiovascular overload and pulmonary edema. The patient receiving fluid replacement must be monitored frequently for adequate urinary output, changes in mental status, skin perfusion, and changes in vital signs. Lung sounds are auscultated frequently to detect signs of fluid accumulation. Adventitious lung sounds, such as crackles, may indicate pulmonary edema.

Abdominal compartment syndrome (ACS) is a serious complication that may occur when large volumes of fluid are administered. It may also occur after trauma, abdominal surgery, severe pancreatitis, or sepsis (Brush, 2007). In ACS, fluid leaks into the intra-abdominal cavity, increasing pressure that is displaced onto surrounding vessels and organs. Venous return, preload, and cardiac output are compromised. The pressure also elevates the diaphragm, making it difficult to breathe effectively. The renal system and GI systems also begin to show signs of dysfunction (eg, decreased urine output, absent bowel sounds, intolerance of tube feeding). Abdominal compartment pressure can be measured. Normally, it is 0 to 5 mm Hg, and a pressure of 12 mm Hg is considered to be indicative of intra-abdominal hypertension (Brush, 2007). If ACS is present, interventions that usually include surgical decompression are necessary to relieve the pressure.

NURSING ALERT

When administering large volumes of crystalloid solutions, monitor the lungs for adventitious sounds and signs and symptoms of interstitial edema (eg, abdominal compartment syndrome).

Often a right atrial pressure line (also known as a central venous pressure [CVP] line) is inserted. In addition to physical assessment, the right atrial pressure value helps in monitoring the patient's response to fluid replacement. A normal right atrial pressure value or CVP is 4 to 12 mm Hg or cm H_2O. Several readings are obtained to determine a range, and fluid replacement is continued to achieve a CVP of at least 8 mm Hg (Dellinger, et al., 2008). With newer technologies, right atrial catheters can be placed that allow the monitoring of intravascular pressures and venous oxygen levels. Assessment of venous oxygenation (venous oxygen saturation ([SvO_2], or $Sc\bar{v}O_2$ with a CVP line) is helpful in evaluating the adequacy of intravascular volume (Goodrich, 2006; Rivers, et al., 2005). Hemodynamic monitoring with arterial and pulmonary artery lines may be implemented to allow close monitoring of the patient's perfusion and cardiac status as well as response to therapy. For additional information about hemodynamic monitoring, see Chapter 26.

Vasoactive Medication Therapy

Vasoactive medications are administered in all forms of shock to improve the patient's hemodynamic stability when fluid therapy alone cannot maintain adequate MAP. Specific medications are selected to correct the particular hemodynamic alteration that is impeding cardiac output. These medications help increase the strength of myocardial contractility, regulate the heart rate, reduce myocardial resistance, and initiate vasoconstriction.

Vasoactive medications are selected for their action on receptors of the sympathetic nervous system. These receptors are known as alpha-adrenergic and beta-adrenergic receptors. Beta-adrenergic receptors are further classified as beta-1 and beta-2 adrenergic receptors. When alpha-adrenergic receptors are stimulated, blood vessels constrict in the cardiorespiratory and GI systems, skin, and kidneys. When beta-1 adrenergic receptors are stimulated, heart rate and myocardial contraction increase. When beta-2 adrenergic receptors are stimulated, vasodilation occurs in the heart and skeletal muscles, and the bronchioles relax. The medications used in treating shock consist of various combinations of vasoactive medications to maximize tissue perfusion by stimulating or blocking the alpha- and beta-adrenergic receptors.

When vasoactive medications are administered, vital signs must be monitored frequently (at least every 15 minutes until stable, or more often if indicated). Vasoactive medications should be administered through a central venous line, because infiltration and extravasation of some vasoactive medications can cause tissue necrosis and sloughing. An IV pump or controller should be used to ensure that the medications are delivered safely and accurately.

Individual medication dosages are usually titrated by the nurse, who adjusts drip rates based on the prescribed dose and the patient's response. Dosages are changed to maintain the MAP at a physiologic level that ensures adequate tissue perfusion (usually greater than 65 mm Hg).

NURSING ALERT

Vasoactive medications should never be stopped abruptly, because this could cause severe hemodynamic instability, perpetuating the shock state.

Dosages of vasoactive medications should be tapered, and the patient should be weaned from medication with frequent monitoring of BP (every 15 minutes). Table 15-2 presents some of the commonly prescribed vasoactive medications used in the treatment of shock. Occasionally, the patient does not respond as expected to vasoactive medications. A current topic of active research is evaluation of patients' adrenal function. Recent studies suggest that critically ill patients should be evaluated for corticosteroid insufficiency, and if this condition is present, corticosteroid replacement (eg, hydrocortisone) should be initiated (Dellinger, et al., 2008).

Nutritional Support

Nutritional support is an important aspect of care for patients with shock. Increased metabolic rates during shock increase energy requirements and therefore caloric requirements. Patients in shock may require more than 3000 calories daily. The release of catecholamines early in the shock continuum causes depletion of glycogen stores in about 8 to 10 hours. Nutritional energy requirements are then met by breaking down lean body mass. In this catabolic process, skeletal muscle mass is broken down even when the patient has large stores of fat or adipose tissue. Loss of skeletal muscle greatly prolongs the patient's recovery time.

Parenteral or enteral nutritional support should be initiated as soon as possible. Enteral nutrition is preferred, promoting GI function through direct exposure to nutrients and limiting infectious complications associated with parenteral feeding (Vincent, 2007). In addition, glutamine (an essential amino acid during stress), which may be administered in enteral formulas, is important in the immunologic function of the GI tract, providing a fuel source for lymphocytes and macrophages (Stapleton, et al., 2007).

Stress ulcers occur frequently in acutely ill patients because of the compromised blood supply to the GI tract. Therefore, antacids, H_2 blockers (eg, famotidine [Pepcid], ranitidine [Zantac]), and proton pump inhibitors (eg, lansoprazole [Prevacid]) are prescribed to prevent ulcer formation by inhibiting gastric acid secretion or increasing gastric pH.

Hypovolemic Shock

Nurses who care for patients in the different stages of shock must tailor interventions to the type of shock, whether hypovolemic, cardiogenic, or circulatory shock. **Hypovolemic shock**, the most common type of shock, is characterized by decreased intravascular volume. Body fluid is contained in the intracellular and extracellular compartments. Intracellular fluid accounts for about two thirds of the total body water. The extracellular body fluid is found in one of two compartments: intravascular (inside blood vessels) or interstitial (surrounding tissues). The volume of interstitial fluid is about three to four times that of intravascular fluid. Hypovolemic shock occurs when there is a reduction in intravascular volume by 15% to 30%, which represents a loss of 750 to 1500 mL of blood in a 70-kg (154-lb) person (American College of Surgeons, 2006).

Pathophysiology

Hypovolemic shock can be caused by external fluid losses, as in traumatic blood loss, or by internal fluid shifts, as in severe dehydration, severe edema, or ascites (Chart 15-2). Intravascular volume can be reduced both by fluid loss and by fluid shifting between the intravascular and interstitial compartments.

The sequence of events in hypovolemic shock begins with a decrease in the intravascular volume. This results in decreased venous return of blood to the heart and subsequent decreased ventricular filling. Decreased ventricular filling results in decreased stroke volume (amount of blood ejected from the heart) and decreased cardiac output. When cardiac output drops, BP drops and tissues cannot be adequately perfused (Fig. 15-3).

Table 15-2 VASOACTIVE AGENTS USED IN TREATING SHOCK

Medication	Desired Action in Shock	Disadvantages
Inotropic Agents		
Dobutamine (Dobutrex) Dopamine (Intropin) Epinephrine (Adrenalin) Milrinone (Primacor)	Improve contractility, increase stroke volume, increase cardiac output	Increase oxygen demand of the heart
Vasodilators		
Nitroglycerin (Tridil) Nitroprusside (Nipride)	Reduce preload and afterload, reduce oxygen demand of heart	Cause hypotension
Vasopressor Agents		
Norepinephrine (Levophed) Dopamine (Intropin) Phenylephrine (Neo-Synephrine) Vasopressin (Pitressin)	Increase blood pressure by vasoconstriction	Increase afterload, thereby increasing cardiac workload; compromise perfusion to skin, kidneys, lungs, gastrointestinal tract

CHART 15-2 *Risk Factors for Hypovolemic Shock*

External: Fluid Losses	Internal: Fluid Shifts
• Trauma	• Hemorrhage
• Surgery	• Burns
• Vomiting	• Ascites
• Diarrhea	• Peritonitis
• Diuresis	• Dehydration
• Diabetes insipidus	

Medical Management

Major goals in the treatment of hypovolemic shock are to restore intravascular volume to reverse the sequence of events leading to inadequate tissue perfusion, to redistribute fluid volume, and to correct the underlying cause of the fluid loss as quickly as possible. Depending on the severity of shock and the patient's condition, it is likely that efforts will be made to address all three goals simultaneously.

Treatment of the Underlying Cause

If the patient is hemorrhaging, efforts are made to stop the bleeding. This may involve applying pressure to the bleeding site or surgical interventions to stop internal bleeding. If the cause of the hypovolemia is diarrhea or vomiting, medications to treat diarrhea and vomiting are administered while efforts are made to identify and treat the cause.

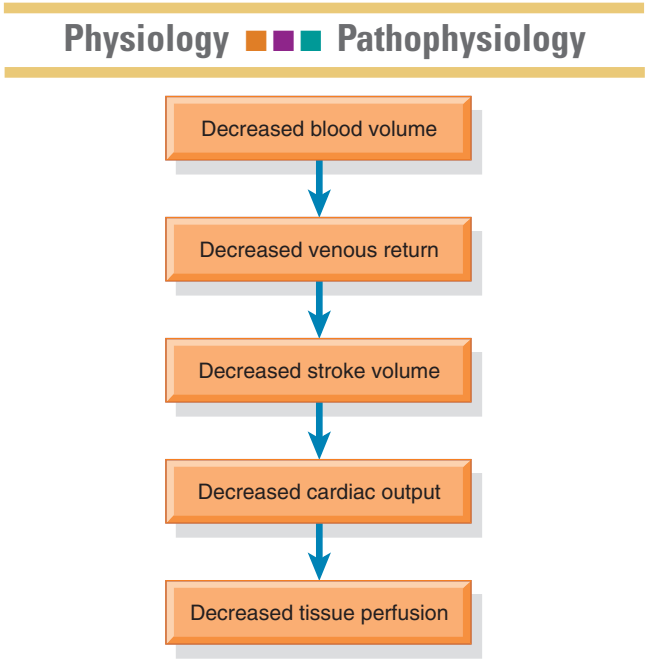

Figure 15-3 Pathophysiologic sequence of events in hypovolemic shock.

In elderly patients, dehydration may be the cause of hypovolemic shock.

Fluid and Blood Replacement

Beyond reversing the primary cause of the decreased intravascular volume, fluid replacement is of primary concern. At least two large-gauge IV lines are inserted to establish access for fluid administration. Two IV lines allow simultaneous administration of fluid, medications, and blood component therapy if required. Because the goal of the fluid replacement is to restore intravascular volume, it is necessary to administer fluids that will remain in the intravascular compartment to avoid fluid shifts from the intravascular compartment into the intracellular compartment. Table 15-3 summarizes the fluids commonly used in the treatment of shock.

As discussed earlier, crystalloid solutions such as lactated Ringer's solution or 0.9% sodium chloride solution are commonly used to treat hypovolemic shock as large amounts of fluid must be administered to restore intravascular volume. If hypovolemia is primarily due to blood loss, the American College of Surgeons recommends administration of 3 mL of crystalloid solution for each milliliter of estimated blood loss. This is referred to as the 3:1 rule (American College of Surgeons, 2006). Colloid solutions (eg, albumin, hetastarch) may also be used. Dextran is not indicated if the cause of the hypovolemic shock is hemorrhage, because it interferes with platelet aggregation.

Blood products, which are also colloids, may need to be administered, particularly if the cause of the hypovolemic shock is hemorrhage. The decision to give blood is based on the patient's lack of response to only crystalloid resuscitation, the volume of blood lost, the need for hemoglobin to assist with oxygen transport, and the necessity to correct the patient's coagulopathy. It should be noted that research indicates that patients who receive massive blood transfusions to achieve near-normal hemoglobin levels tend to have poorer outcomes than those with low hemoglobin levels (eg, less than 7.0 g/dL) (Dellinger, et al., 2008; Holcomb & Hess, 2006). Packed red blood cells are administered to replenish the patient's oxygen-carrying capacity in conjunction with other fluids that will expand volume. Currently, the need for transfusions is based on the patient's oxygenation needs, which are determined by vital signs, blood gas values, and clinical appearance rather than an arbitrary laboratory value. An area of active research is the development of synthetic forms of blood (ie, compounds capable of carrying oxygen in the same way that blood does) as potential alternatives to blood component therapy.

Redistribution of Fluid

In addition to administering fluids to restore intravascular volume, positioning the patient properly assists fluid redistribution. A modified Trendelenburg position (Fig. 15-4) is recommended in hypovolemic shock. Elevation of the legs promotes the return of venous blood. A full Trendelenburg position makes breathing difficult and does not increase BP or cardiac output (Bridges & Jarquin-Valdivia, 2005).

Table 15-3 FLUID REPLACEMENT IN SHOCK

Deliver a minimum of 20 mL/kg of crystalloid (or colloid equivalent).

Fluids	Advantages	Disadvantages
Crystalloids		
0.9% sodium chloride (normal saline solution)	Widely available, inexpensive	Requires large volume of infusion; can cause hypernatremia, pulmonary edema, abdominal compartment syndrome
Lactated Ringer's	Lactate ion helps buffer metabolic acidosis	Requires large volume of infusion; can cause metabolic acidosis, pulmonary edema, abdominal compartment syndrome
Hypertonic saline (3%)	Small volume needed to restore intravascular volume	Danger of hypernatremia and cardiovascular compromise from rapid fluid shifts
Colloids		
Albumin (5%, 25%)	Rapidly expands plasma volume	Expensive; requires human donors; limited supply; can cause heart failure
Dextran	Synthetic plasma expander	Interferes with platelet aggregation; not recommended for hemorrhagic shock
Hetastarch	Synthetic plasma expander	Prolongs bleeding and clotting times

Pharmacologic Therapy

If fluid administration fails to reverse hypovolemic shock, then vasoactive medications that prevent cardiac failure are given. Medications are also administered to reverse the cause of the dehydration. For example, insulin is administered if dehydration is secondary to hyperglycemia, desmopressin (DDAVP) is administered for diabetes insipidus, antidiarrheal agents for diarrhea, and antiemetic medications for vomiting.

Nursing Management

Primary prevention of shock is an essential focus of nursing care. Hypovolemic shock can be prevented in some instances by closely monitoring patients who are at risk for fluid deficits and assisting with fluid replacement before intravascular volume is depleted. In other circumstances, nursing care focuses on assisting with treatment targeted at the cause of the shock and restoring intravascular volume.

General nursing measures include ensuring safe administration of prescribed fluids and medications and documenting their administration and effects. Another important nursing role is monitoring for complications and side effects of treatment and reporting them promptly.

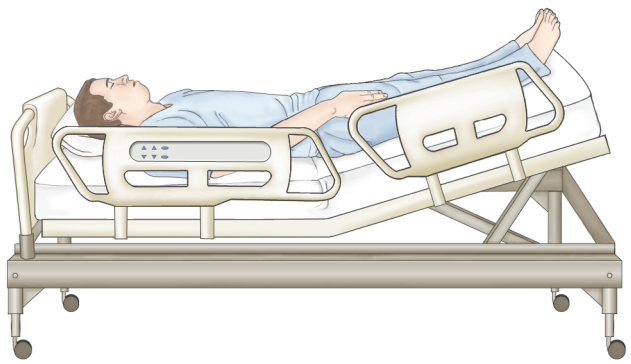

Figure 15-4 Proper positioning (modified Trendelenburg) for the patient who shows signs of shock. The lower extremities are elevated to an angle of about 20 degrees; the knees are straight, the trunk is horizontal, and the head is slightly elevated.

Administering Blood and Fluids Safely

Administering blood transfusions safely is a vital nursing role. In emergency situations, it is important to acquire blood specimens quickly, to obtain a baseline complete blood count, and to type and cross-match the blood in anticipation of blood transfusions. A patient who receives a transfusion of blood products must be monitored closely for adverse effects (see Chapter 33).

Fluid replacement complications can occur, often when large volumes are administered rapidly. Therefore, the nurse monitors the patient closely for cardiovascular overload, signs of difficulty breathing, and pulmonary edema. The risk of these complications is increased in the elderly and in patients with preexisting cardiac disease. Hemodynamic pressures, vital signs, arterial blood gases, serum lactate levels, hemoglobin and hematocrit levels, and fluid intake and output (I&O) are among the parameters monitored. Temperature should also be monitored closely to ensure that rapid fluid resuscitation does not cause hypothermia. IV fluids may need to be warmed during the administration of large volumes. Physical assessment focuses on observing the jugular veins for distention and monitoring jugular venous pressure. Jugular venous pressure is low in hypovolemic shock; it increases with effective treatment and is significantly increased with fluid overload and heart failure. The nurse must monitor cardiac and respiratory status closely and report changes in BP, pulse pressure, CVP, heart rate and rhythm, and lung sounds to the physician.

Implementing Other Measures

Oxygen is administered to increase the amount of oxygen carried by available hemoglobin in the blood. A patient who is confused may feel apprehensive with an oxygen mask or cannula in place, and frequent explanations about the need for the mask may reduce some of the patient's fear and anxiety. Simultaneously, the nurse must direct efforts to the safety and comfort of the patient.

Cardiogenic Shock

Cardiogenic shock occurs when the heart's ability to contract and to pump blood is impaired and the supply of oxygen is inadequate for the heart and tissues. The causes of cardiogenic shock are known as either coronary or noncoronary. Coronary cardiogenic shock is more common than noncoronary cardiogenic shock and is seen most often in patients with acute myocardial infarction (MI) resulting in damage to a significant portion of the left ventricular myocardium (Aymong, Ramanathan & Buller, 2007). Patients who experience an anterior wall MI are at greatest risk for cardiogenic shock because of the potentially extensive damage to the left ventricle caused by occlusion of the left anterior descending coronary artery. Noncoronary causes of cardiogenic shock are related to conditions that stress the myocardium (eg, severe hypoxemia, acidosis, hypoglycemia, hypocalcemia, and tension pneumothorax) as well as conditions that result in ineffective myocardial function (eg, cardiomyopathies, valvular damage, cardiac tamponade, dysrhythmias).

Pathophysiology

In cardiogenic shock, cardiac output, which is a function of both stroke volume and heart rate, is compromised. When stroke volume and heart rate decrease or become erratic, BP falls and tissue perfusion is reduced. Blood supply for tissues and organs and for the heart muscle itself is inadequate, resulting in impaired tissue perfusion. Because impaired tissue perfusion weakens the heart and impairs its ability to pump, the ventricle does not fully eject its volume of blood at systole. As a result, fluid accumulates in the lungs. This sequence of events can occur rapidly or over a period of days (Fig. 15-5).

Clinical Manifestations

Patients in cardiogenic shock may experience the pain of angina, develop dysrhythmias, complain of fatigue, express feelings of doom, and show signs of hemodynamic instability.

Medical Management

The goals of medical management in cardiogenic shock are to limit further myocardial damage and preserve the healthy myocardium and to improve the cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both (Aymong, et al., 2007; Iakobishvili & Hasdai, 2007; Mann & Nolan, 2006). In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demands.

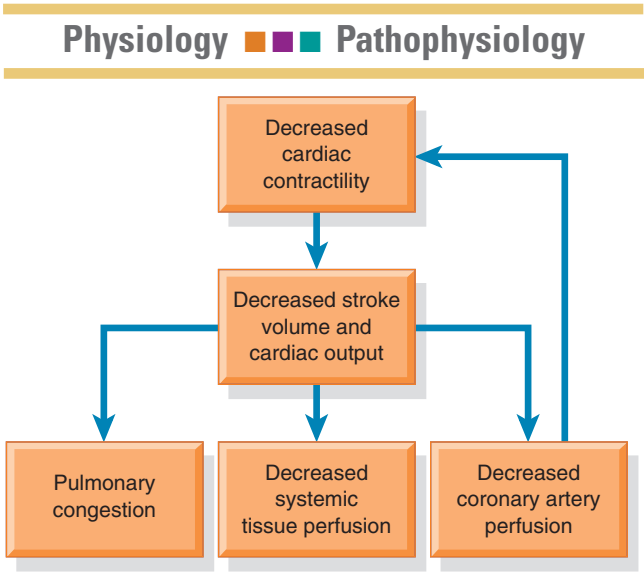

Figure 15-5 Pathophysiologic sequence of events in cardiogenic shock.

Correction of Underlying Causes

As with all forms of shock, the underlying cause of cardiogenic shock must be corrected. It is necessary first to treat the oxygenation needs of the heart muscle to ensure its continued ability to pump blood to other organs. In the case of coronary cardiogenic shock, the patient may require thrombolytic therapy, a percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG) surgery, intraaortic balloon pump therapy, or some combination of these treatments. In the case of noncoronary cardiogenic shock, interventions focus on correcting the underlying cause, such as replacement of a faulty cardiac valve, correction of a dysrhythmia, correction of acidosis and electrolyte disturbances, or treatment of the tension pneumothorax.

Initiation of First-Line Treatment

Oxygenation

In the early stages of shock, supplemental oxygen is administered by nasal cannula at a rate of 2 to 6 L/min to achieve an oxygen saturation exceeding 90%. Monitoring of arterial blood gas values and pulse oximetry values helps determine whether the patient requires a more aggressive method of oxygen delivery.

Pain Control

If a patient experiences chest pain, IV morphine is administered for pain relief. In addition to relieving pain, morphine dilates the blood vessels. This reduces the workload of the heart by both decreasing the cardiac filling pressure (preload) and reducing the pressure against which the heart muscle has to eject blood (afterload). Morphine also decreases the patient's anxiety.

Hemodynamic Monitoring

Hemodynamic monitoring is initiated to assess the patient's response to treatment. In many institutions, this is performed in the intensive care unit (ICU), where an arterial line can be inserted. The arterial line enables accurate and

continuous monitoring of BP and provides a port from which to obtain frequent arterial blood samples without having to perform repeated arterial punctures. A multilumen pulmonary artery catheter is inserted to allow measurement of the pulmonary artery pressures, myocardial filling pressures, cardiac output, and pulmonary and systemic resistance. For more information, see Chapter 30.

Laboratory Marker Monitoring

Laboratory markers for ventricular dysfunction (eg, BNP) and cardiac enzyme levels (CK-MB and cTn-I) are measured, and serial 12-lead ECGs are obtained to assess the degree of myocardial damage. Continuous ECG and ST-segment monitoring is also used to closely monitor the patient for ischemic changes.

Fluid Therapy

Appropriate fluid administration is also necessary in the treatment of cardiogenic shock. Administration of fluids must be monitored closely to detect signs of fluid overload. Incremental IV fluid boluses are cautiously administered to determine optimal filling pressures for improving cardiac output.

NURSING ALERT

A fluid bolus should never be given rapidly, because rapid fluid administration in patients with cardiac failure may result in acute pulmonary edema.

Pharmacologic Therapy

Vasoactive medication therapy consists of multiple pharmacologic strategies to restore and maintain adequate cardiac output. In coronary cardiogenic shock, the aims of vasoactive medication therapy are improved cardiac contractility, decreased preload and afterload, and stabilized heart rate and rhythm.

Because improving contractility and decreasing cardiac workload are opposing pharmacologic actions, two types of medications may be administered in combination: inotropic agents and vasodilators. Inotropic medications increase cardiac output by mimicking the action of the sympathetic nervous system, activating myocardial receptors to increase myocardial contractility (inotropic action) or increasing the heart rate (chronotropic action). These agents may also enhance vascular tone, increasing preload. Vasodilators are used primarily to decrease afterload, reducing the workload of the heart and the oxygen demand. Vasodilators also decrease preload. Medications commonly combined to treat cardiogenic shock include dobutamine, nitroglycerin, and dopamine (see Table 15-2).

Dobutamine. Dobutamine produces inotropic effects by stimulating myocardial beta-receptors, increasing the strength of myocardial activity and improving cardiac output. Myocardial alpha-adrenergic receptors are also stimulated, resulting in decreased pulmonary and systemic vascular resistance (decreased afterload). Dobutamine enhances the strength of cardiac contraction, improving stroke volume ejection and overall cardiac output (Iakobishvili & Hasdai, 2007; Mann & Nolan, 2006).

Nitroglycerin. IV nitroglycerin in low doses acts as a venous vasodilator and therefore reduces preload. At higher doses, nitroglycerin causes arterial vasodilation and therefore reduces afterload as well. These actions, in combination with dobutamine, increase cardiac output while minimizing cardiac workload. In addition, vasodilation enhances blood flow to the myocardium, improving oxygen delivery to the weakened heart muscle (Iakobishvili & Hasdai, 2007).

Dopamine. Dopamine is a sympathomimetic agent that has varying vasoactive effects depending on the dosage. It may be used with dobutamine and nitroglycerin to improve tissue perfusion. Doses of 2 to 8 μg/kg/min improve contractility (inotropic action), slightly increase the heart rate (chronotropic action), and may increase cardiac output. Doses that are higher than 8 μg/kg/min predominantly cause vasoconstriction, which increases afterload and thus increases cardiac workload. Because this effect is undesirable in patients with cardiogenic shock, dopamine doses must be carefully titrated.

Low-dose dopamine (ie, 0.5 to 3.0 μg/kg/min) neither improves renal flow, changes the need for renal support, nor reduces mortality (Freidrich, Adhikari, Herridge, et al., 2005; Iakobishvili & Hasdai, 2007). Thus, low-dose dopamine is no longer recommended. However, some patients respond to lower dosages of dopamine for its inotropic effects (Friedrich, et al., 2005).

In severe metabolic acidosis, which occurs in the later stages of shock, the effectiveness of dopamine is diminished. To maximize the effectiveness of any vasoactive agent, metabolic acidosis must first be corrected (Dellinger, et al., 2008).

Other Vasoactive Medications. Additional vasoactive agents that may be used in managing cardiogenic shock include norepinephrine, epinephrine, milrinone, vasopressin, and phenylephrine. Each of these medications stimulates different receptors of the sympathetic nervous system. A combination of these medications may be prescribed, depending on the patient's response to treatment. All vasoactive medications have adverse effects, making specific medications more useful than others at different stages of shock. Diuretics such as furosemide may be administered to reduce the workload of the heart by reducing fluid accumulation (see Table 15-2).

Antiarrhythmic Medications. Multiple factors, such as hypoxemia, electrolyte imbalances, and acid–base imbalances, contribute to serious cardiac dysrhythmias in all patients with shock. In addition, as a compensatory response to decreased cardiac output and BP, the heart rate increases beyond normal limits. This impedes cardiac output further by shortening diastole and thereby decreasing the time for ventricular filling. Consequently, antiarrhythmic medications are required to stabilize the heart rate. For a full discussion of cardiac dysrhythmias as well as commonly prescribed medications, see Chapter 27. General principles regarding the administration of vasoactive medications are discussed later in this chapter.

Mechanical Assistive Devices

If cardiac output does not improve despite supplemental oxygen, vasoactive medications, and fluid boluses, mechanical assistive devices are used temporarily to improve the heart's ability to pump. Intra-aortic balloon counterpulsation is one means of providing temporary circulatory

assistance (see Chapter 30). Other means of mechanical assistance include left and right ventricular assist devices (VADs) and total temporary artificial hearts (see Chapters 29 and 30). VADs are utilized frequently as bridge therapy to either recovery or heart transplantation. Another short-term means of providing cardiac or pulmonary support to the patient in cardiogenic shock is through an extracorporeal device similar to the cardiopulmonary bypass (CPB) system used in open-heart surgery (see Chapter 28). CPB is used only in emergency situations until definitive treatment, such as heart transplantation, can be initiated.

Nursing Management

Preventing Cardiogenic Shock

Identifying at-risk patients early, promoting adequate oxygenation of the heart muscle, and decreasing cardiac workload can prevent cardiogenic shock. This can be accomplished by conserving the patient's energy, promptly relieving angina, and administering supplemental oxygen. Often, however, cardiogenic shock cannot be prevented. In such instances, nursing management includes working with other members of the health care team to prevent shock from progressing and to restore adequate cardiac function and tissue perfusion.

Monitoring Hemodynamic Status

A major role of the nurse is monitoring the patient's hemodynamic and cardiac status. Arterial lines and ECG monitoring equipment must be well maintained and functioning properly. The nurse anticipates the medications, IV fluids, and equipment that might be used and is ready to assist in implementing these measures. Changes in hemodynamic, cardiac, and pulmonary status and laboratory values are documented and reported promptly. In addition, adventitious breath sounds, changes in cardiac rhythm, and other abnormal physical assessment findings are reported immediately.

Administering Medications and Intravenous Fluids

The nurse plays a critical role in the safe and accurate administration of IV fluids and medications. Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of blood and fluid in the pulmonary tissues. The nurse documents and records medications and treatments that are administered as well as the patient's response to treatment.

The nurse must be knowledgeable about the desired effects as well as the side effects of medications. For example, it is important to monitor the patient for decreased BP after administering morphine or nitroglycerin. Patients receiving thrombolytic therapy must be monitored for bleeding. Arterial and venous puncture sites must be observed for bleeding, and pressure must be applied at the sites if bleeding occurs. Neurologic assessment is essential after the administration of thrombolytic therapy to assess for the potential complication of cerebral hemorrhage associated with this therapy. IV infusions must be observed closely because tissue necrosis and sloughing may occur if vasopressor medications infiltrate the tissues. It is necessary to monitor urine output, BUN, and serum creatinine levels to detect decreased renal function secondary to the effects of cardiogenic shock or its treatment.

Maintaining Intra-Aortic Balloon Counterpulsation

The nurse plays a critical role in caring for the patient receiving intra-aortic balloon counterpulsation (see Chapter 30). The nurse makes ongoing timing adjustments of the balloon pump to maximize its effectiveness by synchronizing it with the cardiac cycle. The patient is at risk for circulatory compromise to the leg on the side where the catheter for the balloon has been inserted; therefore, the nurse must check the neurovascular status of the lower extremities frequently.

Enhancing Safety and Comfort

Throughout care, the nurse must take an active role in safeguarding the patient, enhancing comfort, and reducing anxiety. This includes administering medication to relieve chest pain, preventing infection at the multiple arterial and venous line insertion sites, protecting the skin, and monitoring respiratory and renal function. Proper positioning of the patient promotes effective breathing without decreasing BP and may also increase patient comfort while reducing anxiety.

Brief explanations about procedures that are being performed and the use of comforting touch often provide reassurance to the patient and family. The family is usually anxious and benefits from opportunities to see and talk to the patient. Explanations of treatments and the patient's responses are often comforting to family members.

Circulatory Shock

Circulatory shock occurs when blood volume pools in peripheral blood vessels. This abnormal displacement of blood volume causes a relative hypovolemia because not enough blood returns to the heart, which leads to inadequate tissue perfusion. The ability of the blood vessels to constrict helps return the blood to the heart. The vascular tone is determined both by central regulatory mechanisms, as in BP regulation, and by local regulatory mechanisms, as in tissue demands for oxygen and nutrients. Therefore, circulatory shock can be caused either by a loss of sympathetic tone or by release of biochemical mediators from cells.

The varied mechanisms leading to the initial vasodilation in circulatory shock provide the basis for the further subclassification of shock into three types: septic shock, neurogenic shock, and anaphylactic shock. These types of circulatory shock cause variations in the pathophysiologic chain of events and are explained here separately. In all types of circulatory shock, massive arterial and venous dilation promotes peripheral pooling of blood. Arterial dilation reduces systemic vascular resistance. Initially, cardiac output can be high, both from the reduction in afterload (systemic vascular resistance) and from the heart muscle's increased effort to maintain perfusion despite the incompetent vasculature. Pooling of blood in the periphery results in decreased venous return. Decreased venous return results in decreased stroke volume and decreased cardiac output. Decreased cardiac output, in turn, causes decreased BP and ultimately decreased tissue perfusion. Figure 15-6 presents the pathophysiologic sequence of events in circulatory shock.

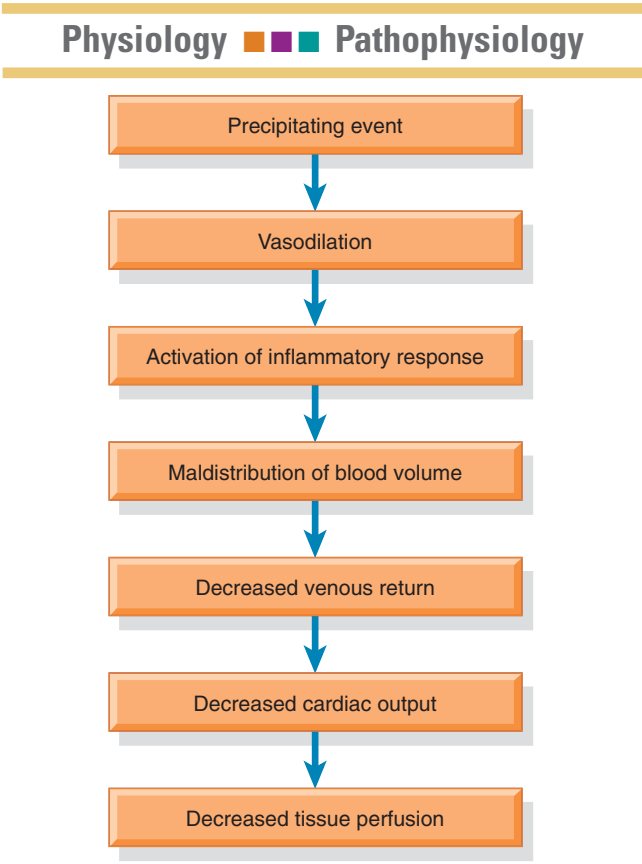

Figure 15-6 Pathophysiologic sequence of events in circulatory shock.

SEPTIC SHOCK

Septic shock, the most common type of circulatory shock, is caused by widespread infection (Chart 15-3). Despite the increased sophistication of antibiotic therapy, the incidence of septic shock has continued to rise during the past 60 years. It is the most common cause of death in noncoronary ICUs in the United States. Each year, severe sepsis affects an estimated 750,000 people in the United States. As the population ages, the rate is expected to rise to 1 million cases a year by 2010 (Surviving Sepsis Campaign, 2007). Finding and aggressively treating the source of infection and quickly restoring tissue perfusion are important interventions that may positively influence the clinical outcome.

Health care–associated infections (infections not incubating at the time of admission to the health care setting) in critically ill patients that may progress to septic shock most frequently originate (in decreasing order of frequency) in the bloodstream (bacteremia), lungs, and urinary tract (urosepsis) (Arugon & Sole, 2006). Other infections include intra-abdominal infections and wound infections. Of increasing concern are bacteremias associated with intravascular catheters and indwelling urinary catheters (Aragon & Sole, 2006; Institute for Healthcare Improvement [IHI], 2007).

Additional risk factors that contribute to the growing incidence of septic shock are the increased use of invasive procedures and indwelling medical devices; the increased number of antibiotic-resistant microorganisms; and the increasingly older population (Aragon & Sole, 2006; King, 2007). Elderly patients are at particular risk for sepsis because of decreased physiologic reserves and an aging immune system (Marik, 2006). Other patients at risk are those undergoing surgical and other invasive procedures; those with malnutrition or immunosuppression; and those with chronic illness such as diabetes mellitus, hepatitis, chronic renal failure, and immunodeficiency disorders (Aragon & Sole, 2006; King, 2007).

The incidence of septic shock can be reduced by using strict infection control practices, beginning with thorough hand-hygiene techniques (Aragon & Sole, 2006). Other interventions include implementing programs to prevent central line infection; early débriding of wounds to remove necrotic tissue; carrying out standard precautions and adhering to infection control practices, including the use of meticulous aseptic technique; and properly cleaning and maintaining equipment.

A significant body of research has been conducted in the past decade in an effort aimed at reducing the morbidity and mortality caused by septic shock and at clarifying the understanding of sepsis and related disorders (Chart 15-4). In 1991, 2003, and again in early 2008, critical care experts and infectious disease experts systematically reevaluated the body of research and provided evidence-based recommendations for the acute management of patients with sepsis and septic shock (Dellinger, et al., 2008; Vincent & Abraham, 2006).

CHART 15-3 *Risk Factors for Circulatory Shock*

Septic Shock
- Immunosuppression
- Extremes of age (<1 yr and >65 yr)
- Malnourishment
- Chronic illness
- Invasive procedures

Anaphylactic Shock
- Penicillin sensitivity
- Transfusion reaction
- Bee sting allergy
- Latex sensitivity
- Severe allergy to some foods or medications

Neurogenic Shock
- Spinal cord injury
- Spinal anesthesia
- Depressant action of medications
- Glucose deficiency

Pathophysiology

Gram-negative bacteria traditionally have been the most commonly implicated microorganisms in septic shock. However, there is also an increased incidence of gram-positive bacterial infections, and gram-positive bacteria currently account for 50% of cases of septic shock (Smith & McInnis, 2007). Other infectious agents, such as viruses and fungi, also can cause septic shock. However, it is estimated that 20% to 30% of patients with severe sepsis may never have an identifiable site of infection (King, 2007).

When microorganisms invade body tissues, patients exhibit an immune response. This immune response provokes the activation of biochemical cytokines and mediators associated with an inflammatory response and produces a

Chart 15-4 • *Definitions to Promote Recognition and Earlier Treatment of Patients With Sepsis*

Bacteremia: the presence of bacteria in the blood

Infection: the presence of microorganisms that trigger an inflammatory response

Hypotension: a systolic blood pressure <90 mm Hg or a drop in systolic blood pressure of ≥40 mm Hg from the patient's baseline blood pressure

Systemic inflammatory response syndrome (SIRS): a syndrome resulting from a *severe clinical insult* that initiates an overwhelming inflammatory response by the body; clinical signs and symptoms may include
- Temperature >38°C or <36°C (>100.4°F or <96.8°F)
- Heart rate >90 bpm
- Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg
- WBC count >12,000 cells/mm^3, <4000 cells/mm^3, or >10% immature WBC (bands)

Sepsis: a systemic response to *infection;* manifested by two or more of the SIRS criteria as a consequence of documented or presumed infection

Severe sepsis: the presence of signs and symptoms of sepsis associated with organ dysfunction, hypotension, or hypoperfusion; clinical signs and symptoms include those of sepsis as well as
- Lactic acidosis
- Oliguria
- Altered level of consciousness
- Thrombocytopenia and coagulation disorders
- Altered hepatic function

Septic shock: shock associated with sepsis; characterized by symptoms of sepsis plus hypotension and hypoperfusion despite adequate fluid volume replacement

Multiple organ dysfunction syndrome (MODS): the presence of altered function of one or more organs in an acutely ill patient requiring intervention and support of the organs to achieve physiologic functioning required for homeostasis; clinical signs and symptoms may be
- Cardiovascular: hypotension and hypoperfusion
- Respiratory: hypoxemia, hypercarbia, adventitious breath sounds
- Renal: increased creatinine, decreased urine output
- Hematologic: thrombocytopenia, coagulation abnormalities
- Metabolic: lactic acidemia, metabolic acidosis
- Neurologic: altered level of consciousness
- Hepatic: elevated liver function tests, hyperbilirubinemia

From Levy, M. M., Fink, M. P., Marshall, J. C., et al. (2003). 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Critical Care Medicine, 31*(4), 1250–1256; and Dellinger, R. P., Levy, M. M., Carlet, J. M., et al. (2008). Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Critical Care Medicine, 36*(1), 296–327.

complex cascade of physiologic events that leads to poor tissue perfusion. Increased capillary permeability, which leads to fluid seeping from the capillaries, and vasodilation are two such effects that interrupt the ability of the body to provide adequate perfusion, oxygen, and nutrients to the tissues and cells. In addition, proinflammatory and anti-inflammatory cytokines released during the inflammatory response activate the coagulation system, which begins to form clots whether or not bleeding is present. The imbalance of the inflammatory response and the clotting and fibrinolysis cascades are considered critical elements of the devastating physiologic progression that occurs in patients with severe sepsis.

Sepsis is an evolving process, with neither clearly definable clinical signs and symptoms nor predictable progression. Initial physiologic changes are subtle. In the early stage of septic shock, BP may remain within normal limits, or the patient may be hypotensive but responsive to fluids. The heart rate increases, progressing to tachycardia. Hyperthermia and fever, with warm, flushed skin and bounding pulses, is evident. The respiratory rate is elevated. Urinary output may remain at normal levels or decrease. GI status may be compromised, as evidenced by nausea, vomiting, diarrhea, or decreased bowel sounds. Signs of hypermetabolism include increased serum glucose and insulin resistance. Subtle changes in mental status, such as confusion or agitation, may be present. The lactate level is elevated because of the maldistribution of blood. Inflammatory markers such as white blood cell counts and C-reactive protein are also elevated (King, 2007).

As sepsis progresses, tissues become less perfused and acidotic, compensation begins to fail, and the patient begins to show signs of organ dysfunction. The cardiovascular system also begins to fail, the BP does not respond to fluid resuscitation and vasoactive agents, and signs of end-organ damage are evident (eg, renal failure, pulmonary failure, hepatic failure). As sepsis progresses to septic shock, the BP drops, and the skin becomes cool, pale, and mottled. Temperature may be normal or below normal. Heart and respiratory rates remain rapid. Urine production ceases, and multiple organ dysfunction progressing to death occurs.

Systemic inflammatory response syndrome (SIRS) presents clinically like sepsis and is part of the initial continuum of sepsis. The physiologic presentation of SIRS is similar to sepsis, except there is no identifiable source of infection (Dellinger, et al., 2008; King, 2007). SIRS stimulates an overwhelming inflammatory immunologic and hormonal response similar to that seen in septic patients. Any overwhelming insult stimulates SIRS and may progress to sepsis. Therefore, despite an absence of infection, antibiotic agents may still be administered because of the possibility of unrecognized infection. Additional therapies directed to support patients with SIRS are similar to those for sepsis. If the inflammatory process progresses, septic shock may develop.

Medical Management

Current treatment of sepsis and septic shock involves identification and elimination of the cause of infection. Current goals are to identify and treat patients in early sepsis within 6 hours to optimize patient outcome (Otero, et al., 2006; Rivers, et al, 2005). Several screening tools can be used to help identify patients with severe sepsis. Chart 15-5 provides key elements that may help identify patients with sepsis and guide interventions in the treatment of severe sepsis and

Chart 15-5 • *Early Identification and Treatment of Patients with Sepsis and Severe Sepsis*

Questions to ask:
Does the patient meet criteria for systemic inflammatory response syndrome (SIRS) (see Chart 15-4)?
Does the patient have signs or symptoms of infection?
- Positive blood cultures
- Currently receiving antibiotic or antifungal therapy
- Examination or chest x-ray suggestive of pneumonia
- Suspected infected wound, abdomen, urine, or other source of infection

Does the patient have signs of acute organ dysfunction?
- Cardiovascular: systolic BP <90 mm Hg or mean arterial pressure (MAP) <65 mm Hg, or drop in systolic BP >40 mm Hg from baseline BP
  - Is hypotension responsive to fluid resuscitation, or is vasopressor support needed?
  - Is the serum lactate >4 mmol/L?
- Respiratory: respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg
  - Is increasing oxygen or mechanical ventilator support needed?
- Renal: urine output <0.5 mL/kg/h
- Hematologic: laboratory analysis and signs and symptoms of coagulopathies
- Metabolic: insulin resistance, metabolic acidosis, or serum lactate >4 mmol/L
- Hepatic: elevated liver function tests or hyperbilirubinemia
- Central nervous system: changes in level of consciousness ranging from agitation to coma

Early interventions:
- Aggressive fluid resuscitation with 20 mL/kg/h of crystalloid (or colloid equivalent)
  - Give fluids to achieve a target central venous pressure of 8 to 12 mm Hg, MAP >65 mm Hg, urine output >0.5 mL/kg/h, and an $Sc\bar{v}O_2$ >70%
  - Vasopressor agents are used if fluid resuscitation does not restore an effective blood pressure and cardiac output
- Obtain blood, sputum, urine, and wound cultures and administer broad-spectrum antibiotics
- Support the respiratory system with mechanical ventilation
- Transfuse with packed red blood cells when hemoglobin is <7 g/dL
- Provide adequate IV sedation; avoid the use of neuromuscular blockade agents when possible
- Control serum glucose <150 mg/dL with IV insulin therapy
- Implement interventions and medications to prevent deep vein thrombosis and stress ulcer prophylaxis
- Consider IV steroid therapy if the patient is not responding to fluid resuscitation and vasopressor therapy
- Consider administration of recombinant human activated protein C (drotrecogin alfa) in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death

From Dellinger, R. P., Levy, M. M., Carlet, J. M., et al. (2008). Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Critical Care Medicine, 36*(1), 296–327; and Rivers, E. P., McIntyre, L., Morro, D. C., et al. (2005). Early and innovative interventions for severe sepsis and septic shock: Taking advantage of a window of opportunity. *Canadian Medical Association Journal, 173*(9), 1054–1065.

septic shock (Dellinger, et al., 2008; King, 2007; Otero, et al., 2006; Surviving Sepsis Campaign, 2007).

Rapid identification of the infectious source is also a critical element in management. Specimens of blood, sputum, urine, wound drainage, and tips of invasive catheters are collected for culture using aseptic technique. Any potential routes of infection must be identified and treated. IV lines are removed and reinserted at alternate sites. Antibiotic-coated IV central lines may be inserted to decrease the risk of invasive line-related bacteremia in high-risk patients (King, 2007). If possible, urinary catheters are removed. Any abscesses are drained, and necrotic areas are débrided.

Research efforts are focusing on better identification and early aggressive treatment of patients with sepsis, rapid and effective restoration of tissue perfusion, evaluation and treatment of the patient's immune response, and treatment of dysregulation of the coagulation system that seems to occur with severe sepsis (Remick, 2007a).

Fluid Replacement Therapy

Fluid replacement must be instituted to correct the tissue hypoperfusion that results from the incompetent vasculature and the inflammatory response. Reestablishing tissue perfusion through aggressive fluid resuscitation is the key to management of severe sepsis and septic shock (Dellinger, et al., 2008; Otero, et al., 2006). See Chart 15-5 for a list of the treatment endpoints of fluid resuscitation.

Pharmacologic Therapy

If the identity of the infecting organism is unknown, broad-spectrum antibiotic agents are started until culture and sensitivity reports are received (Dellinger, et al., 2008; Smith & McInnis, 2007), at which time the antibiotic agents may be changed to agents that are more specific to the infecting organism and less toxic to the patient.

Treatment of the dysregulation of the coagulation system that occurs in patients with severe sepsis and septic shock remains controversial. Nonetheless, current guidelines recommend the administration of recombinant human activated protein C (rhAPC; drotrecogin alfa [Xigris]) to patients with end-organ dysfunction and high risk of death (Dellinger, et al., 2008). In sepsis, an imbalance in proinflammatory mediators activates the coagulation cascade and deposits microthrombi that alter tissue perfusion. Drotrecogin alfa (Xigris) acts as an antithrombotic, anti-inflammatory, and profibrinolytic agent. Drotrecogin alfa acts as an anti-inflammatory cytokine, it stimulates fibrinolysis, restoring balance in the coagulation–anticoagulation homeostatic process of the body's inflammatory response to injury and infection.

Drotrecogin alfa has provided a significant breakthrough in the successful pharmacologic treatment of patients with sepsis. The medication should be administered as early as possible in the sequence of pathophysiologic events of sepsis. It is not without side effects, bleeding being the most common serious effect. Stopping the medication reduces

the risk of bleeding. The patient should be evaluated with regard to the relative risk of bleeding versus the potential benefit from the medication. Drotrecogin alfa is contraindicated in patients with active internal bleeding, recent hemorrhagic stroke, intracranial surgery, or head injury.

Nutritional Therapy

Aggressive nutritional supplementation is critical in the management of septic shock, because malnutrition further impairs the patient's resistance to infection. Nutritional supplementation should be initiated within the first 24 hours after ICU admission (Stapleton, et al., 2007), and continuous infusions of insulin are used to control hyperglycemia (Dellinger, et al., 2008; Vanhorebeek, et al., 2007). Enteral feedings are preferred to the parenteral route because of the increased risk of iatrogenic infection associated with IV catheters; however, enteral feedings may not be possible if decreased perfusion to the GI tract reduces peristalsis and impairs absorption.

Nursing Management

Nurses caring for patients in any setting must keep in mind the risks of sepsis and the high mortality rate associated with sepsis, severe sepsis, and septic shock. All invasive procedures must be carried out with aseptic technique after careful hand hygiene. In addition, IV lines, arterial and venous puncture sites, surgical incisions, traumatic wounds, urinary catheters, and pressure ulcers must be monitored for signs of infection. Nurses need to identify patients who are at particular risk for sepsis and septic shock (ie, elderly and immunosuppressed patients and those with extensive trauma, burns, or diabetes), keeping in mind that these high-risk patients may not develop typical or classic signs of infection and sepsis. For example, confusion may be the first sign of infection and sepsis in elderly patients.

When caring for a patient with septic shock, the nurse collaborates with other members of the health care team to identify the site and source of sepsis and the specific organisms involved. The nurse often obtains appropriate specimens for culture and sensitivity.

Elevated body temperature (hyperthermia) is common with sepsis and raises the patient's metabolic rate and oxygen consumption. Fever is one of the body's natural mechanisms for fighting infections. Therefore, elevated temperatures may not be treated unless they reach dangerous levels (more than 40°C [104°F]) or unless the patient is uncomfortable. Efforts may be made to reduce the temperature by administering acetaminophen or applying a hypothermia blanket. During these therapies, the nurse monitors the patient closely for shivering, which increases oxygen consumption. Efforts to increase comfort are important if the patient experiences fever, chills, or shivering.

The nurse administers prescribed IV fluids and medications, including antibiotic agents and vasoactive medications, to restore vascular volume. Because of decreased perfusion, serum concentrations of antibiotic agents that are normally cleared by the kidneys and liver may increase and produce toxic effects. Therefore, the nurse monitors blood levels (antibiotic agents, BUN, creatinine, white blood cell count, hemoglobin, hematocrit, platelet levels, coagulation studies) and reports changes to the physician. As with other types of shock, the nurse monitors the patient's hemodynamic status, fluid intake and output, and nutritional status. Daily weights and close monitoring of serum albumin and prealbumin levels help determine the patient's protein requirements.

NEUROGENIC SHOCK

In **neurogenic shock**, vasodilation occurs as a result of a loss of balance between parasympathetic and sympathetic stimulation. Sympathetic stimulation causes vascular smooth muscle to constrict, and parasympathetic stimulation causes vascular smooth muscle to relax or dilate. The patient experiences a predominant parasympathetic stimulation that causes vasodilation lasting for an extended period, leading to a relative hypovolemic state. However, blood volume is adequate, because the vasculature is dilated; the blood volume is displaced, producing a hypotensive (low BP) state. The overriding parasympathetic stimulation that occurs with neurogenic shock causes a drastic decrease in the patient's systemic vascular resistance and bradycardia. Inadequate BP results in the insufficient perfusion of tissues and cells that is common to all shock states.

Neurogenic shock can be caused by spinal cord injury, spinal anesthesia, or other nervous system damage (see Chart 15-3). It may also result from the depressant action of medications or from lack of glucose (eg, insulin reaction or shock). Neurogenic shock may have a prolonged course (spinal cord injury) or a short one (syncope or fainting). Normally, during states of stress, the sympathetic stimulation causes the BP and heart rate to increase. In neurogenic shock, the sympathetic system is not able to respond to body stressors. Therefore, the clinical characteristics of neurogenic shock are signs of parasympathetic stimulation. It is characterized by dry, warm skin rather than the cool, moist skin seen in hypovolemic shock. Another characteristic is hypotension with bradycardia, rather than the tachycardia that characterizes other forms of shock.

Medical Management

Treatment of neurogenic shock involves restoring sympathetic tone, either through the stabilization of a spinal cord injury or, in the instance of spinal anesthesia, by positioning the patient properly. Specific treatment depends on the cause of the shock. Further discussion of management of patients with a spinal cord injury is presented in Chapter 63. If hypoglycemia (insulin shock) is the cause, glucose is rapidly administered (see Chapter 41).

Nursing Management

It is important to elevate and maintain the head of the bed at least 30 degrees to prevent neurogenic shock when a patient receives spinal or epidural anesthesia. Elevation of the head helps prevent the spread of the anesthetic agent up the spinal cord. In suspected spinal cord injury, neurogenic shock may be prevented by carefully immobilizing the patient to prevent further damage to the spinal cord.

Nursing interventions are directed toward supporting cardiovascular and neurologic function until the usually transient episode of neurogenic shock resolves. Applying

anti-embolism stockings and elevating the foot of the bed may minimize pooling of blood in the legs. Pooled blood increases the risk of thrombus formation. Therefore, the nurse must check the patient daily for any lower extremity pain, redness, tenderness, and warmth. If the patient complains of pain and objective assessment of the calf is suspicious, the patient should be evaluated for deep vein thrombosis. Administration of heparin or low-molecular-weight heparin (Lovenox) as prescribed, application of anti-embolism stockings, or use of pneumatic compression of the legs may prevent thrombus formation. Passive range of motion of the immobile extremities helps promote circulation.

A patient who has experienced a spinal cord injury may not report pain caused by internal injuries. Therefore, in the immediate postinjury period, the nurse must monitor the patient closely for signs of internal bleeding that could lead to hypovolemic shock.

ANAPHYLACTIC SHOCK

Anaphylactic shock occurs rapidly and is life-threatening. Because anaphylactic shock occurs in patients already exposed to an antigen and who have developed antibodies to it, it can often be prevented. Patients with known allergies should understand the consequences of subsequent exposure to the antigen and should wear medical identification that lists their sensitivities. This could prevent inadvertent administration of a medication that would lead to anaphylactic shock. In addition, patients and families need instruction about emergency use of medications for treatment of anaphylaxis.

Anaphylactic shock is caused by a severe allergic reaction when patients who have already produced antibodies to a foreign substance (antigen) develop a systemic antigen–antibody reaction (see Chart 15-3). This process requires that the patient has previously been exposed to the substance. An antigen–antibody reaction provokes mast cells to release potent vasoactive substances, such as histamine or bradykinin, causing widespread vasodilation and capillary permeability. Characteristics of severe anaphylaxis usually include rapid onset of hypotension, neurologic compromise, respiratory distress, and cardiac arrest (Brown, 2007).

Medical Management

Treatment of anaphylactic shock requires removing the causative antigen (eg, discontinuing an antibiotic agent), administering medications that restore vascular tone, and providing emergency support of basic life functions. Epinephrine is given for its vasoconstrictive action. Diphenhydramine (Benadryl) is administered to reverse the effects of histamine, thereby reducing capillary permeability. These medications are given intravenously. Nebulized medications, such as albuterol (Proventil), may be given to reverse histamine-induced bronchospasm.

If cardiac arrest and respiratory arrest are imminent or have occurred, cardiopulmonary resuscitation is performed. Endotracheal intubation or tracheotomy may be necessary to establish an airway. IV lines are inserted to provide access for administering fluids and medications. Anaphylaxis and specific chemical mediators are discussed further in Chapter 53.

Nursing Management

The nurse has an important role in preventing anaphylactic shock. The nurse must assess all patients for allergies or previous reactions to antigens (eg, medications, blood products, foods, contrast agents, latex) and communicate the existence of these allergies or reactions to others. In addition, the nurse assesses the patient's understanding of previous reactions and steps taken by the patient and family to prevent further exposure to antigens. When new allergies are identified, the nurse advises the patient to wear or carry identification that names the specific allergen or antigen.

When administering any new medication, the nurse observes all patients for allergic reactions. This is especially important with IV medications, including antibiotics. Previous adverse drug reactions increase the risk that the patient will develop an undesirable reaction to a new medication. If the patient reports an allergy to a medication, the nurse must be aware of the risks involved in the administration of similar medications.

At hospital and outpatient diagnostic testing sites, the nurse must identify patients who are at risk for anaphylactic reactions to contrast agents (radiopaque, dyelike substances that may contain iodine) used for diagnostic tests. Patients with a known allergy to iodine or fish and those who have had previous allergic reactions to contrast agents are at high risk. This information must be communicated to the staff at the diagnostic testing site, including x-ray personnel. The nurse must be knowledgeable about the clinical signs of anaphylaxis, must take immediate action if signs and symptoms occur, and must be prepared to begin cardiopulmonary resuscitation if cardiorespiratory arrest occurs.

Community health and home care nurses who administer medications, including antibiotic agents, in the patient's home or other settings must be prepared to administer epinephrine subcutaneously or intramuscularly in the event of an anaphylactic reaction.

After recovery from anaphylaxis, the patient and family require an explanation of the event. Furthermore, the nurse provides instruction about avoiding future exposure to antigens and administering emergency medications to treat anaphylaxis (see Chapter 53).

Multiple Organ Dysfunction Syndrome

Multiple organ dysfunction syndrome (MODS) is altered organ function in acutely ill patients that requires medical intervention to support continued organ function. It is another phase in the progression of shock states. The actual incidence of MODS is difficult to determine, because it develops with acute illnesses that compromise tissue perfusion. Dysfunction of one organ system is associated with 20% mortality, and if more than four organs fail, the mortality may reach 70% (VonRueden, et al., 2008).

Pathophysiology

MODS may be a complication of any form of shock caused by inadequate tissue perfusion. The precise mechanism by which MODS occurs remains unknown. However, MODS

frequently occurs toward the end of the continuum of septic shock when tissue perfusion cannot be effectively restored. It is not possible to predict which patients who experience shock will develop MODS, partly because much of the organ damage occurs at the cellular level and therefore cannot be directly observed or measured. However, a pattern of progressive organ dysfunction and failure typically occurs; organ failure usually begins in the lungs, and cardiovascular instability as well as failure of the hepatic, GI, renal, immunologic, and central nervous systems follow (Abraham & Singer, 2007; VonRueden, et al., 2008). Advanced age, malnutrition, and coexisting disease appear to increase the risk of MODS in acutely ill patients.

Clinical Manifestations

The clinical presentation of MODS is insidious; tissues become hypoperfused at both a microcellular and macrocellular level, eventually causing organ dysfunction that requires intervention to support organ function.

In MODS, the sequence of organ dysfunction varies depending on the patient's primary illness and comorbidities prior to experiencing shock. For simplicity of presentation, the classic pattern is described. Typically, the lungs are the first organs to show signs of dysfunction. The patient experiences progressive dyspnea and respiratory failure requiring intubation and mechanical ventilation (see Chapters 23 and 25). The patient usually remains hemodynamically stable but may require increasing amounts of IV fluids and vasoactive agents to support the BP and cardiac output. Signs of a hypermetabolic state, characterized by hyperglycemia (elevated blood glucose level), hyperlactic acidemia (excess lactic acid in the blood), and increased BUN, are present. The metabolic rate may be 1.5 to 2 times the basal metabolic rate. At this time, there is a severe loss of skeletal muscle mass (autocatabolism) to meet the high energy demands of the body.

After approximately 7 to 10 days, signs of hepatic dysfunction (eg, elevated bilirubin and liver function tests) and renal dysfunction (eg, elevated creatinine and anuria) are evident. As the lack of tissue perfusion continues, the hematologic system becomes dysfunctional, with worsening immunocompromise and increasing risk of bleeding. The cardiovascular system becomes unstable and unresponsive to vasoactive agents, and the patient's neurologic response progresses to a state of unresponsiveness or coma.

The goal of all shock states is to reverse the tissue hypoperfusion and hypoxia. If effective tissue perfusion is restored before organs become dysfunctional, the patient's condition stabilizes. Along the septic shock continuum, the onset of organ dysfunction is an ominous prognostic sign; the more organs that fail, the worse the outcome.

Medical Management

Prevention remains the top priority in managing MODS. Elderly patients are at increased risk for MODS because of the lack of physiologic reserve and the natural degenerative process, especially immune compromise (Marik, 2006). Early detection and documentation of initial signs of infection are essential in managing MODS in elderly patients. Subtle changes in mentation and a gradual rise in temperature are early warning signs. Other patients at risk for MODS are those with chronic illness, malnutrition, immunosuppression, or surgical or traumatic wounds.

If preventive measures fail, treatment measures to reverse MODS are aimed at (1) controlling the initiating event, (2) promoting adequate organ perfusion, and (3) providing nutritional support.

Nursing Management

The general plan of nursing care for patients with MODS is the same as that for patients with septic shock. Primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted. Providing information and support to family members is a critical role of the nurse. It is important that the health care team address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes (see Chapter 17).

Promoting Communication

Nurses should encourage frequent and open communication about treatment modalities and options to ensure that the patient's wishes regarding medical management are met. For patients who survive MODS, it is essential that they be informed about the goals of rehabilitation and expectations for progress toward these goals, because massive loss of skeletal muscle mass makes rehabilitation a long, slow process. A strong nurse–patient relationship built on effective communication provides needed encouragement during this phase of recovery.

Promoting Home and Community-Based Care

Teaching Patients Self-Care

Patients who experience and survive shock may have been unable to get out of bed for an extended period of time and are likely to have a slow, prolonged recovery. The patient and family are instructed about strategies to prevent further episodes of shock by identifying the factors implicated in the initial episode. In addition, the patient and family require instruction about assessments needed to identify the complications that may occur after the patient is discharged from the hospital. Depending on the type of shock and its management, the patient or family may require instruction about treatment modalities such as emergency administration of medications, IV therapy, parenteral or enteral nutrition, skin care, exercise, and ambulation. The patient and family are also instructed about the need for gradual increases in ambulation and other activity. The need for adequate nutrition is another crucial aspect of teaching.

Continuing Care

Because of the physical toll associated with recovery from shock, patients may be cared for in a long-term care facility or rehabilitation setting after hospital discharge. Alternatively, a referral may be made for home care. The home care nurse assesses the patient's physical status and monitors

recovery. The nurse also assesses the adequacy of treatments that are continued at home and the ability of the patient and family to cope with these treatments. The patient is likely to require close medical supervision until complete recovery occurs. The home care nurse reinforces the importance of continuing medical care and helps the patient and family identify and mobilize community resources.

CRITICAL THINKING EXERCISES

1 A patient with a history of severe osteoarthritis is prescribed glucosamine and chondroitin supplements. The patient's chart states that he has no known drug allergies, but he does have food allergies that include shell fish and avocados. Fifteen minutes after the first dose of the medication is administered, the patient complains of anxiety, shortness of breath, and chest discomfort. He is flushed and visibly uncomfortable. What are your nursing priorities in providing care to this patient? What assessment data do you need to obtain to determine if this patient is experiencing cardiogenic or anaphylactic shock? What nursing interventions and medical treatments would you anticipate for cardiogenic shock? What risks did the patient have that may have increased his likelihood of experiencing anaphylactic shock? In terms of anaphylactic shock, what nursing interventions and medical treatments would you anticipate?

EBP **2** An elderly man with a 16-year history of Parkinson's disease is admitted with sudden, increasing confusion and combative behavior. You know that changes in mental status may be an early sign of sepsis in the elderly. How would you assess this patient for the possibility of sepsis? What risk factors place an older patient at higher risk for sepsis? How would you ensure the accuracy of vital signs and interpretation of vital signs in the older patient experiencing sepsis? What is the evidence base for these risk factors? How would the management of the elderly patient differ from that of a younger patient?

3 A 32-year-old man is admitted with severe pancreatitis. He has a long history of addiction to alcohol and was recently on a "drinking binge." The patient is agitated and exhibiting nervous behavior. His BP is 106/88 mm Hg, heart rate is 126 bpm, respiratory rate is 32 breaths/min, and he has not voided for the past 3 hours. Is the patient most likely experiencing withdrawal from alcohol or a type of shock? Describe the type of shock that poses the greatest risk for this patient. What interventions should you anticipate to prevent the progression of shock or development of MODS? Given the patient's history, what organ(s) is least likely to tolerate prolonged tissue hypoperfusion? What assessment data would you look at to monitor organ dysfunction in this patient?

4 A 23-year-old patient underwent surgical repair of her shoulder. She had spinal anesthesia for the surgery and currently has a patent epidural catheter for pain management. What types of shock are possible in this patient? What therapy directed at prevention or treatment of shock would you anticipate? Describe the rationale for the therapies that you have identified. How would you use the patient's history and symptom presentation to help you identify shock states? Describe likely symptoms and the underlying pathophysiology of the shock state.

The Smeltzer suite offers these additional resources to enhance learning and facilitate understanding of this chapter:

- thePoint online resource, thepoint.lww.com/Smeltzer12E
- Student CD-ROM included with the book
- *Study Guide to Accompany Brunner & Suddarth's Textbook of Medical-Surgical Nursing*
- *Handbook for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*

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**Asterisk indicate nursing research.*

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