Other Acute Problems



An 80-year-old patient is in acute kidney failure and has been diagnosed with multiple organ dysfunction syndrome (MODS). She underwent a right hip replacement 2 days ago and currently has a temperature of 102.6°F.

- What are treatment measures to reverse MODS?
- What patient populations are at increased risk for developing MODS?
- Discuss nursing care of patients with MODS.

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CHAPTER 54

Nursing Management: Shock and Multisystem Failure

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Learning Objectives

After reading this chapter, you will be able to:

1. Describe shock and its underlying pathophysiology.
2. Compare clinical findings of the preshock, shock, and end-organ damage stages of shock.
3. Describe similarities and differences in hypovolemic, cardiogenic, distributive, and obstructive shock states.
4. Identify medical and nursing management priorities in treating patients in shock.
5. Identify vasoactive medications used in treating shock, and describe nursing implications associated with their use.

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Shock is a life-threatening condition with a variety of underlying causes. The progression of shock is neither linear nor predictable. 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 and response are essential to the patient's recovery.

OVERVIEW

Shock can best be defined as a condition in which tissue perfusion is inadequate to deliver oxygen and nutrients to support vital organs and cellular function. This definition differs from more traditional views of shock because it does **not** depend on absolute criteria for parameters, such as blood pressure (BP).

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.

Adequate blood flow to the tissues and cells requires the following components: adequate cardiac pump, effective vasculature and circulatory system, and sufficient blood volume. If one component is impaired, perfusion to the tissues is threatened or compromised. Without treatment, inadequate blood flow to the tissues results in poor delivery of oxygen and nutrients to the cells, cellular starvation, cell death, organ dysfunction progressing to organ failure, and eventual death.

Shock affects all body systems. It may develop rapidly or slowly, depending on the underlying cause. During shock, the body struggles to survive, utilizing a variety of homeostatic, compensatory mechanisms to restore blood flow. Any insult to the body can create a cascade of events resulting in poor tissue perfusion, which means that nurses should be vigilant about watching for signs of shock in all patients, regardless of their primary problem or diagnosis.

PATHOPHYSIOLOGY

Tissue perfusion is determined by cardiac output (CO) and systemic vascular resistance (SVR). CO is the amount of blood pumped by the heart in liters per minute (usual CO is about 4 to 6 L/min but varies greatly depending on the metabolic needs of the body). CO depends on stroke volume, determined by ventricular preload (volume), and heart rate, which is controlled in large part by the autonomic nervous system. Afterload is related closely to, and often equated with, the aortic pressure. It depends on the contractility of the right or left side of the heart and SVR (the resistance to the flow of blood out from the ventricles and a key determinant of aortic pressure). It is helpful to think of SVR as a vise grip on the aorta. If the vise is tightened, the SVR and aortic

BOX 54-1

Classifications of Shock

- **Hypovolemic shock** occurs when there is a decrease in the intravascular volume. Hypovolemia can be the result of an imbalance of intake and output, such as in patients with **dehydration** or **hemorrhage**.
- **Cardiogenic shock** occurs when the heart has an impaired pumping ability; it may be of coronary or noncoronary event origin.
- **Obstructive shock** occurs when there is decreased oxygen delivery due to an obstructive cause, such as pericardial tamponade, tension pneumothorax, PE, or abdominal compartment syndrome.
- **Distributive shock** is caused by alterations in vascular smooth muscle tone, caused by nervous system injury, inflammatory release causing vasodilation, or complications associated with medications such as epidural anesthesia. **Septic shock** is technically considered a "distributive" type of shock. Sepsis is a complex condition, caused by an infection with profound vasodilation.

pressure rise, and now it requires more work for the ventricle to pump blood out. In contrast, if the vise grip is released from the aorta, there is little resistance to the outflow of blood and the work required of the heart muscle is diminished. Conventionally, the primary underlying pathophysiologic process is used to classify the shock state (Box 54-1 provides classifications). When considering possible causes of shock and potential interventions, nurses should remember that the interplay of these two factors (CO and SVR) is what determines the extent of the course of shock and the prognosis of the patient.

In all types of shock, the cells lack an adequate blood supply and are inadequately oxygenated; therefore, they must produce energy through anaerobic metabolism. This results in an acidotic intracellular environment, along with a host of electrolyte derangements. In stress states such as shock, catecholamines, cortisol, glucagon, and inflammatory cytokines and mediators are released, causing hyperglycemia and insulin resistance in an effort to mobilize glucose for cellular metabolism.

Nursing Alert

Tight glycemic control (blood glucose, 80 to 110 mg/dL) was considered to reduce morbidity and mortality of patients who were acutely ill. While a higher blood glucose concentration has been associated with adverse outcomes in a variety of clinical settings, recent research studies reveal inconsistent results, suggesting that, in patients who are critically ill, a less aggressive glycemic target is appropriate (e.g., a blood sugar of less than or equal to 120 mg/dL was an independent risk factor associated with an increased mortality in patients who were nondiabetic) (Chan et al., 2016). The optimal strategy for the management of hyperglycemia (also called stress hyperglycemia) in patients who are critically ill with and without diabetes is unknown; however, maintaining the glucose of 140 to 180 mg/dL avoids marked hyperglycemia while minimizing the risk of iatrogenic hypoglycemia (Stapleton & Heyland, 2016).

As the hypoxemia progresses, cellular metabolism becomes anaerobic, which causes the lactic acid level to rise. Excess levels of lactic acid limit the amount of adenosine triphosphate (ATP) produced, and normal cell function cannot be maintained. Recall ATP which is in every cell is responsible for storing and supplying needed energy to the cell. The impaired Na^+/K^+ and ATP membrane pump function results in intracellular accumulation of sodium and loss of K^+. Cellular edema results from the increased intracellular sodium and increased capillary permeability, further decreasing the CO. As the cell membrane becomes more permeable, allowing electrolytes and fluids to seep out of and into the cell, cell structures, primarily the mitochondria, are damaged; and cell death results.

Pathophysiology Alert

Shock often affects the mitochondria within the cell, which normally function at the lowest oxygen tensions in the body (mitochondrial pO_2 is about 5 mm Hg). However, they consume almost all the oxygen used by the body. More than 95% of aerobic chemical energy comes from mitochondrial work (aka oxidative phosphorylation), and the oxygen-dependent combustion of fuel substrates (primarily fats, amino acids, carbohydrates, ketones) into carbon dioxide (CO_2), water, and ATP. Because of this oxygen dependence, the mitochondria of patients in shock can be referred to as "canaries in the coal mine" since often they are affected first during inadequate tissue perfusion. Historically miners would carry canaries into the mine tunnels since the buildup of gases such as carbon monoxide would kill the canary before killing the miners, thus it was a warning to get out! Similarly, without sufficient oxygen, ATP is generated by potential mitochondrial fuels that are anaerobic. Thus lactic acid accumulates in the blood (Jones & Kline, 2014).

STAGES OF SHOCK

Shock is believed to progress along a continuum. One way to understand the physiologic responses and subsequent clinical signs and symptoms is to divide the continuum into separate stages:

- A *nonprogressive stage* (also called the *compensated stage or preshock*)

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TABLE **54-1 Clinical Findings in Stages of Shock**

	Stage		
Finding	*Nonprogressive stage; Compensated stage* or Preshock	*Progressive stage*; *Uncompensated Stage* or Shock	*Irreversible stage or* End-Organ Dysfunction
Blood pressure	Near normal	Systolic below 80–90 mm Hg; Mean arterial pressure (MAP) less than 65 mm Hg	Requires mechanical or pharmacologic support
Heart rate	Over 100 bpm	100–150 bpm	Erratic or asystole
Respiratory status	Fewer than 20 breaths/min	Rapid, shallow respirations; crackles	May require assisted ventilation
Skin	Cold, clammy	Mottled, petechiae	Jaundice
Urinary output	Mildly decreased	Severely decreased	Anuric, requires dialysis
Mentation	Confusion	Lethargy	Unresponsive
Acid–base balance	Respiratory alkalosis	Metabolic acidosis	Profound acidosis

- A *progressive stage* (also called the *uncompensated stage or shock*)
- An *irreversible stage* (also called *End-organ dysfunction* which results in irreversible organ damage)

The earlier on this continuum that shock is recognized and interventions are initiated, the greater the patient's chance of survival. Table 54-1 compares clinical findings in the different stages of shock.

NONPROGRESSIVE STAGE (COMPENSATED STAGE OR PRESHOCK)

Pathophysiology

In preshock, or the compensatory stage of shock, the BP often remains within normal limits. Compensatory responses, such as vasoconstriction, increased heart rate, and increased contractility of the heart, contribute to maintaining adequate CO and SVR. 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. Thus, tachycardia or a modest change in systemic blood pressure (increase or decrease) may be the only clinical sign of early shock. Adult patients can often compensate for up to a 10% total volume loss through normal homeostatic mechanisms. Conversely, patients who have other medical problems or who take certain medications (such as beta blockers or calcium channel blockers) may have a diminished ability to compensate.

Clinical Manifestations and Assessment

Despite a near-normal BP, the patient may show other clinical signs indicating inadequate organ perfusion. Tachycardia, peripheral vasoconstriction, and anxiety are common. Anxiety, along with feelings of doom, are common signs of shock during this early phase and often are not recognized in patients or attributed to something other than developing shock.

Medical and Nursing Management

At this nonprogressive stage, treatment is directed toward identifying the cause of shock, correcting the underlying disorder, and initiating aggressive measures aimed at supporting the body's compensatory mechanisms. Because compensation cannot be maintained indefinitely, measures such as fluid replacement and medication therapy must be initiated early to maintain an adequate BP and reestablish and maintain adequate tissue perfusion. *Early intervention along the continuum of shock is the key to improving the patient's prognosis.* Therefore, the nurse must assess the patient at risk for shock systematically to recognize the subtle clinical signs of the compensatory stage before the patient progresses to uncompensated shock. Box 54-2 highlights considerations for recognizing shock in older patients.

The role of the nurse at this stage is to monitor the patient's hemodynamic status and promptly report deviations to the provider, to assist in identifying and treating the underlying disorder by continuous in-depth assessment of the patient, to administer prescribed fluids and medications, and to promote patient safety. In assessing tissue perfusion, the nurse specifically observes for changes in level of consciousness, vital signs, urinary output, skin, and laboratory values. Constant reassessment is warranted at this stage. Nurses should try to maintain a calm environment for both the patient's and family's sake and also ensure that assessments and interventions are rapid and ongoing. Providing brief explanations about the diagnostic and treatment procedures, supporting the patient during these procedures, and

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BOX 54-2

Gerontologic Considerations

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.

- During hypovolemic states, medications such as beta-blocking agents (metoprolol [Lopressor]) used to treat hypertension may mask tachycardia, a primary compensatory mechanism to increase CO.
- The aging immune system may not mount a truly febrile response (temperature over 40°C or 104°F), but an increasing trend in body temperature should be addressed.
- The heart does not function well in hypoxemic states, and the aging heart may respond to decreased myocardial oxygenation with arrhythmias that may be misinterpreted as a normal part of the aging process.
- 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.

providing information about their outcomes reduces stress and anxiety and thus promotes 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.

Nursing Alert

For patients experiencing shock, if perfusion does not improve despite aggressive interventions, the nurse recognizes that the body will shunt blood from organs such as the skin, kidneys, and gastrointestinal (GI) tract to the brain and heart 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.

PROGRESSIVE STAGE (UNCOMPENSATED STAGE OR SHOCK)

In the progressive stage, the patient loses the ability to compensate for the insult, infection, or injury. It is during this stage that clinical signs become more obvious.

Pathophysiology

The heart and kidneys are among the first organs to show signs of dysfunction. Up until now on the continuum, the myocardium has kept pace by increasing CO, but this can only go on for a limited time. The body's inability to meet increased oxygen requirements produces ischemia, and biochemical mediators cause myocardial depression. This leads to failure of the cardiac pump, even if the underlying cause of the shock is not of cardiac origin.

The systemic impact of hypotension, and thus hypoperfusion, on the kidneys results in decreased urine output. In addition, the inflammatory response to injury is activated, and proinflammatory and anti-inflammatory cytokines and mediators are released, which, in turn, activate the coagulation system in an effort to reestablish homeostasis. At the same time, acidosis impairs all enzymatic functions, including coagulation. A cascade of progressive organ failure resulting from poor tissue perfusion is illustrated in Figure 54-1. Thus it is common for a systemic inflammatory response, coined SIRS, to occur in patients with shock (see section on Septic Shock, page 1521).

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 cycle ensues. It is believed that the body's appropriate or inappropriate response in this stage of shock may be the primary factor determining the patient's survival.

Clinical Manifestations and Assessment

Clinical manifestations that may be seen include symptomatic tachycardia, dyspnea, restlessness, diaphoresis, metabolic acidosis, hypotension, prolonged capillary refill (more than 2 seconds), decreasing pulse pressure, and oliguria. The most frequent neurologic finding in patients with shock is alteration in the level of consciousness, ranging from confusion to coma (Rivers, 2016, p. 673). Skin changes will depend on the type of shock and may include cool, clammy skin (related to vasoconstriction) or warm, pink skin (related to peripheral vasodilation of distributive shock).

Nursing Alert

Although BP readings alone are unreliable indicators of circulatory status, a single episode of systolic BP of less than 80 mm Hg is associated with an 18% increase in hospital mortality (Rivers, 2016).

Nursing Alert

Pulse pressure is the difference between the systolic and diastolic pressure; for example, for a BP of 120/80, the pulse pressure is 40 mm Hg. Pulse pressure rises when the stroke volume increases and narrows when resistance to outflow decreases (Porth, 2015). The pulse pressure may decrease before the systolic pressure decreases, and in patients with shock pulse pressure is less than 25 mm Hg. Nurses are key to identifying narrowing pulse pressures in patients early in the course of shock and should immediately report a pulse pressure of less

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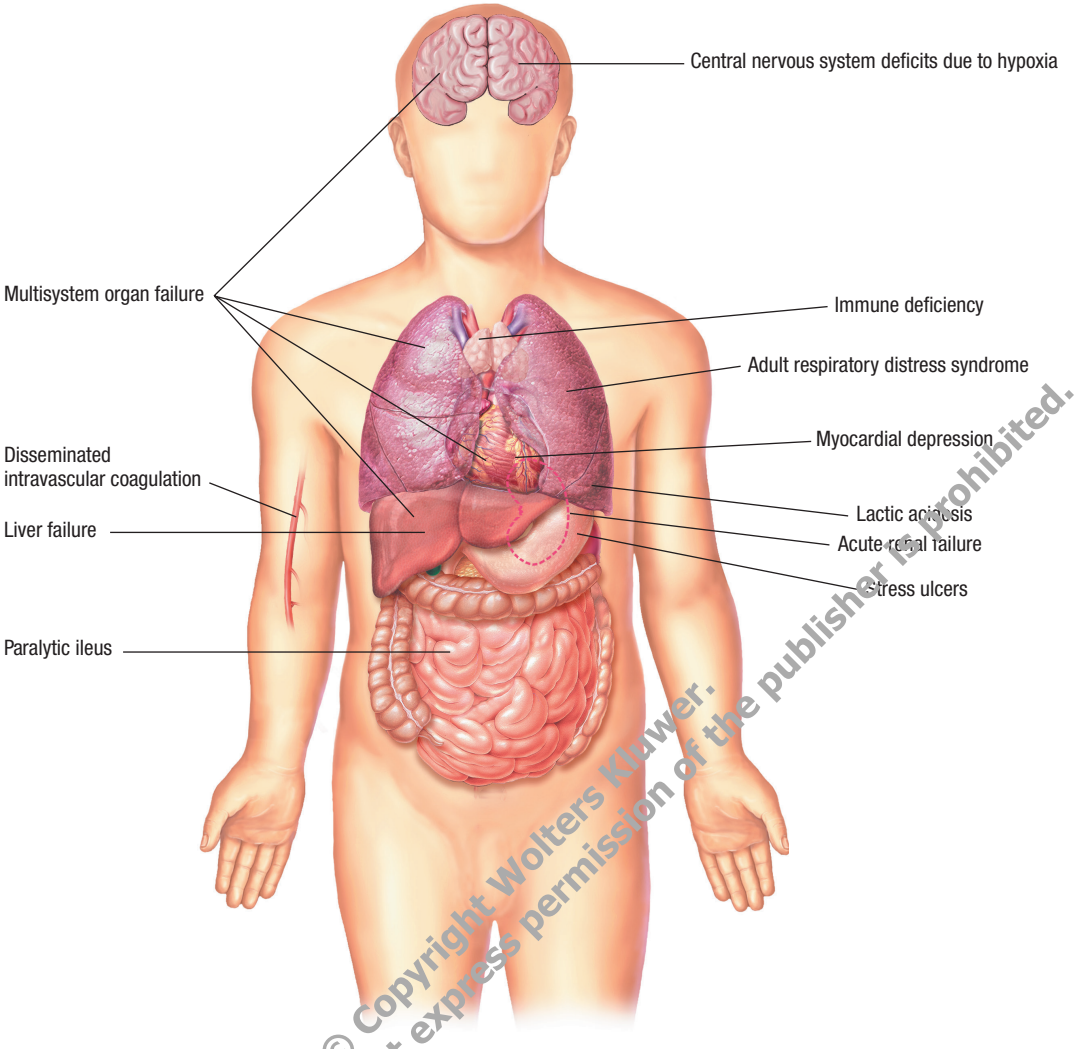


FIGURE 54-1 Multiorgan system effects of shock.

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than 25 mm Hg to the provider, even if other signs of shock have not yet manifested.

Medical and Nursing Management

Early and aggressive interventions are essential to the survival of patients in shock; therefore, identifying patients who may be in shock and reporting subtle changes in assessment are imperative. Initially, two large-bore peripheral IVs (at least 16 gauge) are inserted to prepare for fluid administration (Rivers, 2016). Two IV lines allow simultaneous administration of fluid, medications, and blood component therapy if required. Patients in shock are often cared for in the intensive care (ICU) setting in order to

- facilitate close monitoring (hemodynamic monitoring, arterial line insertion, electrocardiographic [ECG] monitoring, arterial blood gases [ABGs], oxygen saturation, serum electrolyte levels, physical and mental status changes);
- allow for rapid and frequent administration of various prescribed medications and fluids;
- provide access for strict measurement of intake and output (Foley catheter may be inserted, and urine output should be measured vigilantly: A urine output of less than 30 mL/hr or 0.5 mL/kg/hr is indicative of renal hypoperfusion and/or hypovolemia); and possibly
- provide interventions with supportive technologies, such as mechanical ventilation, dialysis, extracorporeal membrane oxygenation (ECMO), and intra-aortic balloon pump.

Nursing care of patients in shock requires expertise in assessing and understanding shock and the significance of changes in assessment data.

Specific management of shock depends on the type of shock and its underlying cause, but, in general, care is aimed at achieving the following goals using the acronym ASSESS:

A: Assess frequently the body's response to shock
S: Support the pumping action of the heart
S: Support the respiratory system
E: Encourage patient and family involvement in care, particularly in end-of-life discussions

S: Strengthen the competence of the vascular system
S: Significantly optimize intravascular volume

IRREVERSIBLE STAGE (END-ORGAN DYSFUNCTION)

Pathophysiology

If adequate tissue perfusion is not restored in a timely fashion, irreversible end-organ damage ensues. This represents the point along the shock continuum where end-organ damage is so severe that the patient does not respond to treatment and cannot survive. As the damage increases, acidosis may become so severe that even aggressive therapies such as vasopressor support may not work. For example, kidney and liver failure, compounded by the release of necrotic tissue toxins, will create an overwhelming metabolic acidosis. If the integrity of the gut barrier is compromised, bacteria and their toxins can be translocated into the bloodstream. With severe hypoperfusion of the liver, massive elevations in the enzyme aminotransferase and extensive hepatocellular damage occurs. Anaerobic metabolism contributes to a worsening lactic acidosis. Respiratory system failure prevents adequate oxygenation and ventilation despite mechanical ventilatory support, and the cardiovascular system is ineffective in maintaining an adequate mean arterial pressure (MAP; MAP = [(2 × diastolic pressure + systolic pressure)/3]) for tissue 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 on page 1528.

Nursing Alert
To optimize end-organ perfusion, the nurse monitors the MAP. Although a specific MAP goal has not been established for all shock states, an MAP of at least 60 to 65 mm Hg is a reasonable goal (Rivers, 2016).

Medical and Nursing Management

Specific therapies in this stage are usually the same as for shock. Although the patient may have progressed from shock to irreversible end-organ damage, the judgment that the shock is irreversible can be made only retrospectively, on the basis of the patient's failure to respond to treatment. As in 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. As it becomes obvious that the patient is unlikely to survive, the family must be informed about the prognosis and likely outcome. Clear, honest communication is imperative as families may not understand what is happening with the patient. 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, 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.

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:

- Optimization of systemic oxygen delivery to improve tissue oxygenation
- 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 (see Fig. 54-2).

Optimize Oxygen Delivery

The first step is to assess oxygenation and provide supplemental oxygen as needed. 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. Frequent assessment of the respiratory status is warranted since many patients with severe shock will require intubation and ventilatory support. Continuous oxygen saturation monitoring is expected. It is important to consider that intubation and mechanical ventilation not only provides for delivery of oxygen and removal of carbon dioxide but also reduces the work of breathing, which, in the patient with hypoperfusion, further exacerbates lactic acidemia (Rivers, 2016). Nurses understand that when patients are "working to breathe," this strenuous use of accessory respiratory muscles can increase

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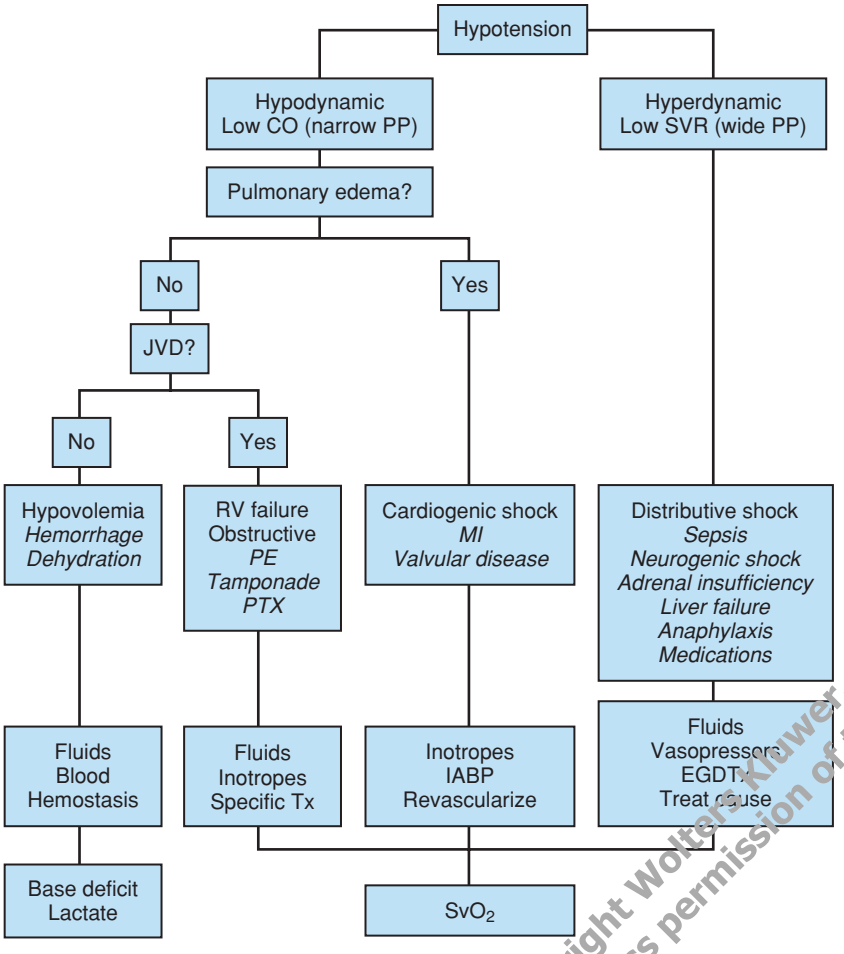


FIGURE 54-2 Shock diagnosis, treatment, and monitoring. CO, cardiac output; PP, pulse pressure; SVR, systemic vascular resistance; JVD, jugular venous distention; PE, pulmonary embolism; PTX, pneumothorax; MI, myocardial infarction; Tx, treatment; IABP, intra-aortic balloon pump; EGDTx, early goal-directed therapy; SvO_2, mixed venous oxygen saturation (percent of oxygenated hemoglobin in blood returning to the right heart). (Reprinted with permission from Lawrence, P., Bell, R., Dayton, M., & Hebert, J. (2012). *Essentials of general surgery* (5th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.)

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oxygen consumption by 50% to 100% and decrease cerebral blood flow by 50% (Jones & Kline, 2014).

Fluid Replacement

The fluids administered vary but may include **crystalloids** (electrolyte solutions that move freely between intravascular and interstitial spaces), **colloids** (large-molecule IV solutions), and blood components (Table 54-2). The best fluid to treat shock remains controversial. In emergencies, the "best" fluid is often the fluid that is readily available and is dependent on the type of shock and patient presentation. Fluid resuscitation should be initiated early and aggressively in patients with shock to maximize intravascular volume. For example, in patients with septic shock, an intravenous fluid challenge (20 to 30 mL/kg) may be given rapidly of crystalloid IV fluid to improve intravascular volume (Dellinger et al., 2013; Ferri, 2016). A bolus of 500 mL titrated to MAP or measurement of preload is recommended according to Rivers (2016), and repeated based on the absence of pulmonary edema and evidence of improving tissue perfusion. Both crystalloids (e.g., saline and lactated Ringer's [LR] solution) and colloids (e.g., albumin) may be administered to restore intravascular volume. However, IV fluid choice remains dependent on the type of shock and on the specific patient presentation. For example, in patients with septic shock, crystalloid fluid remains the IV fluid of choice (Dellinger et al., 2013). Conversely, Hespan, a type of colloid fluid, has been shown to cause increased organ damage and mortality in patients who are critically ill, including those with shock (Rivers, 2016). Blood should be given for hypovolemic shock due to trauma and/or hemorrhage, although initially crystalloids may be given to patients to restore intravascular volume while waiting for blood products to transfuse.

Common crystalloid fluids used for resuscitation in hypovolemic shock include 0.9% sodium chloride solution (normal saline) and LR solution. LR is an electrolyte solution containing the lactate ion, which should not be confused with lactic acid. The exogenous lactate is converted to bicarbonate by the liver, thus assisting in treatment of the acidosis. It is important to assess for liver disease when using this solution as the acidosis may worsen when LR is administered to patients with severe liver dysfunction. Additionally, because each liter contains 4 mEq of potassium per liter, it should not be given to patients with severe hyperkalemia. Care also must be taken when administering normal saline rapidly as large volumes result in hyperchloremic metabolic acidosis and coagulopathy (Rivers, 2016). If colloids are administered, the nurse

TABLE **54-2 Fluid Replacement in Shock**

Fluids	Advantages	Disadvantages
Crystalloids		
0.9% sodium chloride (normal saline solution)	Widely available, inexpensive	Requires large volume of infusion; can cause hyperchloremic metabolic acidosis, pulmonary edema
Lactated Ringer's (LR)	Lactate ion helps buffer metabolic acidosis	Requires large volume of infusion; can cause pulmonary edema; should not be used in patients with severe hyperkalemia or patients with severe liver dysfunction
Hypertonic saline (3%, 5%, 7.5%)	Small volume needed to restore intravascular volume	Danger of hypernatremia, central pontine myelinolysis, and increased serum osmolality
Colloids		
Albumin (5%, 25%)	Rapidly expands plasma volume	Expensive; requires human donors; limited supply; can cause heart failure
Dextran (40, 70)	Synthetic plasma expander	Interferes with platelet aggregation; not recommended for patients with hemorrhagic shock
Hetastarch	Synthetic; less expensive than albumin; effect lasts up to 36 hours	Prolongs bleeding and clotting times

understands that they 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. Albumin is a colloid that is commonly ordered to treat hypovolemic shock. The disadvantage of colloids is their high cost, and outcomes of patients with hypovolemic shock are equivalent regardless if they are given crystalloids or albumin (Rivers, 2016). Regardless of the solution administered it is important to avoid "over resuscitation" and thus causing fluid volume excess.

The patient receiving fluid replacement in the context of shock 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.

Often, a central venous line is inserted in order to measure central venous pressure (CVP). In addition to physical assessment, the CVP helps in monitoring the patient's response to fluid resuscitation. A normal CVP is 2 to 8 mm Hg. Several readings are obtained to determine a range, and fluid replacement is continued to achieve a CVP of 8 to 12 mm Hg; higher-than-"normal" CVPs readings are desired since ventricles tend to stiffen during shock (Jones & Kline, 2014).

Nursing Alert

The nurse understands that the CVP represents the volume in the right atrium and, therefore, is reflective of the amount of preload *to the right ventricle. But in patients with shock, because of the stiffening of the ventricles, the CVP cannot predict what is actually pumped out by the left ventricle; thus the nurse considers parameters such as urinary output (increasing), MAP, and lactate concentrations (decreasing) as measures of improved hemodynamic response to treatment.*

With newer technologies, catheters can be placed that allow the monitoring of intravascular pressures and venous oxygen levels. Assessment of mixed venous oxygen saturation (SvO_2) or $ScvO_2$ (central venous oxygen saturation) is done with a CVP line or from a special pulmonary artery catheter that has fiber optics capable of calculating the oxygen saturation of hemoglobin. SvO_2 reflects the balance between oxygen delivery and oxygen consumption. Knowing the SvO_2 in conjunction with pulse oximetry is helpful in evaluating the adequacy of oxygenation; newer technology allows for continuous $SvO_2/ScvO_2$ measurements similar to the pulse oximetry. A normal $SvO_2/ScvO_2$ is 70% to 80% (i.e., when the blood is returning to the right side of the heart, it has lost only 20% to 30% of its oxygen). An $SvO_2/ScvO_2$ of 50% to 70% reveals that more oxygen is being extracted from the RBCs; when the $SvO_2/ScvO_2$ is at 30% to 50% the oxygen extraction is becoming exhausted and lactic acidosis begins, and at 25% to 30%, severe lactic acidosis exists. $SvO_2/ScvO_2$ below 25% is associated with cellular death (Reyer, 2013). When the SvO_2 drops, it reflects the increased consumption of oxygen and the need for intervention.

Knowing the CVP assists in evaluating the intravascular volume. Also the BP, urine output, and base deficit all are suggestive of intravascular volume status. Hemodynamic monitoring with arterial and pulmonary artery lines may be

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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 55.

Vasoactive Medication Therapy

Vasoactive medications can be used in patients with all forms of shock to improve the patient's hemodynamic stability when fluid therapy alone cannot maintain adequate MAP. Different medications work on different aspects of CO, and specific medications are selected depending on the underlying cause (see Chapter 55, Table 55-9). Although most of the medications have several effects, these medications are used to (1) increase the strength of myocardial contractility, (2) regulate the heart rate, (3) reduce myocardial resistance, and (4) initiate vasoconstriction.

Receptors in the sympathetic nervous system 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, the nurse must monitor vital signs frequently (at least every 5 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 must be used to ensure that the medications are delivered safely and accurately. Often, invasive monitoring using an arterial line is used to titrate medications 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 above 65 mm Hg). Vasoactive medications should be tapered when possible, and the patient should be weaned from medication with frequent monitoring of BP (at least every 15 minutes).

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. 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, with some form of enteral nutrition always administered. The integrity of the GI system depends on direct exposure to nutrients. Stress ulcers occur frequently in patients who are acutely ill because of the compromised blood supply to the GI tract. Therefore, antacids (e.g., Carafate), H_2 blockers (e.g., famotidine [Pepcid], ranitidine [Zantac]), and/or proton pump inhibitors (e.g., lansoprazole [Prevacid]) are prescribed to prevent ulcer formation by inhibiting gastric acid secretion or increasing gastric pH.

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TYPES OF SHOCK

Hypovolemic Shock

Hypovolemic shock, the most common type of shock, is characterized by a decreased intravascular volume. Intracellular fluid accounts for about two thirds of the total body water. The remaining one third of fluid is in the extracellular space that is divided into two compartments: intravascular space (IVS, inside blood vessels) or interstitial space (ISS, surrounding tissues). Approximately three quarters of the extracellular fluid is in the ISS, and the remaining one quarter is in the IVS. Thus, the normal intravascular volume is 4 to 6 L. Hypovolemic shock occurs when there is a reduction in intravascular volume by 15% to 25%, which represents a loss of 750 to 1,500 mL of blood in a 70-kg (154-lb) person.

Pathophysiology

Hypovolemic shock can be caused by sudden fluid losses, such as hemorrhage, or by a gradual deficit in I&O, such as dehydration. See Chapter 56, Table 56-1 for further information on hemorrhagic shock. Figure 54-3 depicts the sequence of events in hypovolemic shock. Box 54-3 discusses risk factors for hypovolemic shock.

Medical Management

Major goals in the treatment of hypovolemic shock are (1) to restore intravascular volume, (2) to reverse the sequence of events leading to inadequate tissue perfusion, and (3) 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.

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

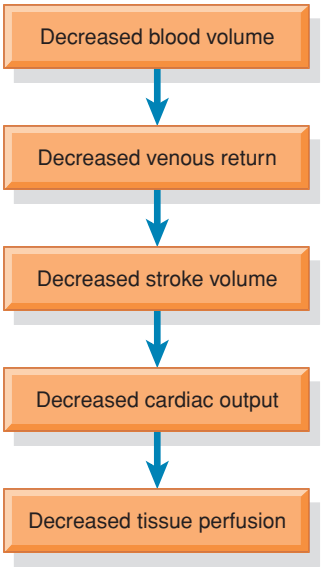


FIGURE 54-3 Pathophysiologic sequence of events in hypovolemic shock.

efforts are made to identify and treat the cause. In elderly patients, dehydration is a common cause of hypovolemia.

If the underlying cause of the hypovolemia is dehydration, medications are also administered to reverse the cause of the dehydration. For example, insulin is administered if dehydration is secondary to hyperglycemia, and desmopressin (DDAVP) is administered for patients with diabetes insipidus.

Beyond reversing the primary cause of the decreased intravascular volume, fluid replacement (also referred to as fluid resuscitation) is of primary concern. At least two large-gauge IV lines are inserted to establish access for fluid administration. 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 or interstitial compartment. Blood products may need to be administered, particularly if the cause of the hypovolemic shock is hemorrhage. Packed red blood cells are administered to replenish the patient's oxygen-carrying capacity, in conjunction with other fluids that will expand volume. Plasma and platelet transfusions can help with fluid resuscitation and also treat other underlying problems. The need for transfusions is based on the patient's individual perfusion 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 (i.e., compounds capable of carrying oxygen in the same way that blood does) as potential alternatives to blood component therapy. Risks inherent to administering blood include the possibility of a transfusion reaction.

Commonly, positioning the patient with his or her head down, known as the *Trendelenburg position*, has been thought to increase blood flow to vital organs by increasing preload. This practice has not been found to be supported by evidence and, in fact, may worsen pulmonary gas exchange and risk of aspiration (Rivers, 2016). An alternative is to elevate the patient's legs slightly to improve cerebral circulation and promote venous return to the heart, but this position is contraindicated for patients with head injuries.

BOX 54-3 Risk Factors for Hypovolemic Shock

External: Fluid losses:
- Trauma
- Surgery
- Vomiting
- Diarrhea
- Diuresis
- Diabetes insipidus
- NPO status

Internal: Fluid shifts:
- Hemorrhage
- Burns
- Ascites
- Peritonitis
- Dehydration

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 signs of complications and side effects of treatment and reporting these signs early in treatment.

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 20).

Fluid replacement complications can occur, often when large volumes are administered rapidly. Therefore, the nurse monitors the patient closely for cardiovascular overload and edema. The risk of these complications is increased in the elderly and in patients with pre-existing cardiac disease. Hemodynamic pressure, vital signs, ABGs, serum lactate levels, hemoglobin and hematocrit levels, and fluid I&O are among the parameters monitored. Temperature should also be monitored closely to ensure that rapid fluid resuscitation does not precipitate

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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 CVP. CVP is typically 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 to the provider any changes in BP, pulse pressure, heart rate and rhythm, and lung sounds.

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. Cardiogenic shock is seen most often in patients with myocardial infarction (MI). Other causes of cardiogenic shock are related to conditions that stress the myocardium (e.g., severe hypoxemia, acidosis, hypoglycemia, and hypocalcemia), as well as in conditions that result in ineffective myocardial function (e.g., cardiomyopathies, valvular damage, and arrhythmias).

Pathophysiology

In cardiogenic shock, CO, which is a function of stroke volume and heart rate, is compromised. When stroke volume and heart rate decrease or become erratic, BP falls (systolic blood pressure is less than 90 mm Hg or there is a decrease from baseline of more than 30 mm Hg for more than 30 minutes), and systemic tissue perfusion is compromised (urine output decreases, skin becomes cool and mottled, mental status changes [sensorium becomes clouded], anxiety is obvious, capillary refill is delayed). Blood supply for the heart muscle itself is inadequate, resulting in further decreases in CO (cardiac index is less than 2.2 L/min/m²). Additionally, diastolic dysfunction increases left atrial pressure, which leads to pulmonary congestion and hypoxemia that can further exacerbate myocardial ischemia and impair ventricular performance (pulmonary capillary wedge pressure is greater than 18 mm Hg). This can occur rapidly or over a period of days. Patients in cardiogenic shock may experience the pain of angina and develop arrhythmias and hemodynamic instability. Figure 54-4 depicts the pathophysiology of cardiogenic shock.

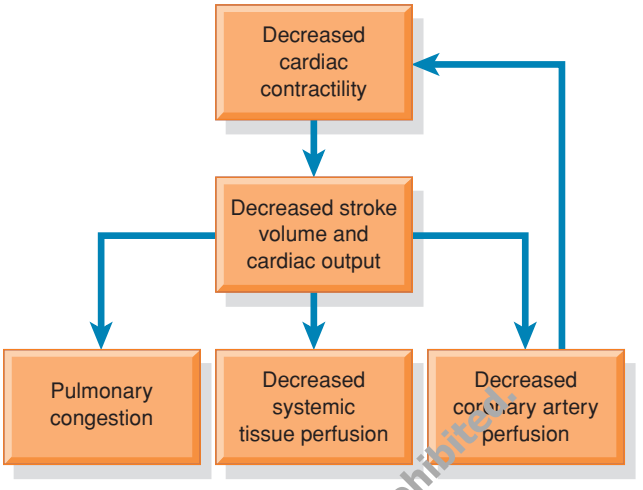


FIGURE 54-4 Pathophysiologic sequence of events in cardiogenic shock.

Nursing Alert
In the case of heart failure resulting in cardiogenic shock, the nurse assesses for jugular vein distention (JVD), rales, shortness of breath, and S_3 gallop.

Medical Management

The goals of management in cardiogenic shock are (1) to limit further myocardial damage and preserve the healthy myocardium and (2) to improve the cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both. In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demands.

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 cardiogenic shock due to ischemia or infarction, the patient may require thrombolytic therapy, angioplasty, coronary artery bypass graft surgery, intra-aortic balloon pump therapy, ventricular assist device (VAD), or some combination of these treatments. General interventions for cardiogenic shock include supplemental oxygen, controlling chest pain, providing selected fluid support, administering vasoactive medications, controlling heart rate with medication or pacemaker, and using a mechanical assist device if needed. Serial laboratory markers for ventricular dysfunction (e.g., BNP) and cardiac enzyme levels (i.e., troponin-I) are measured, serial 12-lead ECGs are obtained, and frequently an echocardiogram is ordered to assess the degree of myocardial damage or dysfunction.

Pharmacologic Therapy

Oxygen

In the early stages of shock, supplemental oxygen is administered at a rate sufficient to achieve an oxygen saturation exceeding 90%. Monitoring of ABG values and pulse oximetry values helps determine whether the patient requires a more aggressive method of oxygen delivery.

Analgesia

If a patient experiences chest pain, an IV analgesic (commonly morphine sulfate) is administered for pain relief. In addition to relieving pain, morphine dilates the blood

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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).

Antiplatelet Agents and Beta Blockers

Antiplatelet agents, such as aspirin, should be given in the case of a patient with acute MI. In addition, a beta blocker is used to decrease workload of the heart and preserve cardiac muscle. The use of these medications in the case of a patient with cardiogenic shock can be complicated since beta blockers will decrease BP. See Chapter 14 for acute treatment of chest pain and MI.

Nursing Alert

During an acute MI, aspirin may be ordered to prevent worsening thrombosis in the coronary arteries. When administering aspirin in this acute situation, the nurse should ask the patient to chew aspirin (162 to 325 mg) rather than swallow a whole enteric-coated aspirin. Chewing aspirin allows for a more rapid absorption rate than does swallowing it whole (Anderson, 2016; Feldman & Cryer, 1999).

Vasoactive Medications

Vasoactive medication therapy consists of multiple pharmacologic strategies to restore and maintain adequate CO. In cardiogenic shock, the aims of vasoactive medication therapy are to improve cardiac contractility, optimize preload and afterload, reduce myocardial oxygen demand, and stabilize heart rate and rhythm. Medications commonly used to treat cardiogenic shock include dobutamine, dopamine, and nitroglycerin. See Table 55-9 for a review of common vasoactive medications.

Nursing Alert

The nurse is vigilant in observing the patient's response to vasoactive agents because of the potential of worsening myocardial ischemia by increasing cardiac work.

Dopamine is a sympathomimetic agent. It acts as an inotrope and a vasopressor (at higher doses). It may be used with dobutamine and nitroglycerin to improve tissue perfusion. Sympathomimetic agents must be used with caution in patients with cardiogenic shock since they increase myocardial oxygen demand and can worsen failure. Also, in patients with severe metabolic acidosis, which occurs in the later stages of shock, the effectiveness of dopamine is diminished. Norepinephrine also may be considered as a first-line agent for hypotension in patients with cardiogenic shock; it is a vasoactive agent that acts primarily as a vasoconstrictor, has a mild inotropic effect, and increases coronary flow while having less of an effect on heart rate in patients with tachycardia (Hollenberg, 2016).

If tissue perfusion remains inadequate, inotropic therapy may be considered with dobutamine, which predominantly affects beta-1 adrenergic receptors (increases the strength of myocardial activity and improves CO). Dobutamine enhances the strength of cardiac contraction, improving stroke volume ejection and overall CO. Because it also has some beta-2 properties, vasodilation occurs (decreased afterload), and the bronchioles relax. The nurse is aware that dobutamine may exacerbate hypotension is some patients (related to vasodilatory effects) and can precipitate tachyarrhythmias (Hollenberg, 2016).

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

Nursing Alert

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

Diuretics

Diuretics such as furosemide (Lasix) may be administered to reduce the workload of the heart by reducing fluid accumulation. Diuretics must be used **cautiously** since they can lead to hypovolemia. In addition, aggressive diuresis can lead to metabolic alkalosis, often referred to as a *contraction alkalosis.* The use of diuretics can also lead to electrolyte abnormalities, the most common of which is hypokalemia.

Antiarrhythmic Medications

Multiple factors, such as hypoxemia, electrolyte imbalances, and acid–base imbalances, contribute to serious cardiac arrhythmias in all patients with shock. In addition, as a compensatory response to decreased CO and BP, the heart rate increases beyond normal limits. This impedes CO 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 arrhythmias, as well as commonly prescribed medications, see Chapter 17.

Fluid Status

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

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IV fluid boluses are cautiously administered to determine optimal filling pressures for improving CO. Nurses should use caution when administering fluids rapidly because rapid fluid administration in patients with cardiac failure may result in acute pulmonary edema. Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of fluid in the pulmonary tissues.

Mechanical Assistive Devices

If CO does not improve despite supplemental oxygen, vasoactive medications, and fluid boluses, mechanical assistive devices can sometimes be used temporarily to improve the heart's ability to pump. Intra-aortic balloon counterpulsation or left and right ventricular assist devices and total temporary artificial hearts are means of providing temporary circulatory assistance and are reviewed in Chapters 16 and 55.

Nursing Management

Preventing Cardiogenic Shock

In many circumstances, identifying at-risk patients early, promoting adequate perfusion of the heart muscle, and decreasing cardiac workload can prevent cardiogenic shock. Risk factors include advanced age, anterior MI or previous MI, diabetes, hypertension, multivessel CAD, and peripheral vascular and cerebrovascular diseases (Hollenberg, 2016). Patients who have decreased ejection fractions, larger infarctions, and echo results that demonstrate a lack of hyperkinesis in the myocardial remote from the infarction should be considered at risk for cardiogenic shock (Hollenberg, 2016). Minimizing patient risk can be accomplished by conserving the patient's energy, promptly relieving angina, and administering supplemental oxygen, aspirin, and beta blockers. When cardiogenic shock is present, 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, pulmonary, and renal 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

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 the therapy. IV infusions must be observed closely because tissue necrosis and sloughing may occur if vasopressor medications infiltrate the tissues. Urine output, blood urea nitrogen (BUN), and serum creatinine levels are monitored to detect decreased kidney function secondary to the effects of cardiogenic shock or its treatment.

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 kidney 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.

DISTRIBUTIVE SHOCK

Distributive shock, also sometimes called circulatory shock, occurs when the body's ability to adjust vascular tone is impaired, and thus blood volume is abnormally displaced in the vasculature (e.g., when blood volume pools in peripheral blood vessels). The displacement of blood volume causes a relative hypovolemia because not enough blood returns to the heart, which leads to subsequent inadequate tissue perfusion. 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, distributive 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 distributive shock further subdivide this classification of shock into three types: (1) **septic shock**, (2) **neurogenic shock,** and (3) **anaphylactic shock**. Other causes of distributive shock are rare but include toxic shock syndrome,

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BOX 54-4

Risk Factors for Distributive Shock

Septic shock:
- Immunosuppression
- Extremes of age (under 1 year of age and over 65 years of age)
- Malnourishment
- Chronic illness
- Invasive procedures

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

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

drug overdose, neurogenic insults, addisonian crisis, and myxedema coma (Rivers, 2016). In all types of distributive shock, massive arterial and venous dilation allows blood to pool peripherally. The different types of shock cause variations in the pathophysiologic chain of events. Risk factors for distributive shock are summarized in Box 54-4.

SEPTIC SHOCK

Septic shock, the most common type of distributive shock, is caused by widespread, overwhelming infection in combination with a dysregulated host immune response. Because of the increased sophistication of antibiotic therapy and improved recognition of sepsis and initiation of intensive care management, the incidence of septic shock has declined. Approximately 750,000 cases of severe sepsis or septic shock occur annually in the United States, with mortality rates of about 25 to 35%, which is down from rates of 70% in 2010 (Russell, 2016). Finding and aggressively treating the source of infection, as well as providing aggressive cardiopulmonary support, are important determinants of the clinical outcome.

Risk Factors

Nosocomial infections (infections occurring in the hospital) in patients who are critically ill that may progress to septic shock commonly originate in the lungs (pneumonia) and urinary (pyelonephritis) and GI tract (peritonitis, intra-abdominal abscess, cholangitis), although they can originate anywhere in the body (refer to Table 54-3 for common sites related to sepsis). Organisms include gram-positive or negative bacteria, fungi, and, rarely, protozoa or rickettsiae are associated with septic shock; commonly gram-positive bacteria, especially methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), penicillin-resistant *Streptococcus pneumoniae*, and resistant gram-negative bacilli are seen (Russell, 2016). Additional risk factors are age, immunocompromise due to malnutrition, alcoholism, malignancy, diabetes mellitus, and AIDS; invasive procedures and indwelling medical devices; and the increased number of resistant microorganisms. Elderly patients continue to be at particular risk for sepsis because of decreased physiologic reserves and an aging immune system. The incidence of septic shock can be reduced by carrying out infection control practices, including the use of meticulous aseptic technique, properly cleaning and maintaining equipment, and using thorough hand hygiene techniques.

Pathophysiology

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 complex cascade of physiologic events that leads to poor tissue perfusion. Widespread endothelial injury leads to increased capillary permeability, which triggers seepage of protein-rich fluid from the capillaries, and vasodilation, which occurs when injured endothelial cells release nitric acid (a potent vasodilator). These two effects 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 in areas where a clot may or may not be needed, further compromising tissue perfusion. The imbalance of the inflammatory response and the clotting and fibrinolysis cascades are considered critical elements of the devastating physiologic progression that is present in patients with severe sepsis. To illustrate, the increased permeability within the lung is a key component of acute lung injury (ALI), whereas increased permeability in the intestine can lead to translocation of intestinal bacteria.

Clinical Manifestations and Assessment

Sepsis is defined by presence of at least two of the four signs of the systemic inflammatory response syndrome: (1) fever (above 38°C) or hypothermia (below 36°C); (2) tachycardia (more than 90 beats/min); (3) tachypnea (more than 20 breaths/min), hypocapnia (partial pressure of carbon dioxide less than 32 mm Hg), or the need for mechanical ventilatory assistance; and (4) leukocytosis (more than 12,000 cells/μL), leukopenia (fewer than 4,000 cells/μL), or a left shift (more than 0% immature band cells) in the circulating white blood cell differential and suspected or proven infection plus a documented or suspected infection (Russell, 2016, p. 685).

Systemic inflammatory response syndrome (SIRS) presents clinically like sepsis. The only difference between SIRS and sepsis is that an identifiable causative infectious

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TABLE **54-3 Common Sites and Diseases Associated with Sepsis/Systemic Inflammatory Response Syndrome (SIRS)**

Organ System	Location	Disease
Respiratory	Upper respiratory tract	Sinusitis Mastoiditis
	Lower respiratory tract	Pneumonia Lung abscess Empyema
Gastrointestinal	Mediastinum Hepatobiliary Intra-abdominal	Esophageal rupture/perforation Hepatic abscess Cholangitis Cholecystitis Intestinal infarction/perforation, pancreatitis Intra-abdominal/diverticular abscess
Cardiovascular	Mediastinum Native or prosthetic cardiac valve	Postoperative mediastinitis Endocarditis
Genitourinary	Kidney, ureter, and bladder	Perinephric abscess Pyelonephritis Cystitis
Neurologic	Brain and meninges	Meningitis Intracranial abscess
Dermatologic	Traumatic wound, surgical wound, or burn site	Soft tissue abscess Necrotizing fasciitis Infected decubitus ulcer Full and partial thickness burn
Prosthetic	Central/peripheral venous catheter Arterial catheter Ventriculoperitoneal shunt Dialysis catheter Articular prosthetic device Dialysis graft/shunt	Catheter infection Infected prosthesis
Other	Vascular system	Septic thrombophlebitis

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agent is established in the latter. SIRS/sepsis results in an inflammatory and immunologic response that is overwhelming, thought to be caused in large part by an array of inflammatory cytokines, including but not confined to tumor necrosis factor (TNF). The inflammatory response also leads to increased coagulation, and coagulation triggers inflammation; a positive feedback loop is at play that has the potential to disrupt essential kidney, cardiovascular, and respiratory function (Russell, 2016). Any overwhelming insult can stimulate SIRS and may progress to sepsis; noninfectious disorders include pancreatitis and drug intoxication. Therefore, despite an absence of infection, antibiotic agents still may 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.

Sepsis is an evolving process, without a predictable linear progression. In the past, septic shock has been described as having two phases, a hyperdynamic (warm) and a hypodynamic (cold) phase. Although the division into phases may promote understanding of sepsis, its actual progression into severe sepsis and septic shock is not always easy to recognize clinically. Initially, a hyperdynamic response occurs; it is characterized by a high CO with systemic vasodilation. The BP may remain within normal limits, or the patient may be hypotensive but responsive to fluids. The heart rate increases, progressing to tachycardia. 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.

As the sepsis progresses, tissues become more underperfused and acidotic, compensation begins to fail, and the

BOX 54-5

Definitions in Sepsis

Bacteremia: The presence of bacteria in the blood

Infection: The presence of microorganisms that trigger an inflammatory response

Systemic inflammatory response syndrome (SIRS): A syndrome resulting from a *severe clinical insult* that initiates an overwhelming inflammatory response by the body; defined as two or more of the following conditions:

- Temperature greater than 38.5°C or less than 35.0°C
- Heart rate of more than 90 bpm
- Respiratory rate of more than 20 breaths/min or $PaCO_2$ of less than 32 mm Hg
- White blood cell count of more than 12,000 cells/mL, less than 4,000 cells/mL, or greater than 10% immature (band) forms

Sepsis: A systemic response to *infection;* may occur after a burn, surgery, or serious illness and is defined as the presence of SIRS, plus the presence of an infectious source (either by documented culture or by visualizing focal infection)

Severe sepsis: Defined as sepsis, plus at least one of the following signs of organ hypoperfusion:

- Areas of mottled skin
- Capillary refill of more than 3 seconds
- Decreased urinary output (less than 0.5 mg/kg for 1 hour) (oliguria)
- Lactate greater than 2 mmol/L (lactic acidosis)
- Abrupt change in mental status (decreased Glasgow Coma Scale)
- Platelet count of less than 100,000 or disseminated intravascular coagulation (DIC) (thrombocytopenia)
- Acute lung injury or acute respiratory distress syndrome (ARDS) (hypoxemia)
- Cardiac dysfunction

Septic shock: Shock associated with sepsis; defined as severe sepsis, plus one of the following:

- Mean arterial pressure (MAP) of less than 60 after fluid resuscitation; systolic blood pressure less than 90 mm Hg or a decrease from baseline that is greater than 40 mm Hg, despite adequate fluid resuscitation.
- Need for vasoactive medication in order to maintain MAP of greater than 60

Adapted from Annane, D., & Cavaillon, J. M. (2005). Septic shock. *Lancet*, *365*(9453), 63–78; Sommers, M. S. (2003). The cellular basis of septic shock. *Critical Care Nursing Clinics of North America*, *15*(1), 13–26.

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patient becomes more hypodynamic. The cardiovascular system also begins to fail, the BP does not respond to vasoactive agents and fluid resuscitation, and signs of end-organ damage are visible (e.g., kidney failure, pulmonary failure, hepatic failure). As sepsis progresses to septic shock, the BP drops, and the skin becomes cool and pale. Temperature may be normal or below normal. Heart and respiratory rates remain rapid. Urine output decreases, and multiple organ dysfunction syndrome (MODS) develops (discussed on page 1528).

In an effort to promote recognition and earlier treatment of patients with sepsis, the Society of Critical Care Medicine defined a common set of terms and cues for clinicians (Box 54-5).

Medical Management

Current treatment of septic shock involves *identification and elimination of the cause of infection and aggressive cardiopulmonary support* to prevent or limit end-organ damage and death. Specimens of blood, sputum, urine, and wound drainage are collected for culture using aseptic technique. Any potential routes of infection must be eliminated. IV lines may need to be removed and reinserted at other body sites; the patient will require scrupulous sterile techniques for the insertion of catheters, with consideration of the use of antibiotic-impregnated catheters (Russell, 2016). If possible, urinary catheters are removed or replaced. Any abscesses are drained, and necrotic areas are surgically débrided.

Fluid replacement must be instituted to correct the hypovolemia that results from the incompetent vasculature and the inflammatory response. Crystalloids, colloids, and blood products may be administered to increase intravascular volume. IV fluids boluses (in the range of 20 to 40 mL/kg) may be ordered to improve intravascular volume (Rivers, 2016). The CVP, MAP, urinary output, and SvO_2 are used to guide therapy.

Oxygen is administered and guided by the pulse oximeter, SvO_2, and clinical presentation. Many patients will require mechanical ventilation, with a lower tidal volume (typically greater than 6 mL/kg of ideal body weight [IBW]), which has been associated with decreased mortality from ALI and acute respiratory distress syndrome (ARDS). ARDS and ALI are discussed in Chapter 10.

Preventing the progress from sepsis to septic shock necessitates early diagnosis and aggressive resuscitation. The patient's responses to treatment are closely monitored, and resuscitation is continued until CVP is greater than 8, MAP is greater than 65 mm Hg, urine output is 0.5 mL/kg/hr or more, and SvO_2 is greater than 70%. This approach is associated with a decreased mortality and should be considered standard of care in the management of septic shock.

Pharmacologic Therapy

Antibiotics

Before antibiotic therapy is initiated, it is important that all cultures be obtained. However, if obtaining cultures is delayed, the nurse is aware that antimicrobials should be administered within 1 hour of identification of septic shock. The nurse anticipates the need for blood, sputum, urine, and wound cultures, and, depending on the site of the expected infection, additional cultures may be obtained from the cerebrospinal

fluid (in the case of suspected meningitis) or pleural space (if empyema is suspected). If the infecting organism is unknown, empiric broad-spectrum antibiotics are started. When culture and sensitivity reports become available, the antibiotic agents can be changed to those that are more specific to the infecting organism and less toxic to the patient.

Nursing Alert

It is important for the nurse to consider all possible sources of the infection. Symptoms such as cough, sputum production, adventitious lung sounds and increased work of breathing suggests pneumonia; pleuritic chest pain, decreased fremitus, and a dull, flat sound on percussion suggests empyema; dysuria and costovertebral angle tenderness suggests pyelonephritis; erythema and drainage and warmth from IV lines suggests line sepsis; whereas headache and stiff neck suggest meningitis.

Vasopressors

Therapy with vasopressors (e.g., norepinephrine, epinephrine) is expected if a mean arterial blood pressure of greater than 65 mm Hg is not achieved by IV hydration alone. The nurse monitors the MAP, the CVP, O_2 saturation, and SvO_2, as well as the hourly urine output (aim is greater than 0.5 mL/kg/hr) to assess evidence of improving perfusion (improved mental status, peripheral perfusion).

Glucocorticoids

Glucocorticoids have been used in the treatment of septic shock with varying effect for decades. The general concept is that, since septic shock represents a significant physiologic stress, dysfunction of the hypothalamic–pituitary–adrenal axis may be one factor that leads to increased mortality. Several studies have attempted to investigate the effect of administering steroids to septic patients. Drawbacks to steroid administration include increased infection rate and increased risk of superinfection, worsening glycemic control, and impaired wound healing. High-dose corticosteroids do not improve outcomes in the full spectrum of patients with sepsis or ARDS, and the evidence supporting the use of low-dose corticosteroids in septic shock remains controversial (Russell, 2016, p. 690). If the patient has not responded to vasopressor support, corticosteroids may be considered.

Nutritional Therapy and Glycemic Control

Aggressive nutritional supplementation is critical in the management of patients with septic shock because malnutrition further impairs the patient's compensatory mechanisms. Nutritional supplementation should be initiated as soon as possible. Enteral feeding, rather than parental nutrition, is associated with improved outcomes, although reasons for this remain unclear. Evidence is particularly strong for enteral nutrition in surgical patients who are critically ill. Regarding glycemic control, intense insulin therapy increases the risk of higher rates of hypoglycemia and perhaps increased mortality and, therefore, is not recommended (Russell, 2016). However, the nurse assesses and alerts the team when the patient's blood glucose level is outside the range of 140 to 180 mg/dL.

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 in all patients. Nurses should identify patients who are at particular risk for sepsis and septic shock (i.e., 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.

Monitoring and constant reassessment are primary nursing tasks when caring for a patient with septic shock. Vital signs, hemodynamics (CVP, SvO_2, SVR, CO), urinary output, mental status, and physical assessment findings must be reported accurately and in a timely fashion so that interventions can be instituted. 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. Appropriate specimens for culture and sensitivity are often obtained by the nurse.

Elevated body temperature (hyperthermia) is common in patients 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 (greater 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 to the kidneys and liver, serum concentrations of antibiotic agents that are normally cleared by these organs may increase and produce toxic effects. Therefore, the nurse monitors blood levels (antibiotic agent, peak and trough levels of antibiotics (if available), BUN, creatinine, white blood cell count, hemoglobin, hematocrit, platelet levels, coagu-

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lation studies) and reports changes to the provider. Daily weights and close monitoring of serum prealbumin levels help determine the patient's protein requirements. Additionally, the nurse expects that deep vein thrombosis (DVT) and stress ulcer prophylaxis will be instituted.

NEUROGENIC SHOCK

Pathophysiology

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 the vascular smooth muscle to relax or dilate. The patient experiences a predominant parasympathetic stimulation that causes vasodilation lasting for an extended period because of acute interruption of sympathetic function. It is important to understand that the blood volume is adequate, but because the vasculature is dilated, the blood volume is displaced, producing a hypotensive (low BP) state (see Box 54-1, p. 1509). Thus, this is a situation where blood volume is stable, but the drastic dilatation of the vasculature causes a relative hypovolemia. The overriding parasympathetic stimulation that occurs with neurogenic shock causes a severe decrease in the patient's SVR and sometimes bradycardia. Inadequate BP results in the insufficient perfusion of tissues and cells that is common to all shock states.



Nursing Alert

The nurse understands that the preganglionic axons of the SNS have origins in the thoracolumbar region of the spinal cord. Thus, patients with spinal cord injuries in the cervical or upper thoracic spinal cord are at risk for neurogenic shock. Without the tonic sympathetic input to the systemic vasculature, the vagal response of bradycardia and decreased SVR are unopposed and culminate in hypotension. Prompt recognition and treatment is essential to avoid potential organ damage.

Neurogenic shock can be caused by spinal cord injury (commonly above the level of T6), spinal anesthesia, or nervous system damage. It may also result from the depressant action of medications or from lack of glucose (e.g., 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.

Clinical Manifestations and Assessment

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 IV fluid or plasma expanders, vasopressors, and 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 cause of the shock. Further discussion of management of patients with a spinal cord injury is presented in Chapter 45. If hypoglycemia (insulin shock) is the cause, glucose is rapidly administered. Hypoglycemia and the insulin reaction are described further in Chapter 30. Current guidelines recommend maintaining an MAP of 85 to 90 mm Hg for 1 week post injury to promote spinal cord perfusion (Hickey, 2014, p. 400).

Nursing Management

In suspected spinal cord injury, neurogenic shock may be prevented by carefully immobilizing the patient to prevent further damage to the spinal cord. The nurse is aware that orthostatic hypotension caused by loss of vasomotor tone below the level of the spinal cord lesion can occur with position changes. Even slightly raising the head of the bed for a new tetraplegic patient can result in a drastic hypotension (Hickey, 2014).

Nursing interventions are directed toward supporting cardiovascular and neurologic function until the usually transient episode of neurogenic shock resolves. Apply sequential compression devices since pooled blood increases the risk for thrombus formation. The nurse must check the patient daily for any lower extremity pain, redness, tenderness, unilateral edema, and warmth of the calves. If the patient complains of pain, and objective assessment of the calf is suspicious, the patient should be evaluated for DVT. Administration of heparin or low–molecular-weight heparin (Lovenox) may prevent thrombus formation. Passive range of motion of the immobile extremities also 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. In hemorrhagic shock, the nurse is aware that tachycardia will be present versus bradycardia with neurogenic shock.

ANAPHYLACTIC SHOCK

Anaphylactic shock occurs rapidly and is life-threatening. Annual mortality rates of 1,500 to 2,000 deaths in the United States per year are attributed to systemic anaphylaxis (Schwartz, 2016). Medications (e.g., antibiotics, NSAIDs,

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contrast media, and blood products) are triggers in 55% of cases, while food (26%) and insect bites (19%) are other frequent causes; combined they are the most common causes of anaphylaxis (Brown & Turner, 2017; Ferri, 2016). Approximately 6% of children and 3% to 4% of adults have food allergy (Brown & Turner, 2017).

Because anaphylactic shock occurs in patients already exposed to an antigen and who have developed antibodies to it, it can sometimes 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.



Nursing Alert

Latex allergies are a risk for health care workers and patients who have multiple mucosal exposures to latex. Nurses should use nonlatex gloves when possible, and patients at risk (patients with spina bifida, spinal cord injuries, and congenital abnormalities of the genitourinary tract) should be catheterized when needed with nonlatex equipment.

Pathophysiology

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. This reaction provokes mast cells and basophils to release potent vasoactive substances, such as histamine or bradykinin, causing widespread vasodilation, capillary permeability, and potentially catastrophic vascular collapse. Mast cells in any organ system may be involved, depending on the distribution of the instigating stimulus; in general the principal targets include the cardiovascular, cutaneous, respiratory, and GI systems (where mast cells are most abundant) (Schwartz, 2016, p. 1698). An equally important feature of anaphylactic shock is the onset of severe bronchospasm, which causes airway compromise (stridor, wheezing, shortness of breath) and urticaria. A delayed recurrence of the reaction, without re-exposure to the allergen, can sometimes occur. This has been termed a *biphasic reaction* and most commonly occurs 8 to 10 hours after the first symptoms; therefore, patients with severe reactions are generally observed for at least 10 hours.

Clinical Manifestations and Assessment

The clinical manifestations are related to the potent vasoactive and smooth muscle contractile actions due to mast cell release of vasoactive substances. For example, the vasodilatation results in erythema, decreased venous return to the heart, and subsequent hypotension; activation of multiple inflammatory pathways causes fluid extravasation, seen as angioedema, tissue edema, and hypovolemia; whereas the smooth muscle contraction is associated with bronchospasm and abdominal and pelvic cramps. Thus the nurse observes for generalized hives, pruritus or flushing, angioedema, swelling of the tongue and/or uvula, dyspnea, wheezing, stridor, hypoxemia, and reduced blood pressure. GI symptoms, including nausea, vomiting, abdominal cramps, and diarrhea, may precede the more generalized clinical manifestations described previously after ingestion of an antigen (Haupt & Grandhi, 2017, p. 437). Serum mast cell tryptase (MCT) is the only widely available assay used to confirm the diagnosis of anaphylaxis (Brown & Turner, 2017, p. 354).

Medical Management

Treatment of anaphylactic shock requires removing the causative antigen (e.g., discontinuing an antibiotic agent) and administering medications that restore vascular tone. In addition, the health care team will need to provide emergency airway support, position the patient in supine (see Alert below), administer oxygen, provide ventilation support (endotracheal intubation or tracheotomy may be necessary to establish an airway), perform external chest compressions if cardiac arrest occurs, and prepare for early administration of epinephrine. Epinephrine is usually given intramuscularly in the thigh in a dose of 0.01 mg/kg with a usual dose of 0.2 to 0.5 mg for adults (maximum dose of 0.6 mg), which can be repeated every 5 to 30 minutes if the response is inadequate. Epinephrine is given for its vasoconstrictive action as well its effect of reducing bronchospasm (Brown & Turner, 2017). When epinephrine is given early (before the onset of shock), systemic absorption of IM epinephrine is rapid, with a response seen in minutes. IV epinephrine carries more risks, and incorrect dosing has been associated with serious adverse events (Brown & Turner, 2017). However, in patients with severe anaphylaxis, the nurse considers that decreased perfusion to the muscle may result in slow absorption of epinephrine into the circulation; therefore, IV epinephrine may be necessary. Diphenhydramine (Benadryl), given intramuscularly or intravenously, may be administered to reverse the urticaria and pruritus but is not likely to reduce the hypotension of tissue edema (Schwartz, 2016). Nebulized medications, such as albuterol (Proventil), may also be given to reverse histamine-induced bronchospasm. Glucocorticoids may be considered to prevent a rebound, biphasic reaction, although it is unlikely to be beneficial acutely because of the slow onset of action (Ferri, 2016; Schwartz, 2016). IV lines are inserted to provide access for administering fluids and medications. Aggressive IV fluid resuscitation is expected with hypotension. The patient's cardiac status is monitored, and the electrocardiogram is assessed because anaphylaxis and treatments can be associated with arrhythmias and

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cardiac ischemia. Pulse oximetry is also expected to monitor oxygenation status and response to treatment. Anaphylaxis and specific chemical mediators are discussed further in Chapter 38.

Nursing Alert

It is important to note that Fowler's (upright) or semi-Fowler's (semi recumbent) position has been associated with fatal anaphylaxis. Changing the patient's position to one that is upright reduces venous return to the heart and is proposed to exacerbate the pathophysiologic processes occurring in anaphylaxis; therefore, keeping a patient flat is suggested to improve venous return to the heart, a key aspect of the initial response to anaphylaxis (Brown & Turner, 2017).

Nursing Alert

Nurses understand that epinephrine is typically supplied as 1 mg/mL (1:1,000 dilution). Most authorities recommend that 0.3 to 0.5 mg of the 1:1,000 solution (0.3 to 0.5 mL) be given intramuscularly in the anterolateral aspect of the thigh.

Nursing Management

The nurse plays an important role in preventing anaphylactic shock. The nurse must assess all patients for allergies or previous reactions to antigens (e.g., 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 the 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. Allergy to penicillin is one of the most common causes of anaphylactic shock. Patients who have a penicillin allergy may also develop an allergy to similar medications. For example, they may react to cefazolin sodium (Ancef) because it has a similar antimicrobial action of attaching to the penicillin-binding proteins found on the walls of infectious organisms. 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.

In the hospital and outpatient diagnostic testing sites, the nurse must identify patients who are at risk for anaphylactic reactions to the contrast agents (radiopaque, dye-like substances that may contain iodine) that are used for diagnostic tests. These include patients with a known allergy to iodine or fish and those who have had previous allergic reactions to contrast agents. This information must be conveyed to the staff at the diagnostic testing site, including radiology staff. The nurse anticipates that "pre-medication" regimens are likely to be administered in patients with known contrast allergies.

Nursing Alert

Pre-medication with steroids and Benadryl may be recommended for patients who have had a previous reaction to contrast agent (gadolinium-based contrast or iodinated contrast) and a similar contrast will be administered (ACR, 2016).

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 cardiopulmonary arrest occurs. In addition to monitoring the patient's response to treatment, the nurse assists with intubation if needed, monitors the hemodynamic status, ensures IV access for administration of medications, administers prescribed medications and fluids, and documents treatments and their effects.

Nursing Alert

If IV epinephrine is required, the nurse carefully titrates the medication to prevent a hypertensive surge. Symptoms of tachycardia, pallor, diaphoresis, and arrhythmias in a patient with normal or raised blood pressure are indicative of epinephrine toxicity. The provider should be notified and the infusion rate reduced (Brown & Turner, 2017). Additionally, the nurse observes carefully the effect of epinephrine in patients with cerebrovascular or coronary artery disease, hypertension, diabetes, hyperthyroidism, cardiomyopathy, or narrow-angle glaucoma, in whom adverse events, such as MI, stroke, or pulmonary edema, can be precipitated (Schwartz, 2016, p. 1702).

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. Prescriptions for prefilled epinephrine syringe (EpiPen or EpiPen Jr.) should be given, and the patient and significant others are instructed on how to administer the EpiPen and that the patient needs to carry it on his or her person at all times. After recovery from anaphylaxis, the patient and family require an explanation of the event. Furthermore, the nurse provides instruction and counseling about avoiding future exposure to antigens and administering emergency medications to treat anaphylaxis.

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OBSTRUCTIVE SHOCK

Obstructive shock is caused by a physical obstruction to blood flow, either in the heart or major blood vessels. This causes a decrease in CO and thus a decrease in tissue perfusion. Common causes of obstructive shock include cardiac tamponade, tension pneumothorax, and pulmonary embolism (PE). Obstructive shock is common in the setting of trauma.

Pathophysiology

In the case of tension pneumothorax or hemothorax, air or blood has entered the pleural space and caused a restriction in lung expansion. If enough air is trapped, then eventually pressure builds up and compresses the vessels and myocardium. This compression reduces preload (volume of blood) and increases afterload, in addition to restricting the expansion of the myocardium during filling. Clinical presentation may include JVD, crepitus (with pneumothorax), dyspnea, chest pain, tachycardia and tachypnea, tracheal shifting **away** from the tension pneumothorax, and decreased breath sounds on the affected side. Tension pneumothorax is common in trauma but can also develop spontaneously or as a complication from a procedure such as central line placement. Patients who are mechanically ventilated are at higher risk of developing a clinically significant pneumothorax due to increased ventilatory pressures.

Cardiac tamponade results from excessive blood or fluid inside the pericardium, the sac that surrounds the heart. This can result from a traumatic or infectious cause with a resultant restriction on the pumping ability of the heart. Signs and symptoms of cardiac tamponade include narrowing pulse pressure (difference between the systolic and diastolic pressure; normally 40 mm Hg), chest pain, distant or muffled heart sounds, JVD, hypotension, and tachycardia.

A PE is a clot that gets lodged in the pulmonary vasculature and causes ischemia to the capillary beds. If large enough, the embolism interferes significantly with pulmonary blood flow, which causes blood to back up into the right side of the heart. As the clot increases the pressure in the pulmonary artery, the right heart must beat faster and harder to overcome the pressure. Overall, CO is decreased as the patient goes into right-sided heart failure. Clots that have traveled from the lower extremities cause most PEs. Signs and symptoms of PE include pleuritic chest pain, shortness of breath, tachycardia, and hypoxia (refer to Chapter 10 for more details).

Medical Management

In the case of cardiac tamponade and tension pneumothorax, a procedure done by a qualified clinician should be performed immediately to relieve the obstruction. If the patient is experiencing obstructive shock, then these procedures are done emergently at the bedside. A chest radiograph is often used to diagnose a pneumothorax, although computed tomography (CT) scans and even ultrasound are more sensitive. A chest tube or needle decompression is the primary procedure to relieve tension pneumothorax. Cardiac tamponade is diagnosed similarly, with echocardiography being the most effective. Pericardiocentesis is the treatment for cardiac tamponade and involves inserting a needle into the pericardium to draw off fluid or blood.

The treatment of PE is usually aimed toward preventing clot expansion, although if the patient is experiencing obstructive shock, then other treatment, such as thrombolytic drugs or surgical embolectomy, may be considered. Depending on the severity of the PE, mechanical ventilation and hemodynamic support may be needed to stabilize the patient. PE is usually diagnosed using a CT angiogram, but other imaging and laboratory tests can be used as well.

Nursing Management

Because patients with tension pneumothorax and cardiac tamponade who are experiencing obstructive shock will need an emergent procedure, nurses should focus on facilitating that process. This includes collecting and setting up equipment, communicating effectively with staff, and helping gain consent from the patient and/or family. Careful monitoring of the patient prior to, during, and after the procedure is essential. If a chest tube is placed, then a chest drainage system (e.g., Pleuravac) will need to be set up and connected to suction as ordered. The chest tube setup needs to be monitored to ensure that it is functioning correctly and also to record output. A postprocedure chest radiograph will be ordered, and the results should be followed closely.

Nursing care of a patient with a PE that is causing obstructive shock involves monitoring and initiating ventilatory and hemodynamic support similar to what is done for patients with other types of shock. A primary function of the nurse in this situation is to facilitate diagnostic testing and imaging and then institute treatment in a timely manner. Patients with a PE large enough to cause obstructive shock have a very high mortality and often die within an hour of presentation.

Patients who experience obstructive shock are often quite suddenly acutely ill. This affects the entire health care team as decisions need to be made quickly, and the nurse should work hard to maintain a calm environment. Since obstructive shock usually has a sudden onset, families are quite often surprised when given news about massive PE or other causes of obstructive shock. Having other support personnel in place, such as a social worker or a chaplain, to provide support is advised.

MULTIPLE ORGAN DYSFUNCTION SYNDROME

MODS is altered organ function in patients who are acutely ill that requires medical intervention to support continued

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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. It is defined as severe organ dysfunction of two or more organ systems lasting at least 24 to 48 hours in the setting of sepsis, trauma, burns, or severe inflammatory conditions (Tiesi et al., 2016). It is important to note that the definition includes the number of dysfunctional organs and duration. Mortality rates when two organ systems are involved are 40%; and persistent dysfunction (more than 72 hours) present in three organ systems can result in a mortality risk of 80%.

Nursing Alert

Clinicians may use the SOFA scale to rapidly identify adult patients with organ dysfunction and risk for poor outcomes. The acronym stands for Sequential (Sepsis-related) Organ Failure Assessment (SOFA) and includes parameters such as the PaO_2/FiO_2 ratio (respiratory system), the platelet count (coagulation), bilirubin level (hepatic system), MAP (cardiovascular system), Glasgow Coma Scale (CNS system), and creatinine and urine output (renal system). A higher SOFA score is associated with an increased probability of mortality (Singer et al., 2016).

Pathophysiology

The inflammation, tissue injury, and other sequelae associated with MODS are thought to be caused by an unregulated host response. MODS may result from any form of shock because of inadequate tissue perfusion. As previously described, in patients with shock, all organ systems suffer

Diagnosis	Systemic Blood Pressure	Heart Rate	Central Venous Pressure (CVP)	Pulmonary Artery Pressure	Cardiac Output (CO)	Systemic Vascular Resistance (SVR)
Cardiogenic: Myocardial Infarction (MI), Cardiac Tamponade, Pulmonary Emboli	↓	↑	↑	↑	↓	↑
Hypovolemic: Hemorrhage, Diarrhea, Burns, Dehydration	↓	↑	↓	↓	↓	↑
Septic: Severe Infection	↓	↑	↓	↓	↑ ↓	↓
Anaphylactic: Type 1 Hypersensitivity Reaction	↓	↑	↓	↓	↓	↓
Neurogenic: Brain Damage, Spinal Cord Injury	↓	↓	↓	↓	↓	↓

FIGURE 54-5 Types of shock. Shock results from (1) an inability of the heart to pump adequately (cardiogenic shock); (2) decreased effective blood volume as a consequence of severely reduced blood or plasma volume (hypovolemic shock); or (3) widespread vasodilation (septic; anaphylactic or neurogenic shock). Increased vascular permeability may complicate vasodilation by contributing to reduced effective blood volume. (Adapted from Lawrence, P., Bell, R., Dayton, M., & Hebert, J. (2012). *Essentials of general surgery* (5th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.)

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damage from a lack of adequate perfusion, which can result in organ failure.

Clinical Manifestations and Assessment

Various causes of MODS have been identified, including dead or injured tissue, infection, and perfusion deficits. However, it is not possible as yet to predict which patients will develop MODS, partly because much of the organ damage occurs at the cellular level and, therefore, cannot be directly observed or measured. The most common types of organ dysfunction seen with MODS are acute kidney failure and ARDS. However, organ failure can affect the other body systems:

- Hepatic: jaundice, elevated liver function tests (LFTs), decreased albumin and elevated PT
- Hematologic: decreased platelets, elevated PT/APTT, increased D-dimer
- Central nervous system (CNS): altered level of consciousness (LOC), confusion, psychosis
- Respiratory: tachypnea, hypoxemia, PaO_2/FiO_2 greater than 300 (see Chapter 10)
- Cardiovascular: tachycardia, hypotension, altered CVP and pulmonary artery pressure (PAP) readings

Advanced age, malnutrition, and coexisting diseases appear to increase the risk of MODS in patients who are acutely ill.

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 associated with aging and the natural degenerative process, especially immune compromise. 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 in septic shock. Primary nursing interventions are aimed at early and aggressive hemodynamic support 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 3).

Refer to Figure 54-5 for a review of the various forms of shock.

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CHAPTER REVIEW

Critical Thinking Exercises

1. You administer an antibiotic to a patient before he goes to surgery. The patient's chart states that he has no known drug allergies. After 15 minutes, 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 clinical signs and symptoms would you look for to determine if he is experiencing anaphylactic shock? What nursing interventions and medical treatments would you anticipate?
2. An elderly woman is admitted to the medical floor for a urinary tract infection. You notice that she has become steadily more tachycardic and her urine output has dropped to 15 cc for the last hour. How would you assess this patient for the possibility of sepsis?
3. While working in the emergency department, you see a victim of a motor vehicle accident brought in by ambulance. His pelvis appears deformed, he is pale, diaphoretic, and his heart rate is 124. What type of shock is this patient most likely in? What led you to that conclusion? Name some interventions that would help this patient.
4. You are taking care of a patient in the ICU with a spinal cord injury. His BP has not been responsive to repeated fluid boluses. What type of shock is he mostly likely experiencing? Name some appropriate interventions.

NCLEX-Style Review Questions

1. During a conversation about a patient, a new nurse states to the charge nurse, "His BP is 92/50, so he can't be in shock." Which response by the charge nurse indicates that the new nurse does not completely understand the symptoms of shock?
   a. Patients can have a near-normal BP, yet still have inadequate tissue perfusion.
   b. The cutoff for shock is a systolic pressure of 100 mm Hg.

c. Shock is determined by mean arterial pressure.
d. Shock is determined by a heart rate of over 100.

2. A patient with a GI bleed arrives at the emergency department with the following vital signs: Temperature 37.4°C, HR 112, RR 26, BP 88/44. The nurse recognizes that this patient is most likely experiencing or at risk for which type of shock?
a. Anaphylactic
b. Neurogenic
c. Hypovolemic
d. Septic

3. A patient comes in to the urgent care center complaining of shortness of breath and dizziness after being stung by a bee. The patient loses consciousness in the waiting room. The receptionist has called 911. The nurse anticipates that which medication will be considered first?
a. Diphenhydramine (Benadryl)
b. Phenytoin (Dilantin)
c. Epinephrine IV
d. Epinephrine IM

Try these additional resources, available at **http://thepoint.lww.com/Honan2e**, to enhance your learning and understanding of this chapter:
- NCLEX-Style Student Review Questions
- Journal Articles

4. In early goal-directed therapy for shock, the nurse knows that which parameter is the highest priority when evaluating effectiveness of therapy?
a. Heart rate and BP
b. SvO_2 and O_2 saturation
c. Base deficit and hematocrit
d. SvO_2 and CVP

5. A patient develops low BP and tachycardia and is very agitated. The nurse remembers that a central line was placed 3 hours ago. The patient's lung sounds are decreased on one side, and tracheal deviation is present. The nurse realizes that this patient is most likely experiencing which type of shock?
a. Hypovolemic
b. Obstructive
c. Anaphylactic
d. Neurogenic



REFERENCES AND SELECTED READINGS

References and selected readings associated with this chapter can be found on the website that accompanies the book. Visit **http://thepoint.lww.com/Honan2e** to access the references and other additional resources associated with this chapter.

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