Treatment

Following a blood culture/rapid test to identify the causative agent, antimicrobial drugs are given, usually for a minimum of 4 weeks, to eradicate the infection completely. Other medication to support heart function is usually required.

THINK ABOUT 12-15

Describe the possible destination of an embolus from the mitral valve.

Pericarditis

Pathophysiology

Pericarditis may be acute or chronic and is usually secondary to another condition in either the heart or the surrounding structures. Pericarditis can be classified by cause or by the type of exudate associated with the inflammation. Acute pericarditis may involve a simple inflammation of the pericardium, in which the rough, swollen surfaces cause chest pain and a friction rub (a grating sound heard on the chest with a stethoscope). In some cases, an *effusion* may develop, with a large volume of fluid accumulating in the pericardial sac (Fig. 12-29). This fluid may be serous as with inflammation, may be fibrinous and purulent as with infection, or may contain blood (hemopericardium) as with trauma or cancer.

Small volumes of fluid in the pericardium have little effect on heart function, but a large amount of fluid that accumulates rapidly may compress the heart and impair its expansion and filling, thus decreasing cardiac output (*cardiac tamponade*). The right side (low-pressure side) of the heart is affected first, causing increased pressure in the systemic veins and, if acute, distended neck veins. If fluid accumulates slowly, the heart adjusts, and a very large amount can build up before signs appear. A radiograph would show the enlargement of the heart.

Chronic pericarditis results in formation of adhesions between the pericardial membranes which may become constrictive, causing the pericardium to become a tight fibrous enclosure, thus limiting movement of the heart.

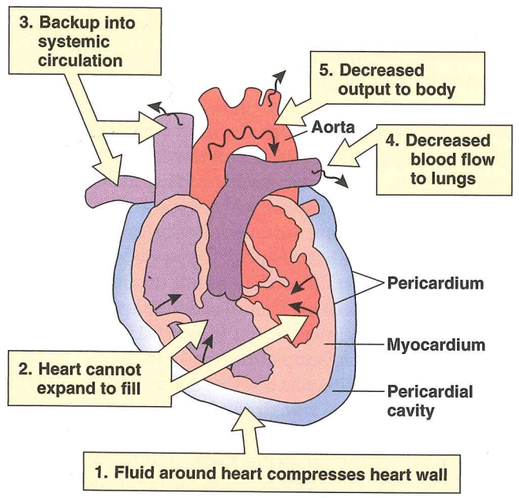


FIGURE 12-29 Effects of pericardial effusion

Etiology

Acute pericarditis may be secondary to open heart surgery, myocardial infarction, rheumatic fever, systemic lupus erythematosus, cancer, renal failure, trauma, or viral infection. The fibrous tissue of chronic pericarditis often results from tuberculosis or radiation to the mediastinum. Inflammation or infection may develop from adjacent structures, for example, pleurisy or pneumonia in the lungs. Effusion may be secondary to hypoproteinemia resulting from liver or kidney disease.

Signs and Symptoms

Signs vary with the underlying problem and its effects on the pericardium. Tachycardia is present, and chest pain, dyspnea, and cough are common signs. Electrocardiogram changes and a friction rub may be present.

Effusion and cardiac tamponade lead to distended neck veins, faint heart sounds, and pulsus paradoxus, in which systolic pressure drops 10 mmHg during inspiration.

Chronic pericarditis causes fatigue, weakness, and abdominal discomfort owing to systemic venous congestion.

Treatment

First, the primary problem must be treated successfully. Fluid may be aspirated from the cavity (paracentesis) and analyzed to determine the cause. If effusion is severe, immediate aspiration of the excess fluid may be required to prevent tamponade and shock.

THINK ABOUT 12-16

Explain the process by which a large volume of fluid in the pericardial cavity decreases cardiac output.

Vascular Disorders

Arterial Disorders

Hypertension

Pathophysiology

Hypertension, or high blood pressure, in both its primary and secondary forms is a very common problem. Recent estimates indicate one in three adults

has high blood pressure. Within this group, one third are undiagnosed, and some are not controlled. Men are more likely to have high blood pressure than women until age 55; after menopause the proportion of women exceeds that of men. Another third of the adult population are considered to have *prehypertension*, with blood pressure in the high normal range and not currently prescribed medication. African Americans have a higher prevalence of hypertension; the onset is earlier and the average blood pressure is higher. Because of the insidious onset and mild signs, hypertension is often undiagnosed until complications arise and has been called the "silent killer." However, it is hoped that the availability of self-testing machines and other screening programs will aid in an early diagnosis. Compliance with treatment measures may not occur until the problem is severe enough to interfere with function.

Hypertension is classified in three major categories:

1. Primary or *essential* hypertension is idiopathic and is the form discussed in this section.
2. *Secondary* hypertension results from renal (e.g., nephrosclerosis) or endocrine (e.g., hyperaldosteronism) disease, or pheochromocytoma, a benign tumor of the adrenal medulla or SNS chain of ganglia. In this type of hypertension, the underlying problem must be treated to reduce the blood pressure.
3. *Malignant* or *resistant* hypertension, the third type, is an uncontrollable when treated with three or more drugs, severe, and rapidly progressive form with many complications. Diastolic pressure is very high. Sometimes hypertension is classified as systolic or diastolic, depending on the measurement that is elevated. For example, elderly persons with loss of elasticity in the arteries frequently have a high systolic pressure and low diastolic value.

Essential hypertension develops when the blood pressure is consistently above 140/90. This figure may be adjusted for the individual's age. The diastolic pressure is important because it indicates the degree of peripheral resistance and the increased workload of the left ventricle. The condition may be mild, moderate, or severe.

In essential hypertension there is an increase in arteriolar vasoconstriction, which is attributed variously to increased susceptibility to stimuli or increased stimulation or perhaps a combination of factors. A very slight decrease in the diameter of the arterioles causes a major increase in peripheral resistance, reduces the capacity of the system, and increases the diastolic pressure or afterload substantially. Frequently, vasoconstriction leads to decreased blood flow through the kidneys, leading to increased renin, angiotensin, and aldosterone secretion. These substances increase vasoconstriction and blood volume, further increasing blood pressure (Fig. 12-30). If this cycle is not broken, blood pressure can continue to increase.

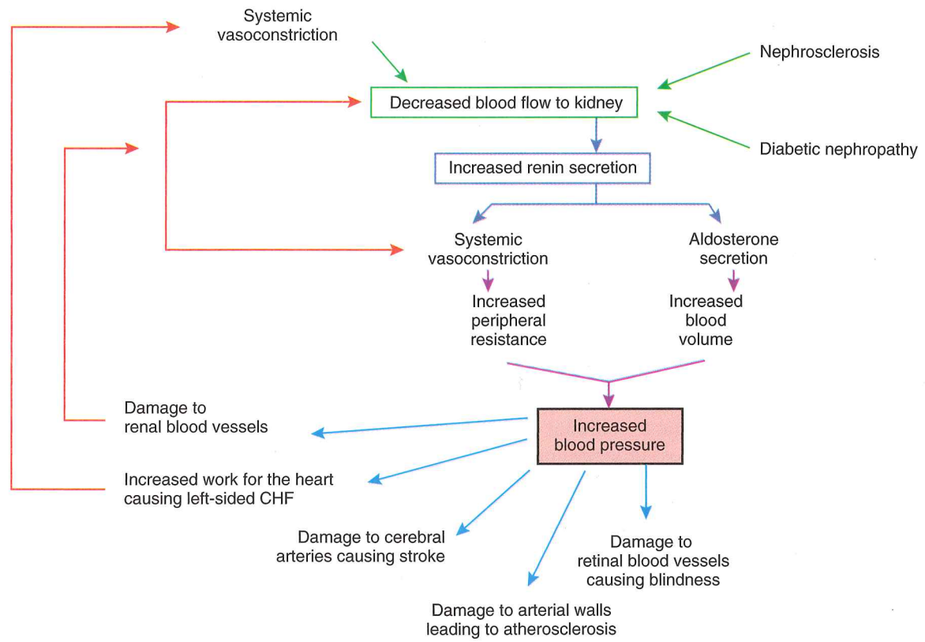


FIGURE 12-30 Development of hypertension

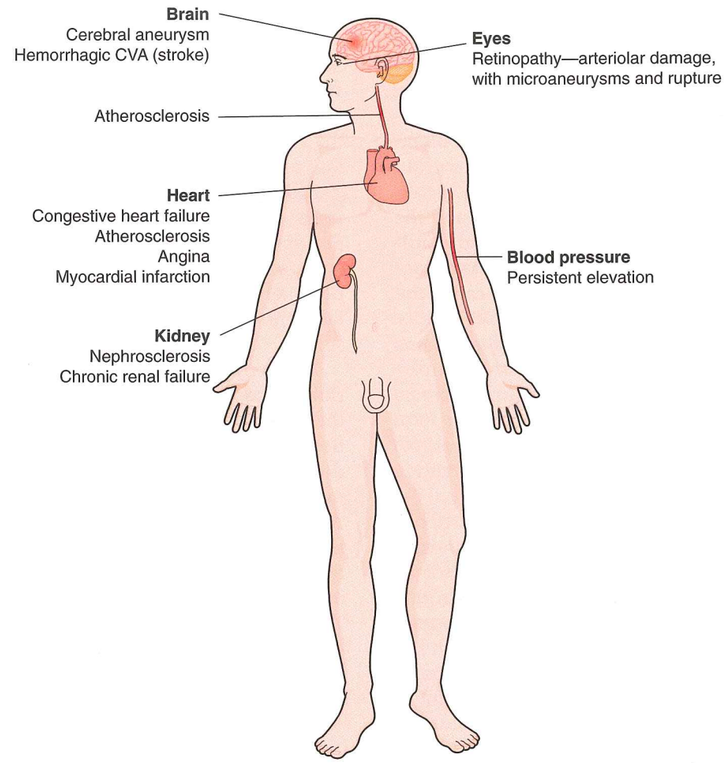


FIGURE 12-31 Effects of uncontrolled hypertension

Over a long period of time, the increased blood pressure causes damage to the arterial walls. They become hard and thick *(sclerotic)*, narrowing the lumen. The wall may dilate or tear, forming an aneurysm, or encourage atheroma formation. Blood supply to the involved area is reduced, leading to ischemia and necrosis with loss of function. In many cases, the progressive changes are asymptomatic until well advanced.

The areas most frequently damaged by elevated pressure are the kidneys, brain, and retina. One area that is easily checked through the pupil of the eye is the retina, where the blood vessels can easily be observed for sclerotic changes and rupture (Fig. 12-31). The end result of poorly controlled hypertension can be chronic renal failure, stroke due to hemorrhage, loss of vision, or congestive heart failure. Lifespan may be considerably shorter, particularly in men, when hypertension is not controlled.

Etiology

Even in idiopathic hypertension, the form discussed here, many factors appear to predispose to the condition. The incidence increases with age, although hypertension does occur in children. Men are affected more frequently and more severely, but the incidence in women increases after middle age. Genetic factors are reflected by the fact that African Americans have a higher incidence than do Caucasians and experience a more severe form of hypertension. There are also familial trends, but these reflect lifestyle characteristics as well as heredity.

Other factors implicated in the development of essential hypertension include high sodium intake, excessive alcohol intake (small amounts of alcohol appear to decrease blood pressure), obesity, and prolonged or recurrent stress.

Signs and Symptoms

Hypertension is frequently asymptomatic in the early stages, and the initial signs are often vague and nonspecific. They include:

- Fatigue
- Malaise
- Morning headache
- Consistently elevated blood pressure under various conditions is the key sign of hypertension. The complications are also asymptomatic until they are well advanced.

Treatment

Essential hypertension is usually treated in a sequence of steps, beginning with lifestyle changes, as needed, to reduce salt intake, reduce body weight and stress, and generally increase cardiovascular fitness.

The recommendations and drugs selected are individualized. Mild diuretics such as the thiazide diuretics, which also have an antihypertensive action, are suggested for the next stage. Physicians recommend ACE inhibiters for many as the initial treatment. Subsequently, one or more drugs may be added to the regimen until blood pressure is reduced. Combinations of drugs with different actions are quite effective, and the adverse effects are minimal. The choice of drug also depends on the individual situation. For example, a patient with a high serum sodium level needs a stronger diuretic, such as furosemide, and a patient with high renin levels may take an ACE inhibitor. Other antihypertensive agents block the sympathetic stimulation in various ways: alpha$_1$-blockers causing vasodilation, calcium blockers reducing heart action and peripheral resistance, and beta-blockers reducing heart action and sometimes renin release (see Table 12-1).

Patient compliance can be difficult when no obvious signs of illness are present. However it is important to continue to follow all the physician's recommendations to prevent unseen damage and complications. Unfortunately, some of the drugs do have significant side effects, such as nausea, erectile dysfunction, and orthostatic hypotension. Orthostatic hypotension results from the lack of reflex vasoconstriction when rising from a supine position causing a decrease in blood flow to the brain. This results in dizziness and fainting and can result in falls. Rising slowly to a standing position and using support will decrease the risk of falls. Diuretics may cause increased urinary frequency in the morning and generalized weakness. Beta-blockers may prevent the heart rate from increasing with exercise. This interference with normal responses can lead to misinterpretation of the results of exercise stress testing.

Prognosis depends on treating any underlying problems and maintaining constant control of blood pressure to prevent complications.

THINK ABOUT 12-17

a. State the cause of elevated blood pressure in essential hypertension.
b. Describe the long-term effects of uncontrolled hypertension.
c. Explain why orthostatic (postural) hypotension may occur with vasodilator drugs.
d. Explain how compensation by the renin-angiotensin pathway aggravates hypertension.

Peripheral Vascular Disease and Atherosclerosis

Pathophysiology

Peripheral vascular disease refers to any abnormality in the arteries or veins outside the heart. The cause, development, and effects of atheromas have been discussed previously in this chapter (see Figs. 12-8 to 12-11). The most common sites of atheromas in the peripheral circulation are the abdominal aorta and the femoral and iliac arteries (see Fig. 10-1), where partial occlusions may impair both muscle activity and sensory function in the legs. Total occlusions may result from a thrombus obstructing the lumen or breaking off (an embolus) and eventually obstructing a smaller artery. Loss of blood supply in a limb leads to necrosis, ulcers, and gangrene, which is a bacterial infection of necrotic tissue.

Signs and Symptoms

- Increasing fatigue and weakness in the legs develop as blood flow decreases.
- *Intermittent claudication*, or leg pain associated with exercise due to muscle ischemia, is a key indicator. Initially pain subsides with rest. As the obstruction advances, pain becomes more severe and may be present at rest, particularly in the distal areas such as the feet and toes.
- Sensory impairment may also be noted as paresthesias, or tingling, burning, and numbness.
- Peripheral pulses distal to the occlusion (e.g., the popliteal and pedal pulses) become weak or absent (see Fig. 12-4).
- The appearance of the skin of the feet and legs changes, with marked pallor or cyanosis becoming evident when the legs are elevated and rubor or redness when they are dangling. The skin is dry and hairless, the toenails are thick and hard, and poorly perfused areas in the legs or feet feel cold.

Diagnostic Tests

Blood flow can be assessed by Doppler studies (ultrasonography) and arteriography. Plethysmography measures the size of limbs and blood volume in organs or tissues.

Treatment

Treatment has several aspects, including slowing the progress of atherosclerosis, maintaining circulation in the leg, and treating complications.

- Reduction of serum cholesterol levels is recommended.
- Thrombus formation can be reduced by platelet inhibitors or anticoagulant medications.
- Cessation of smoking, which causes increased platelet adhesion, is highly recommended.
- An exercise program can be helpful in preserving existing circulation.
- Maintaining a dependent position for the legs can improve arterial perfusion.

- Peripheral vasodilators such as calcium blockers may be helpful because they may enhance the collateral circulation.
- Surgical procedures to restore blood flow include bypass grafts using a vein or synthetic material, angioplasty to reduce plaques, or **endarterectomy** (removal of the intima and obstructive material).
- Care should be taken to avoid any skin trauma, and regular examination of the feet is important to avoid pressure from shoes, especially if there is sensory impairment. Specially fitted shoes may be required.
- Gangrenous ulcers can be treated with antibiotics and débridement of dead tissue.
- Amputation of a gangrenous toe or foot is often required to prevent spread of the infection into the systemic circulation and to relieve the severe pain of ischemia. In many cases, multiple amputations are required, beginning with a toe, then a foot, lower leg, and so on. Vascular disease is the primary reason for amputation. Healing is very slow because of the poor blood supply, and a prosthesis may be difficult to fit and maintain unless circulation can be improved.

THINK ABOUT 12-18

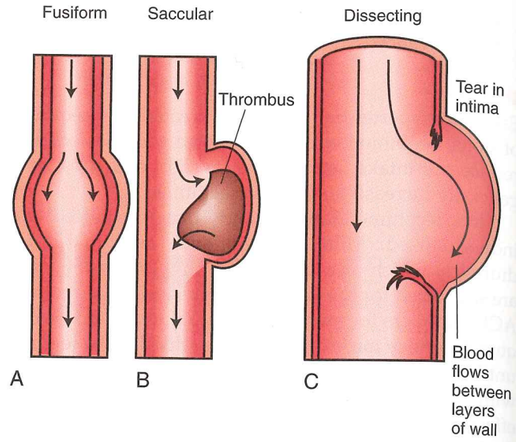
a. What is the cause of weak peripheral pulses when the iliac artery is blocked?
b. Why should the feet be carefully inspected on a daily basis in arterial PVD?
c. How does gangrene develop in arterial PVDs and why may healing following amputation to treat gangrene be reduced?

Aortic Aneurysms

Pathophysiology

An aneurysm is a localized dilatation and weakening of an arterial wall. The most common location is either the abdominal or thoracic aorta. The aneurysm may take different shapes: a saccular shape is a bulging wall on one side, whereas a *fusiform* shape is a circumferential dilatation along a section of artery (Fig. 12-32). *Dissecting* aneurysms develop when there is a tear in the intima, allowing blood to flow along the length of the vessel between the layers of the arterial wall. Aneurysms also occur in the cerebral circulation and are discussed in Chapter 14.

The aneurysm develops from a defect in the medial layer, often associated with turbulent blood flow at the site, from a bifurcation, or from an atheroma. Trauma such as a motor vehicle accident may result in tearing of tissues. Syphilis may also damage the tissues in the arterial wall. Over time the dilatation enlarges, particularly if hypertension develops. Frequently, a thrombus forms in the dilated area, obstructing branching arteries



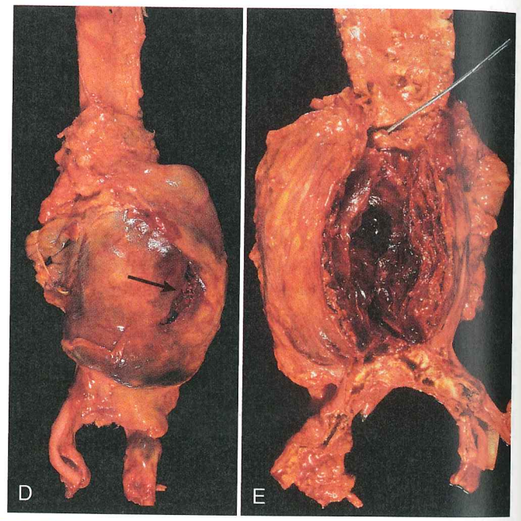


FIGURE 12-32 **A-C,** Types of aortic aneurysms. **D,** External view of abdominal aortic aneurysm with arrow marking rupture. **E,** Open view with probe marking rupture. Note thin bulging wall of aneurysm and lumen filled with thrombus. *(**D** and **E** from Kumar V, Abbas AK, Fausto M:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

such as the renal arteries, or becoming a source of embolus. Many aneurysms eventually rupture, causing massive hemorrhage (see Fig. 12-32*E*).

Etiology

Common causes are atherosclerosis, trauma (particularly car accidents), syphilis and other infections, as well as congenital defects. Hypertension is present in half the patients diagnosed with aortic aneurysms.

Signs and Symptoms

Aneurysms are frequently asymptomatic for a long period of time until they become very large or rupture. Abdominal aneurysms are sometimes detected as palpable pulsating masses with bruits (abnormal sounds). In certain locations, earlier diagnosis may be achieved if a large aneurysm compresses the nearby structures, causing signs such as dysphagia from pressure on the esophagus or pain if a spinal nerve is compressed.

Rupture occasionally leads to moderate bleeding but most often causes severe hemorrhage and death. Signs include severe pain and indications of shock. A dissecting aneurysm causes obstruction of the aorta and its branches as the intima peels back and blood flow is diverted between the layers. The dissection tends to progress down the aorta and sometimes back toward the heart as well. Dissection causes severe pain, loss of pulses, and organ dysfunction, as normal blood flow is lost. Many dissecting aneurysms ultimately rupture.

Diagnostic Tests

Radiography, ultrasound, CT scans, or MRI confirm the problem.

Treatment

Pending surgery, it is of critical importance to maintain blood pressure at a normal level, preventing sudden elevations due to exertion, stress, coughing, or constipation. In some cases, small tears may occur before a major rupture; these need immediate surgical repair. Surgical repair with resection and replacement with a synthetic graft can prevent rupture.

Venous Disorders

Varicose Veins

Pathophysiology

Varicosities are irregular dilated and tortuous areas of the superficial or deep veins (see Fig. 10-2). The most common location is the legs, but varicosities are also found in the esophagus (esophageal varices) and the rectum (hemorrhoids).

Varicose veins in the legs may develop from a defect or weakness in the vein walls or in the valves (Fig. 12-33). Long periods of standing during which the pressure within the vein is greatly elevated can also lead to varicosities. Superficial veins lack the muscle support of the deep veins. If a section of vein wall is weak, eventually the excessive hydrostatic pressure of blood under the influence of gravity causes the wall to stretch or dilate. The weight of blood then damages the valve below, leading to backflow of blood into the section distal to the starting point. If the basic problem is a defective valve, reflux of blood into the section of vein distal to the valve occurs, the overload distending and

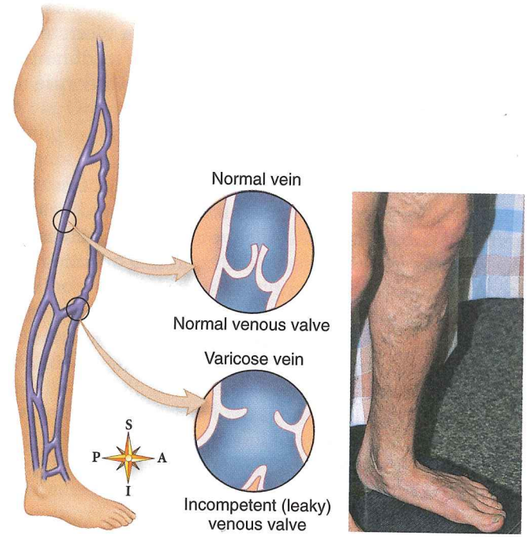


FIGURE 12-33 Varicose veins. **A,** Veins near the surface of the body—especially in the legs—may bulge and cause venous valves to leak. **B,** Photograph showing varicose veins on the surface of the leg. *(**A**, From Patton KT, Thibodeau GA:* Anatomy & Physiology, *ed 8, St. Louis, 2013, Mosby. **B,** From Kumar V, Abbas A, Fausto N:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

stretching the walls. The continued back pressure of blood in the leg veins leads to progressive damage down the vein. Some blood may be diverted into other veins, such as blood flowing from the deep veins through connecting veins into the superficial veins, further extending the damage. Varicosities can predispose to thrombus formation in the presence of other contributing factors such as immobility.

Etiology

A familial tendency to varicose veins is probably related to an inherent weakness in the vein walls. The superficial leg veins are frequently involved because there is less muscle support for these veins. Valves may be damaged by trauma, intravenous administration of fluids, or thrombophlebitis. Many factors can increase pressure in the leg veins, such as standing for long periods of time, crossing the legs, wearing tight clothing, or pregnancy.

Signs and Symptoms

Superficial varicosities on the legs appear as irregular, purplish, bulging structures. There may be edema in the feet as the venous return is reduced. Fatigue and aching are common as the increased interstitial fluid interferes with arterial flow and nutrient supply (see Chapter 2).

Increased interstitial fluid or edema also leads to a shiny, pigmented, and hairless skin, and varicose ulcers may develop as arterial blood flow continues to diminish leading to skin break down. Healing is slow because of impaired blood flow.

Treatment

Treatment is directed toward keeping the legs elevated and using support stockings to encourage venous return and relieve discomfort. Restrictive clothing and crossing the legs should be avoided. When standing or sitting for long periods, intermittent voluntary muscle contractions or position changes are helpful. For more severe varicosities, sclerosing agents that obliterate the veins or surgical vein stripping may be tried, rerouting the blood to functional veins.

THINK ABOUT 12-19

a. Compare the ideal position in a chair for a client with arterial obstruction with that for a client with varicose veins.
b. Explain how leg ulcers may develop in people with varicose veins.

Thrombophlebitis and Phlebothrombosis

Pathophysiology

The terms *thrombophlebitis* and *phlebothrombosis,* as well as *phlebitis* and *thromboembolic disease* are often used interchangeably. It can be difficult to differentiate the two conditions, but sometimes there is a significant difference in the predisposing factors, early signs, and risks of emboli.

Thrombophlebitis refers to the development of a thrombus in a vein in which inflammation is present. The platelets adhere to the inflamed site, and a thrombus develops. In phlebothrombosis, a thrombus forms spontaneously in a vein without prior inflammation, although inflammation may develop secondarily in response to thrombosis. The clot is less firmly attached in this case, and its development is asymptomatic or silent.

Several factors usually predispose to thrombus development:

- The first group of factors involves stasis of blood or sluggish blood flow, which is often present in people who are immobile or where blood flow is constricted by clothing or other devices.
- Endothelial injury, which may have arisen from trauma, chemical injury, intravenous injection, or inflammation, is another factor.
- The third factor involves increased blood coagulability, which may result from dehydration, cancer, pregnancy, or increased platelet adhesion.

The critical problem is that venous thrombosis may lead to pulmonary embolism (see Chapter 13). A piece of thrombus (often the tail) breaks off, usually because of some activity, and flows in the venous blood returning to the heart. The first smaller blood vessels along the route are those of the lungs, where the clot lodges, obstructing the pulmonary circulation and causing both respiratory and cardiovascular complications. Sudden chest pain and shock are indicators of pulmonary embolus.

THINK ABOUT 12-20

Based on predisposing factors, explain why the elderly, immobile or extremely obese individuals often experience thrombophlebitis or phlebothrombosis.

Signs and Symptoms

Often thrombus formation is unnoticed until a pulmonary embolus occurs, with severe chest pain and shock. Thrombophlebitis in the superficial veins may present with aching or burning and tenderness in the affected leg. The leg may be warm and red in the area of the inflamed vein. A thrombus in the deep veins may cause aching pain, tenderness, and edema in the affected leg as the blood pools distal to the obstructing thrombus. A positive Homans' sign (pain in the calf muscle when the foot is dorsiflexed) is common, but not always reliable. Systemic signs such as fever, malaise, and leukocytosis may be present.

Treatment

Preventive measures, such as exercise, elevating the legs, and minimizing the effects of primary conditions, are important. Depending on the particular situation, compression or elastic stockings as well as exercise may be needed to reduce stasis. Anticoagulant therapy, including heparin, fibrinolytic therapy, and surgical interventions such as thrombectomy, may be used to reduce or remove the clot and prevent embolization.

Shock

Shock or hypotension results from a decreased *circulating* blood volume, leading to decreased *tissue perfusion* and general hypoxia. In most cases, cardiac output is low. There are several methods of classifying shock. Shock is most easily classified by the cause, which also indicates the basic pathophysiology and treatment (Table 12-4).

Shock may be caused by a loss of circulating blood volume (hypovolemic shock), inability of the heart to pump the blood through the circulation (cardiogenic shock), and its subcategory, interference with blood flow through the heart (obstructive shock), or changes

TABLE 12-4 Types of Shock

Type	Mechanism	Specific Causes
Hypovolemic	Loss of blood or plasma	Hemorrhage, burns, dehydration, peritonitis, pancreatitis
Cardiogenic	Decreased pumping capability of the heart	Myocardial infarction of left ventricle, cardiac arrhythmia, pulmonary embolus, cardiac tamponade
Vasogenic (neurogenic or distributive)	Vasodilation owing to loss of sympathetic and vasomotor tone	Pain and fear, spinal cord injury, hypoglycemia (insulin shock)
Anaphylactic	Systemic vasodilation and increased permeability owing to severe allergic reaction	Insect stings, drugs, nuts, shellfish
Septic (endotoxic)	Vasodilation owing to severe infection, often with gram-negative bacteria	Virulent microorganisms (gram-negative bacteria) or multiple infections

in peripheral resistance leading to pooling of blood in the periphery (distributive, vasogenic, neurogenic, septic, or anaphylactic shock).

Pathophysiology

Blood pressure is determined by blood volume, heart contraction, and peripheral resistance. When one of these factors fails, blood pressure drops (Fig. 12-34). When blood volume is decreased, it is difficult to maintain pressure within the distribution system. If the force of the pump declines, blood flow slows, and venous return is reduced. The third factor, peripheral resistance, is altered by general vasodilation, which increases the capacity of the vascular system, leading to a lower pressure within the system and sluggish flow.

In patients with shock there is usually less cardiac output, and blood flow through the **microcirculation** is decreased, leading to reduced oxygen and nutrients for the cells. Less oxygen results in *anaerobic* metabolism and increased lactic acid production.

Compensation mechanisms are initiated as soon as blood pressure decreases:

- The SNS and adrenal medulla are stimulated to increase the heart rate, the force of contractions, and systemic vasoconstriction.
- Renin is secreted to activate angiotensin, a vasoconstrictor, and aldosterone to increase blood volume.
- Increased secretion of ADH also promotes reabsorption of water from the kidneys to increase blood volume and acts as a vasoconstrictor.
- Glucocorticoids are secreted that help stabilize the vascular system.
- Acidosis stimulates respirations, increasing oxygen supplies and reducing carbon dioxide levels.

Note: Organs which are the source of the problem cannot compensate for the problem. Thus cardiogenic shock cannot be compensated for by increased cardiac output.

If shock is prolonged, cell metabolism is diminished, and cell wastes are not removed, leading to lower pH, or *acidosis,* which impairs cell enzyme function. Acidosis also tends to cause vasodilation and relaxes precapillary sphincters first, contributing further to the pooling of blood in the periphery and decreasing venous return to the heart (Fig. 12-35).

If shock is not reversed quickly, it becomes even more difficult to reverse because the compensations and effects of shock tend to aggravate the problem. Vasoconstriction reduces arterial blood flow into tissues and organs, causing ischemia and eventually necrosis. Thrombi form in the microcirculation, further reducing venous return and cardiac output. Fluid shifts from the blood to the interstitial fluid as more cytokines are released from damaged cells. Organs and tissues can no longer function or undergo mitosis. Eventually the cells degenerate and die. When organ damage occurs, shock may be irreversible. Of concern is the occurrence of multiple organ failure after the patient appears stabilized.

Decompensation causes complications of shock, such as:

- Acute renal failure owing to tubular necrosis
- Shock lung, or acute respiratory distress syndrome (ARDS), due to pooling of blood and alveolar damage
- Hepatic failure due to cell necrosis
- Paralytic ileus, and stress or hemorrhagic ulcers
- Infection or septicemia from digestive tract ischemia or from the primary problem; septic shock, primarily endotoxic shock, has a much higher mortality rate because the toxins cause depressed myocardial function and acute respiratory distress syndrome (ARDS) and activate the coagulation process
- Disseminated intravascular coagulation (DIC) as the clotting process is initiated
- Depression of cardiac function by the oxygen deficit, acidosis and hyperkalemia, and myocardial depressant factor released from the ischemic pancreas; eventually cardiac arrhythmias and ischemia develop, perhaps resulting in cardiac arrest
- With multiorgan failure, shock becomes irreversible and death ensues

Etiology

Shock has a multitude of causes. A few are mentioned here.

1. *Hypovolemic shock* results from loss of blood or loss of plasma from the circulating blood. In patients with

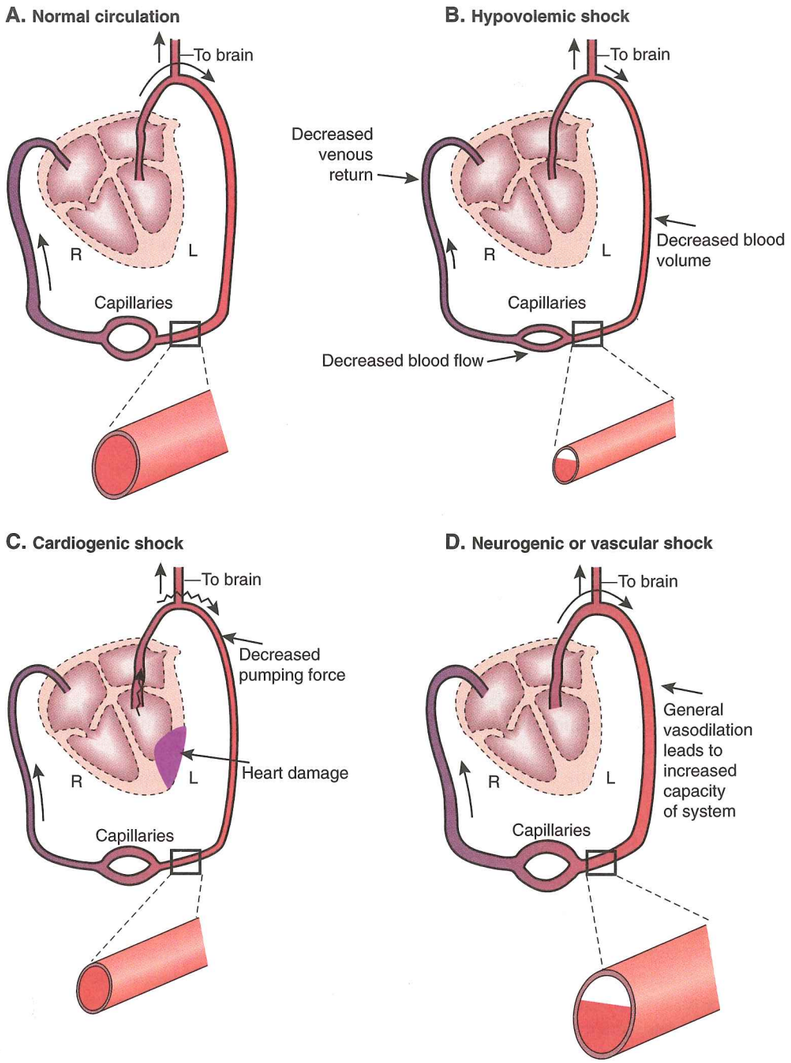


FIGURE 12-34 Causes of shock.

burns (see Chapter 5) the inflammatory response leads to edema with shift of fluid and protein from the blood into surrounding tissues and continued loss from the burn wound area due to loss of skin. Peritonitis (see Chapter 17) causes hypovolemia when infection and inflammation in the peritoneal membranes cause a fluid shift out of the blood into another compartment, the peritoneal space, a condition termed "third-spacing." Dehydration can reduce the circulating blood volume and blood pressure.

2. *Cardiogenic shock* is associated with cardiac impairment, such as acute infarction of the left ventricle, or arrhythmias. A subcategory, *obstructive shock,* is caused by cardiac tamponade or a pulmonary embolus that blocks blood flow through the heart.
3. The causes of *vasogenic shock* (it may be called *distributive shock,* since the blood has been relocated within the system because of vasodilation) may be classified in a variety of ways:
   - *Neurogenic* or *vasogenic shock* may develop from pain, fear, drugs, or loss of SNS stimuli with spinal cord injury. Metabolic dysfunction, such as hypoglycemia or insulin shock (see Chapter 16) or severe acidosis, may lead to this type of shock.

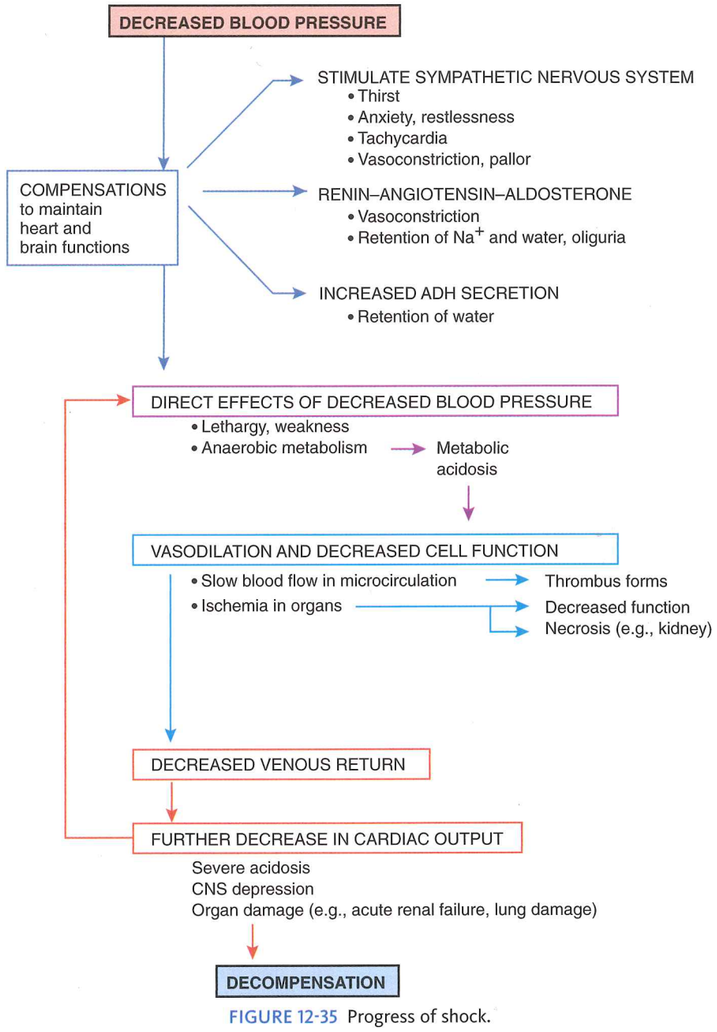


FIGURE 12-35 Progress of shock.

- *Anaphylactic shock* results from rapid general vasodilation due to the release of large amounts of histamine in a severe allergic reaction (see Chapter 7).

4. *Septic shock* may develop in persons with severe infection, particularly infections with gram-negative endotoxins, such as *Escherichia coli, Klebsiella pneumoniae,* and *Pseudomonas.* Initial circulatory changes vary with the causative organism, but eventually systemic vasodilation develops. In some cases, the organism affects the heart as well.

Signs and Symptoms

Often missed, the first signs of shock are thirst and agitation or restlessness because the SNS is quickly stimulated by hypotension. This is followed by the characteristic signs of compensation: cool, moist, pale skin; tachycardia; and oliguria (Fig. 12-36). Vasoconstriction shunts blood from the viscera and skin to the vital areas.

In cases of septic shock, the patient may experience "warm shock" with fever; warm, dry, flushed skin; rapid, strong pulse; and hyperventilation, evidence of infection.

Then the direct effects of a decrease in blood pressure and blood flow become manifest by lethargy, weakness, dizziness, and a weak, thready pulse. Initially hypoxemia and respiratory alkalosis are present as respirations increase. Acidosis or low serum pH due to anaerobic metabolism is compensated for by increased respirations (see Chapter 2). As shock progresses, metabolic acidosis

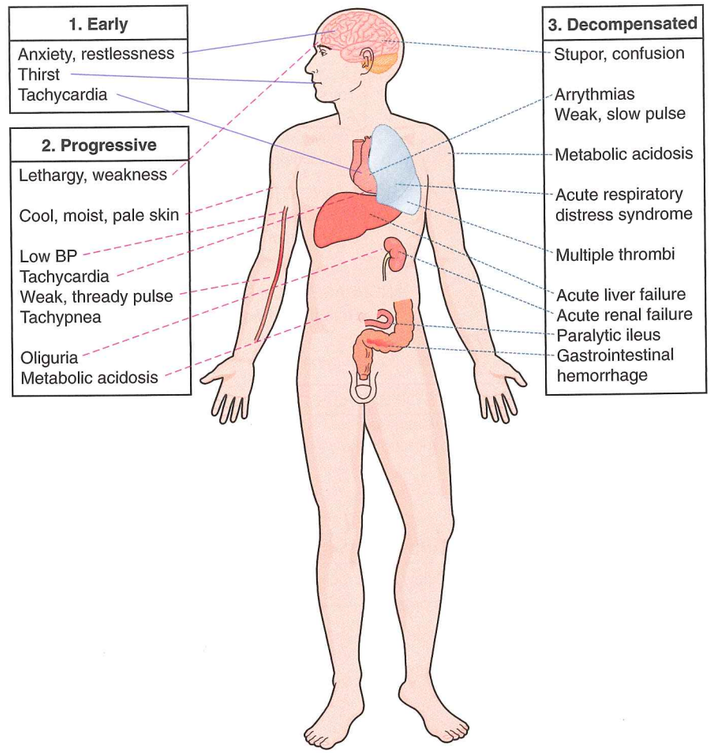


FIGURE 12-36 General effects of shock.

dominates. Manifestations of shock with rationale are summarized in Table 12-5.

If shock is prolonged, the body's responsiveness decreases as oxygen supplies dwindle and wastes accumulate in the body. Compensated metabolic acidosis progresses to decompensated acidosis when serum pH drops below 7.35 (see Chapter 2). Decompensated acidosis leads to central nervous system depression, reduced of cell metabolism and diminished effectiveness of medications. Acute renal failure, indicated by increasing serum urea and creatinine due to tubular ischemia and necrosis, is a common occurrence in decompensated shock.

When shock is severe and prolonged, monitoring may include the use of arterial catheters to assess blood pressure, ventricular filling, and cardiac output. Constant monitoring of arterial blood gases is essential to maintain acid-base balance.

Treatment

The Emergency Treatment box that follows lists the treatment for shock.

EMERGENCY TREATMENT FOR SHOCK

1. Place patient in supine position.
2. Cover and keep warm.
3. Call for assistance.
4. Administer oxygen if possible.
5. Determine underlying cause and treat if possible, such as using an EpiPen for anaphylaxis or applying pressure for bleeding.

The primary problem must be treated as quickly as possible to prevent decompensation. In patients with hypovolemic shock, whole blood, plasma or fluid with electrolytes and bicarbonate is required. When the cause is anaphylaxis, antihistamines and corticosteroids are given as well. Antimicrobials and glucocorticoids are necessary with septic shock. The oxygen supply should be maximized. The use of vasoconstrictors and vasodilators depends on the specific situation. Epinephrine acts both to reinforce heart action and constrict blood vessels. Dopamine and dobutamine increase heart

TABLE 12-5 Manifestations of Shock

	Manifestations	Rationale
Early signs	Anxiety and restlessness	Hypotension stimulates SNS
Compensation	Tachycardia	SNS response stimulates heart
	Cool, pale, moist skin	Peripheral vasoconstriction
	Oliguria	Renal vasoconstriction and renin mechanism
	Thirst	Osmoreceptors stimulated
	Rapid respirations	Anaerobic metabolism increases lactic acid secretion, which leads to increased respiratory rate
Progressive	Lethargy, weakness, faintness	Decreased blood flow and cardiac output
	Metabolic acidosis secretion.	Anaerobic metabolism increases lactic acid
		Decreased renal excretion of acids and production of bicarbonate owing to decreased glomerular filtration rate

SNS, Sympathetic nervous system.

function and, in low doses, dilate renal blood vessels, which may prevent acute renal failure.

The prognosis is good in the early stages. However, the mortality rate increases as decompensated shock develops in conjunction with renal failure, ARDS, or DIC.

THINK ABOUT 12-21

a. List and explain the signs indicating that compensation is occurring in patients with shock.
b. Explain two reasons why acidosis develops in shock.
c. State the expected changes in the arterial blood gas measurements with rationale for compensated acidosis with shock (see Chapter 2).
d. Explain several reasons why shock tends to become progressively more serious.
e. Explain why septic shock could be referred to as "warm shock."

CASE STUDY A

Myocardial Infarction

Ms. X., aged 55 years, has been complaining of severe fatigue and "indigestion." Her son is quite concerned and decides to take her to the emergency department. On arrival she appears very anxious, and her facial skin is cool and clammy; her blood pressure is 90/60, and the pulse is around 90, weak, and irregular. She is given oxygen, an intravenous line is opened, and leads for ECG are attached. Blood is taken for determination of serum enzymes and electrolytes. Tentative diagnosis is myocardial infarction involving the left ventricle. Her son provides information that indicates Ms. X is a long-time smoker, has a stressful job as a high school teacher, is recently separated after 20 years of marriage, and is fearful of losing the family home. She has also seemed to be more fatigued and stopped going to the gym about 18 months ago. She has begun to rely on "fast foods" like pizza and fried chicken and cooks infrequently. Her father had died of a heart attack at age 50. She had also noticed more fatigue and intermittent leg pain when walking or climbing stairs at work. Generalized atherosclerosis is suspected.

1. List the high-risk factors for atherosclerosis in this patient's history.
2. Describe how atherosclerosis causes myocardial infarction.
3. It is suspected that the indigestion reported in the history was really angina. Explain how this pain may have occurred.
4. Explain each of the admitting signs.
5. What is "atypical" in Ms. X's symptoms? How does this affect treatment and prognosis?
6. What information do serum enzyme and electrolyte levels provide?
7. What purpose does the ECG serve?

   It is determined that Ms. X. has a large infarct in the anterior left ventricle.
8. Ms. X. is showing increasing PVCs on the ECG. State the cause and describe the effect if these continue to increase in frequency.
9. On day 6 after admission Ms. X is preparing to go home with her son and they receive instructions on lifestyle modifications that are desirable if Ms. X is to avoid another MI. What measures should be included in such a discussion?

   Ms. X's condition becomes less stable and she remains in the hospital. On the seventh day following admission, she is found unconscious on the floor of her bathroom. Her pulse is weak and elevated, and her skin is moist with pallor evident. Her BP is 50 systolic. A diagnosis of cardiogenic shock is made and resuscitation efforts are started.
10. Explain why Ms. X. has experienced cardiogenic shock at this time.
11. Describe the effects of cardiogenic shock on the organs of the body.
12. What problems will occur if decompensated shock occurs? How is compensation limited in this situation?
13. Ms. X dies shortly later. What is the cause of death in this case?

CASE STUDY B

Essential Hypertension

Ms. J., aged 48 years, has essential hypertension, diagnosed 4 years ago. She has not been taking her medication during the past 6 months because she has been feeling fine. Now she has a new job and has been too busy to enjoy her usual swimming and golf. She has decided to have a checkup because she is feeling tired and dyspneic and has had several bouts of dizziness, blurred vision, and epistaxis (nosebleeds) lately. On examination, her blood pressure is found to be 190/120, some rales are present in the lungs, and the retinas of her eyes show some sclerosis and several arteriolar ruptures. The physician orders rest and medication to lower the blood pressure, as well as an appointment with a nutritionist and urinary tests to check kidney function.

1. Describe the pathophysiology of essential hypertension.
2. Explain the possible problems associated with the high diastolic pressure.
3. Explain the significance of the retinal changes.
4. The doctor suspects mild congestive heart failure. Explain how this can develop from hypertension.
5. Give two other possible signs of CHF.
6. List two medications that are helpful in treating hypertension and describe their actions.

CHAPTER SUMMARY

Heart function may be impaired by conduction system abnormalities, interference with the blood supply to the myocardium, or structural abnormalities. Arterial and venous disorders usually affect cardiac function as well. Multiple long-term factors usually predispose to heart dysfunction. Treatment of cardiovascular disorders frequently involves dietary changes, exercise programs, and cessation of cigarette smoking, as well as drug therapy and possibly surgery.

- Arteriosclerosis refers to degeneration of small arteries with loss of elasticity; development of thick, hard walls and narrow lumens causing ischemia; and possibly local necrosis.
- In atherosclerosis, large arteries such as the aorta and the coronary and carotid arteries are obstructed by cholesterol plaques and thrombi. Obstructions may be partial or complete, and emboli are common. Factors such as genetic conditions, high cholesterol diet, elevated serum LDL levels, and elevated blood pressure predispose patients to development of atheromas.
- Angina pectoris attacks are precipitated when the demand for oxygen by the myocardium exceeds the supply. Chest pain is relieved by intake of the vasodilator nitroglycerin and decreasing demands on the heart.
- MI results from total obstruction in a coronary artery, resulting in tissue necrosis and loss of function. Continuing chest pain, hypotension, and typical changes in the ECG are diagnostic. Arrhythmias are a common cause of death shortly after infarction occurs.
- Cardiac arrhythmias may result from MI or systemic abnormalities such as electrolyte imbalance, infection, or drug toxicity. Arrhythmias include abnormally slow or rapid heart rates, intermittent additional heart contractions (extrasystoles), or missed contractions (heart blocks).
- Depending on the cause, congestive heart failure may develop first in either the right or the left side of the heart, causing systemic backup and congestion or pulmonary congestion, respectively. In either case, cardiac output to the body is reduced, causing general fatigue and weakness, and stimulating the renin-angiotensin mechanism.
- Congenital heart defects consist of a variety of single or multiple developmental abnormalities in the heart. These structural abnormalities may involve the heart valves, such as mitral stenosis; the septae, such as ventricular septal defect; or the proximal great vessels. The primary outcome is decreased oxygen to all cells in the body.
- Cyanotic defects such as the tetralogy of Fallot refer to congenital defects where blood leaving the left ventricle consists of mixed oxygenated and unoxygenated blood, thereby delivering only small amounts of oxygen to all parts of the body.
- Rheumatic fever is a systemic inflammatory condition caused by an abnormal immune response to certain strains of hemolytic streptococcus. Inflammation causes scar tissue on heart valves and in the myocardium, leading to rheumatic heart disease.
- Infectious endocarditis causes destruction and permanent damage to heart valves and chordae tendineae. Individuals with heart defects or damage should take prophylactic antibacterial drugs before invasive procedures in which bacteremia is a threat.
- When pericarditis leads to a large volume of fluid accumulating in the pericardial cavity, filling of the heart is restricted, and cardiac output is reduced.
- Essential or primary hypertension is idiopathic and marked by a persistent elevation of blood pressure above 140/90, related to increased systemic vasoconstriction. It is frequently asymptomatic, but if not monitored and controlled may cause permanent damage to the kidneys, brain, and retinas as well as possible congestive heart failure.
- Atherosclerosis in the abdominal aorta or iliac arteries may cause ischemia in the feet and legs, resulting in fatigue, intermittent claudication, sensory impairment, ulcers, and possibly gangrene and amputation.
- Aortic aneurysms are frequently asymptomatic until they are very large or rupture occurs.

- Varicose veins in the legs tend to be progressive. They cause fatigue, swelling, and possible ulcers in the skin.
- Pulmonary emboli are a greater risk with phlebothrombosis, usually a silent problem, than with thrombophlebitis, in which inflammation is more apparent.
- Circulatory shock may result from decreased blood volume, impaired cardiac function with reduced output, or generalized vasodilation, any of which reduce blood flow and available oxygen in the microcirculation. Compensation mechanisms include the sympathetic nervous system; renin mechanism; increased secretion of ADH, aldosterone, and cortisol; and increased respirations. Decompensated shock develops with complications such as organ failure or infection.

STUDY QUESTIONS

1. Name three mechanisms that can increase cardiac output.
2. Explain the effect on blood flow of mitral valve incompetence.
3. Explain the importance/function for each of the following:
   a. High elastic fiber content in the aorta
   b. Smooth muscle in the arterioles
   c. Extensive capillaries in the liver and lungs
   d. Valves in the leg veins
4. Differentiate angina from myocardial infarction with regard to its cause and the characteristics of pain associated with it.
5. If you had a client with persistent chest pain following rest and administration of nitroglycerin, what action would you take?
6. Explain why vasodilator drugs are of limited value in arterial disease.
7. List and explain briefly three possible causes of cardiac dysrhythmias.
8. Differentiate heart blocks from PVCs with regard to causes and effects on heart action.
9. Describe the stages in the development of an atheroma in an artery
10. Why would you recommend avoidance of prolonged stress for a patient with congenital heart disease?
11. Explain how aortic stenosis may develop following rheumatic fever.
12. For which conditions could secondary polycythemia develop as compensation: VSD, CHF, chronic lung disease, aplastic anemia, multiple myeloma?
13. Explain why untreated essential hypertension is dangerous.
14. Define and explain the term *intermittent claudication.*
15. Describe three early signs of shock and the rationale for each.
16. Explain how neurogenic and hypovolemic shock may occur with major burns.
17. List four types of congenital heart defects and briefly describe each.

ADDITIONAL RESOURCES

Applegate EJ: *The Anatomy and Physiology Learning System Textbook*, ed 3, Philadelphia, 2006, Saunders.
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http://www.nhlbi.nih.gov *National Heart, Lung, and Blood Institute, National Institutes of Health*
http://www.webmd.com *Web MD*
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http://www.mayoclinic.com *Mayo Clinic*