CHAPTER
18

Heart Failure and Circulatory Shock

Adequate perfusion of body tissues depends on the pumping ability of the heart, a vascular system that transports blood to the cells and back to the heart, sufficient blood to fill the circulatory system, and tissues that are able to extract and use the oxygen and nutrients from the blood. Heart failure and circulatory shock are separate conditions that reflect failure of the circulatory system. Both conditions exhibit common compensatory mechanisms even though they differ in terms of pathogenesis and causes.

HEART FAILURE

Heart failure affects an estimated 4.8 million Americans.[1] Although morbidity and mortality rates from other cardiovascular diseases have decreased during the past several decades, the incidence of heart failure is increasing at an alarming rate. This change undoubtedly reflects treatment improvements and survival from other forms of heart disease. Despite advances in treatment, the 5-year survival rate for heart failure is only about 50%.

Physiology of Heart Failure

The term *heart failure* denotes the failure of the heart as a pump. The heart has the amazing capacity to adjust its pumping ability to meet the varying needs of the body. During sleep, its output declines, and during exercise, it increases markedly. The ability to increase cardiac output during increased activity is called the *cardiac reserve*. For example, competitive swimmers and long-distance runners have large cardiac reserves. During exercise, the cardiac output of these athletes rapidly increases to as much as five to six times their resting level. In sharp contrast with healthy athletes, persons with heart failure often use their cardiac reserve at rest. For them, just climbing a flight of stairs may cause shortness of breath because they have exceeded their cardiac reserve.

The pathophysiology of heart failure involves an interaction between two factors: (1) a decrease in cardiac output with a consequent decrease in blood flow to the kidneys and other body organs and tissues; (2) the recruitment of compensatory mechanisms designed to maintain tissue perfusion.

KEY CONCEPTS

HEART FAILURE

- The function of the heart is to move deoxygenated blood from the venous system through the right heart into the pulmonary circulation and to move the oxygenated blood from the pulmonary circulation through the left heart into the arterial system.
- To function effectively, the right and left hearts must maintain an equal output.
- Right heart failure represents failure of the right heart to pump blood forward into the pulmonary circulation; blood backs up in the systemic circulation, causing peripheral edema and congestion of the abdominal organs.
- Left heart failure represents failure of the left heart to move blood from the pulmonary circulation into the systemic circulation; blood backs up in the pulmonary circulation.

Cardiac Output

The cardiac output is the amount of blood that the heart pumps each minute. It reflects how often the heart beats each minute (heart rate) and how much blood the heart pumps with each beat (stroke volume) and can be expressed as the product of the heart rate and stroke volume (cardiac output = heart rate × stroke volume). Heart rate is a function of sympathetic nervous system reflexes, which accelerate heart rate and parasympathetic nervous system reflexes, which slows it down. Stroke volume is a function of preload, afterload, and cardiac contractility.

Preload and Afterload. The work that the heart performs consists mainly of ejecting blood into the pulmonary or systemic circulations. It is determined largely by the loading conditions or what is called the *preload* and *afterload*.

Preload reflects the loading condition of the heart at the end of diastole just before the onset of systole. It is the volume of blood stretching the resting heart muscle and is determined mainly by the venous return to the heart. *Afterload* represents the force that the contracting heart must generate to eject blood from the filled heart. The main components of afterload are ventricular wall tension and the peripheral vascular resistance. The greater the peripheral vascular resistance, the greater the ventricular wall tension and intraventricular pressure required to open the aortic valve and pump blood into the peripheral circulation.

Cardiac Contractility. Cardiac contractility refers to the mechanical performance of the heart: the ability of the contractile elements (actin and myosin filaments) of the heart muscle to interact and shorten against a load. Contractility increases cardiac output independent of preload filling and muscle stretch.

An *inotropic influence* is one that increases cardiac contractility. Sympathetic stimulation increases the strength of cardiac contraction (*i.e.*, positive inotropic action), and hypoxia and ischemia decrease contractility (*i.e.*, negative inotropic effect). The inotropic drug digitalis, which is used in the treatment of heart failure, increases cardiac contractility such that the heart is able to eject more blood at any level of preload filling.

Compensatory Mechanisms

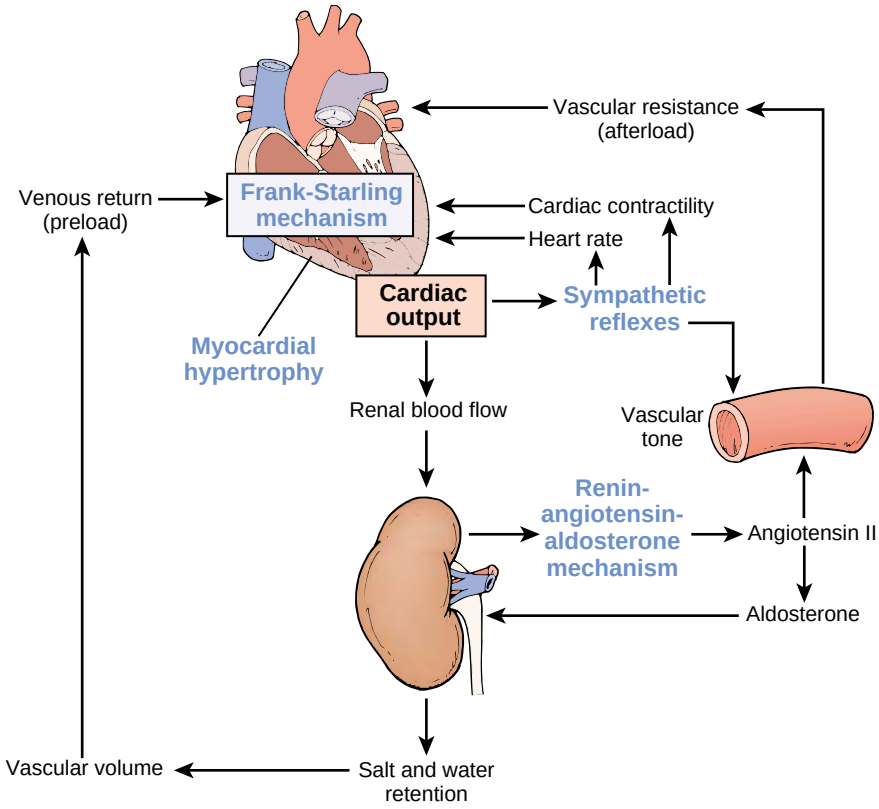
Heart failure is characterized by a decrease in cardiac output with a consequent decline in blood flow to the kidneys as well as other body organs and tissues. With a decrease in cardiac performance, tissue and organ perfusion is largely maintained through compensatory mechanisms such as the Frank-Starling mechanism, activation of the sympathetic nervous system and the renin-angiotensin-aldosterone mechanism, and myocardial hypertrophy (Fig. 18-1). In the failing heart, early decreases in cardiac function often go unnoticed because these compensatory mechanisms are used to maintain the cardiac output. This state is called *compensated heart failure*. Unfortunately, these mechanisms were not intended for long-term use. In severe and prolonged heart failure, the compensatory mechanisms are no longer effective and may themselves worsen the failure, causing what is termed *decompensated failure*.

Frank-Starling Mechanism. The Frank-Starling mechanism relies on an increase in venous return and a resultant increase in diastolic filling of the ventricles. Known as the *end-diastolic volume*, this volume causes the tension in the wall of the ventricles and the pressure in the ventricles to rise. With increased ventricular end-diastolic volume, there is increased stretching of the myocardial fibers, more optimal approximation of the actin and myosin filaments, and a resultant increase in the stroke volume in accord with the Frank-Starling mechanism (see Chapter 14, Fig. 14-20).

In heart failure a decrease in cardiac output and renal blood flow leads to increased salt and water retention, a resultant increase in vascular volume and venous return to the heart, and an increase in ventricular end-diastolic volume. Within limits, as preload and ventricular end-diastolic volume increase, there is a resultant increase in cardiac output. Thus, cardiac output may be normal at rest in persons with heart failure. However, as myocardial function deteriorates, the heart becomes overfilled, the muscle fibers become overstretched, and the ventricular function curve flattens (Fig. 18-2). The maximal increase in cardiac output that can be achieved may severely limit activity, while producing an elevation in left ventricular and pulmonary capillary pressure and development of dyspnea and pulmonary congestion.

An important determinant of myocardial energy consumption is ventricular wall tension. Overfilling of the ventricle produces a decrease in wall thickness and an increase in wall tension. Because increased wall tension increases myocardial oxygen requirements, it can produce ischemia and further impairment of cardiac function. The use of diuretics in the treatment of heart failure helps to reduce vascular volume and ventricular filling, thereby unloading the heart and reducing ventricular wall tension.

Increased Sympathetic Nervous System Activity. Stimulation of the sympathetic nervous system plays an important role in the compensatory response to decreased cardiac output and the pathogenesis of heart failure.[2,3] Both cardiac sympathetic tone



■ **FIGURE 18-1** ■ Compensatory mechanisms in heart failure. The Frank-Starling mechanism, sympathetic reflexes, renin-angiotensin-aldosterone mechanism, and myocardial hypertrophy function in maintaining the cardiac output for the failing heart.

and circulating catecholamine (epinephrine and norepinephrine) levels are elevated during the late stages of most forms of heart failure. By direct stimulation of heart rate and cardiac contractility and by regulation of vascular tone, the sympathetic nervous system helps to maintain perfusion of the various organs, particularly the heart and brain.

The negative aspects of increased sympathetic activity include an increase in peripheral vascular resistance and the afterload against which the heart must pump. Excessive sympathetic stimulation also may result in decreased blood flow to skin, muscle, kidney, and abdominal organs. The catecholamines also may contribute to the high rate of sudden death by promoting dysrhythmias.[4]

Renin-Angiotensin Mechanism. One of the most important effects of a lowered cardiac output in heart failure is a reduction in renal blood flow and glomerular filtration rate, which leads to salt and water retention. Normally, the kidneys receive approximately 25% of the cardiac output, but this may be decreased to as low as 8% to 10% in persons with heart failure. With decreased renal blood flow, there is a progressive increase in renin secretion by the kidneys along with parallel increases in circulating levels of angiotensin II. The increased concentration of angiotensin II contributes to a generalized vasoconstriction and serves as a stimulus for aldosterone production by the adrenal cortex (see Chapter 16). Aldosterone, in turn, increases tubular reabsorption of sodium, with an accompanying increase in water retention. Because aldosterone is metabolized in the liver, its levels are further increased when heart failure causes liver congestion.

Recent evidence suggests that angiotensin is also a growth factor for cardiac muscle cells and fibroblasts and, as such, may play a central role in modifying the structure and function of the myocardium in persons with heart failure.[2] Angiotensin-converting enzyme (ACE) inhibitor drugs, which block the conversion of angiotensin I to angiotensin II, are often used in the treatment of heart failure.[2–4]

Myocardial Hypertrophy. Myocardial hypertrophy is a long-term compensatory mechanism. Cardiac muscle, like skeletal muscle, responds to an increase in work demands by undergoing hypertrophy. Hypertrophy increases the number of contractile elements in myocardial cells as a means of increasing their contractile performance.

Myocardial hypertrophy occurs early in the course of heart failure and is an important risk factor for subsequent morbidity and mortality. Although hypertrophy increases the systolic function of the heart, it also eventually can lead to diastolic dysfunction and myocardial ischemia. Some forms of hypertrophy may lead to abnormal remodeling of the ventricular wall with a reduction in chamber size, reduced diastolic filling, and increased ventricular wall tension. For example, untreated hypertension causes hypertrophy that may preserve systolic function for a time, but eventually the work performed by the ventricle exceeds the augmented muscle mass and the heart dilates.[2] The increased muscle mass of the hypertrophied heart increases the need for oxygen delivery. When the oxygen requirements of the increased muscle mass exceed the ability of the coronary vessels to bring blood to the area, myocardial hypertrophy is no longer beneficial and may result in ischemia with decreased contractility. In addition, hypertrophy of cardiac muscle cells may be accompanied by the growth of nonmyocardial tissue (*e.g.*, fibrous tissue) that produces stiffness of the ventricle and further impairment of ventricular function.

Congestive Heart Failure

Heart failure occurs when the pumping ability of the heart becomes impaired. The term *congestive heart failure* (CHF) refers to heart failure that is accompanied by congestion of body tissues.

Heart failure may be caused by a variety of conditions, including acute myocardial infarction, hypertension, valvular heart disease, or degenerative conditions of the heart muscle known collectively as *cardiomyopathies* (see Chapter 17). Heart failure also may occur because of excessive work demands, such as occurs with hypermetabolic states, or with volume overload, such as occurs with renal failure. Either of these states may exceed the work capacity of even a healthy heart. In persons with asymptomatic heart disease, heart failure may be precipitated by an unrelated illness or stress. Table 18-1 lists major causes of heart failure.

Types of Heart Failure

Heart failure may be described as high-output or low-output failure, systolic or diastolic failure, and right-sided or left-sided failure.

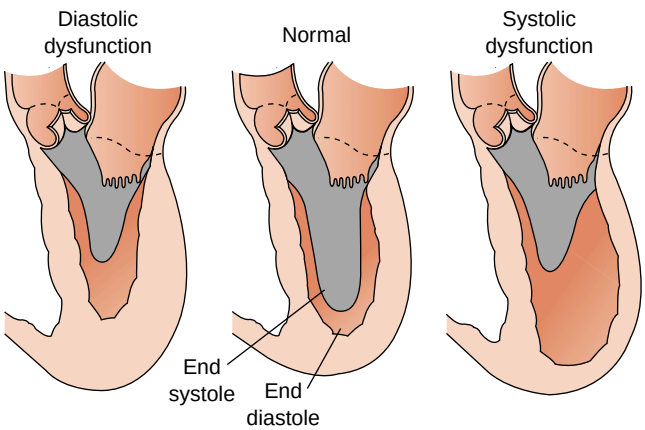
High- and Low-Output Failure. High- and low-output failure are described in terms of cardiac output. *High-output failure* is an uncommon type of heart failure that is caused by an excessive need for cardiac output. With high-output failure, the function of the heart may be supernormal but inadequate because of excessive metabolic needs. Causes of high-output failure include severe anemia, thyrotoxicosis, and conditions that cause arteriovenous shunting. High-output failure tends to be specifically treatable. *Low-output failure* is caused by disorders that impair the pumping ability of the heart, such as ischemic heart disease and cardiomyopathy.

TABLE 18-1 Causes of Heart Failure

Impaired Cardiac Function	Excess Work Demands
Myocardial Disease	**Increased Pressure Work**
Cardiomyopathies	Systemic hypertension
Myocarditis	Pulmonary hypertension
Coronary insufficiency	Coarctation of the aorta
Myocardial infarction	
Valvular Heart Disease	**Increased Volume Work**
Stenotic valvular disease	Arteriovenous shunt
Regurgitant valvular disease	Excessive administration of intravenous fluids
Congenital Heart Defects	**Increased Perfusion Work**
	Thyrotoxicosis
	Anemia
Constrictive Pericarditis	

Systolic Versus Diastolic Failure. A recent classification separates the pathophysiology of CHF into two categories—systolic dysfunction and diastolic dysfunction. Systolic dysfunction is characterized by impaired ejection of blood from the heart during systole and diastolic dysfunction by impaired filling of the ventricles during diastole (Fig. 18-3). Many persons with heart failure fall into an intermediate category, with combined elements of both systolic and diastolic failure.

Systolic dysfunction involves a decrease in cardiac contractility and ejection fraction. It commonly results from conditions that impair the contractile performance of the heart (*e.g.*, ischemic heart disease and cardiomyopathy), produce a volume



■ **FIGURE 18-3** ■ Congestive failure due to systolic and diastolic dysfunction. The ejection fraction represents the difference between the end-diastolic and end-systolic volumes. Normal systolic and diastolic function with normal ejection fraction (**middle**); diastolic dysfunction with decreased ejection fraction due to decreased diastolic filling (**left**); systolic dysfunction with decreased ejection fraction due to impaired systolic function (**right**).

overload (*e.g.*, valvular insufficiency and anemia), or generate a pressure overload (*e.g.*, hypertension and valvular stenosis) on the heart.

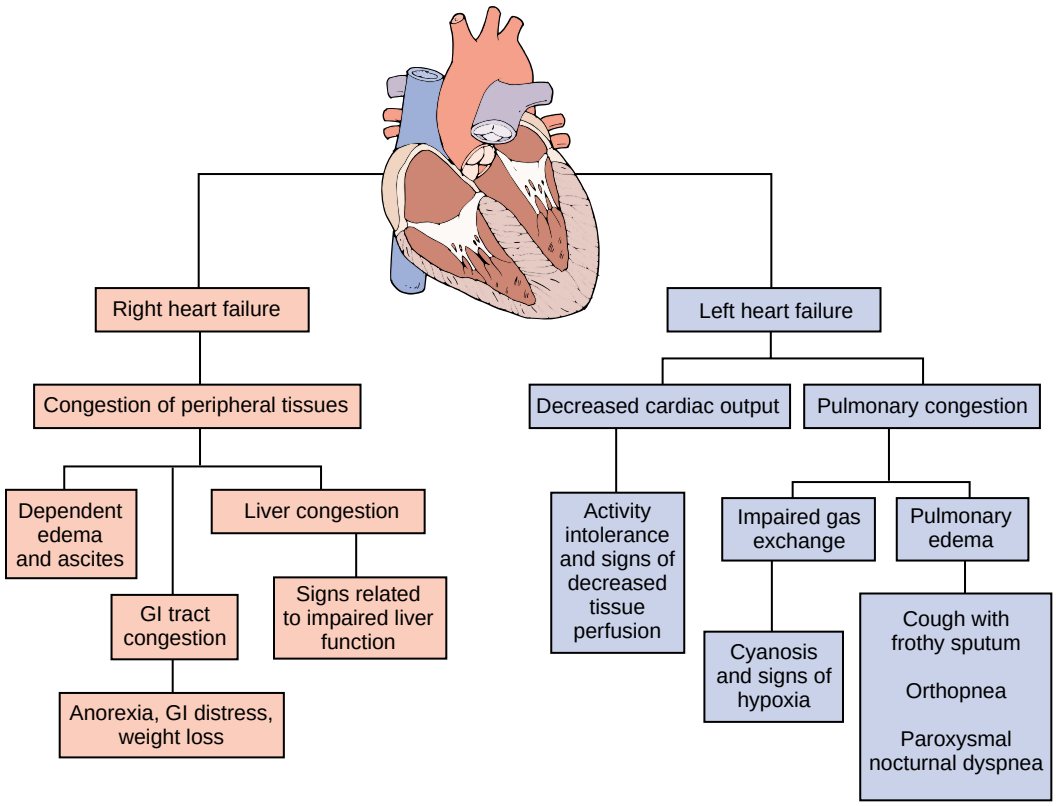
A normal heart ejects approximately 65% of the blood that is present in the ventricle at the end of diastole when it contracts. This is called the *ejection fraction*. In systolic heart failure, the ejection fraction declines progressively with increasing degrees of myocardial dysfunction. In very severe forms of heart failure, the ejection fraction may drop to a single-digit percentage. With a decrease in ejection fraction, there is a resultant increase in diastolic volume, ventricular dilation, ventricular wall tension, and ventricular end-diastolic pressure. The symptoms of persons with systolic dysfunction result mainly from reductions in ejection fraction and cardiac output.

Diastolic dysfunction, which reportedly accounts for approximately 40% of all cases of CHF, is characterized by a smaller ventricular chamber size, ventricular hypertrophy, and poor ventricular compliance (*i.e.*, ability to stretch during filling).[5] Because of impaired filling, congestive symptoms tend to predominate in diastolic dysfunction. Among the conditions that cause diastolic dysfunction are those that restrict diastolic filling (*e.g.*, mitral stenosis), those that increase ventricular wall thickness and reduce chamber size (*e.g.*, myocardial hypertrophy caused by lung disease and hypertrophic cardiomyopathy), and those that delay diastolic relaxation (*e.g.*, aging, ischemic heart disease). Aging often is accompanied by a delay in relaxation of the heart during diastole; diastolic filling begins while the ventricle is still stiff and resistant to stretching to accept an increase in volume.[6] A similar delay occurs with myocardial ischemia, resulting from a lack of energy to break the rigor bonds that form between the actin and myosin filaments of the contracting cardiac muscle.[7] Because tachycardia produces a decrease in diastolic filling time, persons with diastolic dysfunction often become symptomatic during activities and situations that increase heart rate.

Right-Sided Versus Left-Sided Heart Failure. Heart failure also can be classified according to the side of the heart (right or left) that is affected. An important feature of the circulatory system is that the right and left ventricles act as two pumps that are connected in series. To function effectively, the right and left ventricles must maintain an equal output. Although the initial event that leads to heart failure may be primarily right sided or left sided in origin, long-term heart failure usually involves both sides.

Right-Sided Heart Failure. Right-sided heart failure impairs the ability to move deoxygenated blood from the systemic venous circulation into the pulmonary circulation. Consequently, when the right heart fails, there is an accumulation of blood in the systemic venous system (Fig. 18-4). This causes an increase in the right atrial, right ventricular end-diastolic, and systemic venous pressures.

A major effect of right-sided heart failure is the development of peripheral edema. Because of the effects of gravity, the edema is most pronounced in the dependent parts of the body—in the lower extremities when the person is in the upright position and in the area over the sacrum when the person is supine. The



■ **FIGURE 18-4** ■ Manifestations of left- and right-sided heart failure.

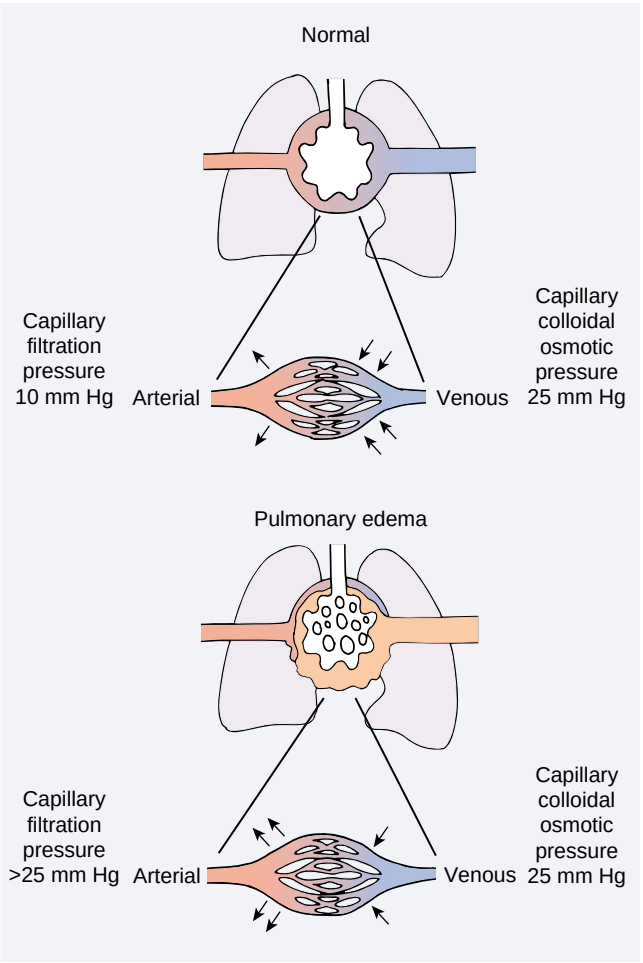
accumulation of edema fluid is evidenced by a gain in weight (*i.e.*, 1 pint of accumulated fluid results in a 1-lb weight gain). Daily measurement of weight can be used as a means of assessing fluid accumulation in a patient with chronic CHF. As a rule, a weight gain of more than 2 lb in 24 hours or 5 lb in 1 week is considered a sign of worsening failure.

Right-sided heart failure also produces congestion of the viscera. As venous distention progresses, blood backs up in the hepatic veins that drain into the inferior vena cava, and the liver becomes engorged. This may cause hepatomegaly and right upper quadrant pain. In severe and prolonged right-sided failure, liver function is impaired and hepatic cells may die. Congestion of the portal circulation also may lead to engorgement of the spleen and the development of ascites. Congestion of the gastrointestinal tract may interfere with digestion and absorption of nutrients, causing anorexia and abdominal discomfort. The jugular veins, which are above the level of the heart, are normally collapsed in the standing position or when sitting with the head at higher than a 30-degree angle. In severe right-sided failure, the external jugular veins become distended and can be visualized when the person is sitting up or standing.

The causes of right-sided heart failure include conditions that restrict blood flow into or through the lungs. Stenosis or regurgitation of the tricuspid or pulmonic valves, right ventricular infarction, cardiomyopathy, and persistent left-sided failure are common causes. *Cor pulmonale* refers to right heart failure resulting from pulmonary disease. *Acute cor pulmonale* results from right heart strain or overload secondary to acute pulmonary hypertension, often caused by massive pulmonary embolism. *Chronic cor pulmonale* occurs secondary to diseases that affect pulmonary tissues (chronic obstructive lung disease) or pulmonary vasculature (primary pulmonary hypertension).

Left-Sided Heart Failure. Left-sided heart failure impairs the pumping of blood from the low-pressure pulmonary circulation into the high-pressure arterial side of the systemic circulation (Fig. 18-4). With impairment of left heart function, there is a decrease in cardiac output, an increase in left atrial and left ventricular end-diastolic pressures, and congestion in the pulmonary circulation. When the pulmonary capillary filtration pressure (normally approximately 10 mm Hg) exceeds the capillary osmotic pressure (normally approximately 25 mm Hg), there is a shift of intravascular fluid into the interstitium of the lung and development of pulmonary edema (Fig 18-5). An episode of pulmonary edema often occurs at night, after a person has been reclining for some time and gravitational forces have been removed from the circulatory system. It is then that edema fluid that has been sequestered in the lower extremities during the day is returned to the vascular compartment and redistributed to the pulmonary circulation.

The most common causes of left-sided heart failure are acute myocardial infarction and cardiomyopathy. Left-sided heart failure and pulmonary congestion can develop very rapidly in persons with acute myocardial infarction. Even when the infarcted area is small, there may be a surrounding area of ischemic tissue. This may result in a large area of nonpumping ventricle and rapid onset of pulmonary edema. Aortic and aortic valve disorders can also cause left-sided heart failure. Pulmonary edema also may develop during rapid infusion of intravenous fluids or blood transfusions in an elderly person or in a person with limited cardiac reserve.



■ **FIGURE 18-5** ■ Mechanism of respiratory symptoms in left-sided heart failure. Normal exchange of fluid in the pulmonary capillaries (**top**). The capillary filtration pressure that moves fluid out of the capillary into the lung is less than the capillary colloidal osmotic pressure that pulls fluid back into the capillary. Development of pulmonary edema (**bottom**) occurs when the capillary filtration pressure exceeds the capillary colloidal osmotic pressure that pulls fluid back into the capillary.

Manifestations of Heart Failure

The manifestations of heart failure depend on the extent and type of cardiac dysfunction (*e.g.*, systolic versus diastolic) that is present and the rapidity with which it develops. Many persons with heart failure have concomitant cardiovascular and noncardiovascular disorders that may exacerbate the condition.[7] The concomitant conditions include cardiovascular disorders such as hypertension, coronary artery disease, diabetes, and cardiac dysrhythmias. Associated noncardiovascular diseases include renal insufficiency, thyroid disease, and pulmonary disease. A person with previously stable compensated heart failure may experience signs of heart failure for the first time when the condition has advanced to a critical point, such as with a progressive increase in pulmonary hypertension in a person with mitral valve regurgitation. Overt heart failure also may be precipitated by conditions such as infection, emotional stress, uncontrolled hypertension, administration of

fluid overload, or inappropriate reduction in therapy. Many persons with serious underlying heart disease, regardless of whether they have previously experienced heart failure, may be relatively asymptomatic as long they carefully adhere to their treatment regimen. A dietary excess of sodium is a frequent cause of sudden cardiac decompensation.

The manifestations of heart failure reflect the physiologic effects of the impaired pumping ability of the heart, decreased renal blood flow, and activation of the sympathetic compensatory mechanisms. The signs and symptoms include fluid retention and edema, shortness of breath and other respiratory manifestations, fatigue and limited exercise tolerance, cyanosis, cachexia and malnutrition, and cyanosis. Distention of the jugular veins may be present in right-sided failure. Excessive sympathetic stimulation may produce diaphoresis and tachycardia in persons with severe heart failure.

Fluid Retention and Edema. Many of the manifestations of CHF result from the increased capillary pressures that develop in the peripheral circulation in right-sided heart failure and in the pulmonary circulation in left-sided heart failure. The increased capillary pressure reflects an overfilling of the vascular system because of increased salt and water retention and venous congestion resulting from the impaired pumping ability of the heart.

Nocturia is a nightly increase in urine output that occurs relatively early in the course of CHF. It results from the return to the circulation of edema fluids from the dependent parts of the body when the person assumes the supine position for the night. As a result, the cardiac output, renal blood flow, glomerular filtration, and urine output increase. Oliguria is a late sign related to a severely reduced cardiac output and resultant renal failure.

Respiratory Manifestations. Shortness of breath caused by congestion of the pulmonary circulation is one of the major manifestations of left-sided heart failure. Perceived shortness of breath (*i.e.*, breathlessness) is called *dyspnea*. Dyspnea related to an increase in activity is called *exertional dyspnea*. *Orthopnea* is shortness of breath that occurs when a person is supine. The gravitational forces that cause fluid to become sequestered in the lower legs and feet when the person is standing or sitting are removed when a person with CHF assumes the supine position; fluid from the legs and dependent parts of the body is mobilized and redistributed to an already distended pulmonary circulation. *Paroxysmal nocturnal dyspnea* is a sudden attack of dyspnea that occurs during sleep. It disrupts sleep, and the person awakens with a feeling of extreme suffocation that resolves when he or she sits up. Initially, the experience may be interpreted as awakening from a bad dream.

A subtle and often overlooked symptom of heart failure is a chronic dry, nonproductive cough, which becomes worse when the person is lying down. Bronchospasm caused by congestion of the bronchial mucosa may cause wheezing and difficulty in breathing. This condition is sometimes referred to as *cardiac asthma*.

Cheyne-Stokes respiration, also known as *periodic breathing*, is characterized by a slow waxing and waning of respiration. The person breathes deeply for a period when the arterial carbon dioxide pressure (PCO_2) is high and then slightly or not at all when the PCO_2 falls. In persons with left-sided heart failure, the condition is thought to be caused by a prolongation of the heart-to-brain circulation, particularly in persons with hypertension and associated cerebral vascular disease. Cheyne-Stokes breathing may contribute to daytime sleepiness, and occasionally the person awakens at night with dyspnea precipitated by Cheyne-Stokes breathing.

Fatigue and Limited Exercise Tolerance. Fatigue and limb weakness often accompany diminished output from the left ventricle. Cardiac fatigue is different from general fatigue in that it usually is not present in the morning but appears and progresses as activity increases during the day. In acute or severe left-sided failure, cardiac output may fall to levels that are insufficient for providing the brain with adequate oxygen, and there are indications of mental confusion and disturbed behavior. Confusion, impairment of memory, anxiety, restlessness, and insomnia are common in elderly persons with advanced heart failure, particularly in those with cerebral atherosclerosis. These very symptoms may confuse the diagnosis of heart failure in the elderly because of the myriad other causes associated with aging.

Cachexia and Malnutrition. Cardiac cachexia is a condition of malnutrition and tissue wasting that occurs in persons with end-stage heart failure. A number of factors probably contribute to its development, including the fatigue and depression that interfere with food intake, congestion of the liver and gastrointestinal structures that impairs digestion and absorption and produces feelings of fullness, and the circulating toxins and mediators released from poorly perfused tissues that impair appetite and contribute to tissue wasting.

Cyanosis. Cyanosis is the bluish discoloration of the skin and mucous membranes caused by excess desaturated hemoglobin in the blood; it often is a late sign of heart failure. Cyanosis may be central or peripheral. Central cyanosis is caused by conditions that impair oxygenation of the arterial blood, such as pulmonary edema, left heart failure, or right-to-left shunting. Central cyanosis is best monitored in the lips and mucous membranes because these areas are not subject to conditions such as cold that cause peripheral cyanosis. Peripheral cyanosis is caused by conditions such as low-output failure that cause delivery of poorly oxygenated blood to the peripheral tissues, or by conditions such as peripheral vasoconstriction that cause excessive removal of oxygen from the blood.

Diagnosis and Treatment

Diagnostic methods in heart failure are directed toward establishing the cause of the disorder and determining the extent of the dysfunction. Because heart failure represents the failure of the heart as a pump and can occur in the course of a number of heart diseases or other systemic disorders, the diagnosis of heart failure often is based on signs and symptoms related to the failing heart itself, such as shortness of breath and fatigue. The functional classification of the New York Heart Association is one guide to classifying the extent of dysfunction (Table 18-2).

The diagnostic methods used for determining the presence of heart failure, its cause, and extent of dysfunction include history and physical examination, laboratory studies, chest radiography, electrocardiography (ECG), and echocardiography.[8] The history should include information related to dyspnea, cough, nocturia, generalized fatigue, and other signs and symptoms of heart failure. A complete physical examination in-

TABLE 18-2 New York Heart Association Functional Classification of Patients With Heart Disease

Classification	Characteristics
Class I	Patients with cardiac disease but without the resulting limitations in physical activity. Ordinary activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.
Class II	Patients with heart disease resulting in slight limitations of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. The symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort increases.

(From Criteria Committee of the New York Heart Association. [1964]. *Diseases of the heart and blood vessels: Nomenclature and criteria for diagnosis* [6th ed., pp. 112–113]. Boston: Little, Brown)

cludes assessment of heart rate, heart sounds, blood pressure, jugular veins for venous congestion, lungs for signs of pulmonary congestion, and lower extremities for edema. Laboratory tests are used in the diagnosis of anemia and electrolyte imbalances, and to detect signs of chronic liver congestion.

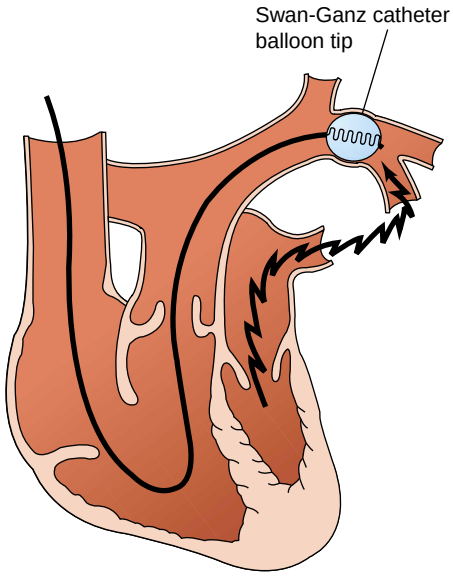
Chest radiographs provide information about the size and shape of the heart and pulmonary vasculature. The cardiac silhouette can be used to detect cardiac hypertrophy and dilatation. X-ray films can indicate the relative severity of the failure by revealing if pulmonary edema is predominantly vascular, interstitial, or advanced to the alveolar and bronchial stages. ECG findings may indicate atrial or ventricular hypertrophy, underlying disorders of cardiac rhythm, or conduction abnormalities such as right or left bundle branch block. *Echocardiography* plays a key role in assessing the anatomic and functional abnormalities in CHF, which include the size and function of cardiac valves, the motion of both ventricles, and the ventricular ejection fraction.[9] Radionuclide angiography and cardiac catheterization are other diagnostic tests used to detect the underlying causes of heart failure, such as heart defects and cardiomyopathy.

Invasive hemodynamic monitoring often is used in the management of acute, life-threatening episodes of heart failure. These monitoring methods include measurement of central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), thermodilution cardiac output, and intra-arterial blood pressure. Measurements of CVP are obtained from a catheter inserted into the right atrium; they provide information about the pumping ability of the right heart and its ability to move blood into the pulmonary circulation. PCWP is obtained from a balloon-tipped catheter that is advanced into a small pulmonary vessel (Fig. 18-6). When the balloon on pulmonary catheter is inflated, the catheter monitors pulmonary capillary pressures that are in direct communication with those of the left heart. Thus, PCWP provide information about the pumping ability of the left heart.

The goals of treatment for chronic heart failure are directed toward relieving the symptoms and improving the quality of life, with a long-term goal of slowing, halting, or reversing the cardiac dysfunction.[10,11] Treatment measures include correction of reversible causes such as anemia or thyrotoxicosis, surgical repair of a ventricular defect or an improperly functioning valve, pharmacologic and nonpharmacologic control of afterload stresses such as hypertension, modification of activities and lifestyle to a level consistent with the functional limitations of a reduced cardiac reserve, and the use of medications to improve cardiac function and limit excessive compensatory mechanisms. Restriction of salt intake and diuretic therapy facilitate the excretion of edema fluid.

In severe heart failure, restriction of activity, including bed rest if necessary, often facilitates temporary recompensation of cardiac function. However, there is no convincing evidence that continued bed rest is of benefit. Carefully designed and managed exercise programs for patients with CHF are well tolerated and beneficial to patients with stable New York Heart Association (NYHA) class I to III heart failure.[12]

Pharmacologic Treatment. Once heart failure is moderate to severe, polypharmacy becomes a management standard and often includes diuretics, digoxin, ACE inhibitors, and β-adrenergic–blocking agents.[13,14] The choice of pharmacologic agents is determined by problems caused by the disorder (*i.e.*, systolic or diastolic dysfunction) and those brought about by activation of compensatory mechanisms (*e.g.*, excess fluid retention, inappropriate activation of sympathetic mechanisms).



■ **FIGURE 18-6** ■ Swan-Ganz balloon-tipped catheter positioned in a pulmonary capillary. The pulmonary capillary wedge pressure, which reflects the left ventricular diastolic pressure, is measured with the balloon inflated.

Diuretics are among the most frequently prescribed medications for heart failure. They promote the excretion of edema fluid and help to sustain cardiac output and tissue perfusion by reducing preload and allowing the heart to operate at a more optimal part of the Frank-Starling curve. *Digitalis* drugs are inotropic agents that improve cardiac function by increasing the force and strength of ventricular contraction. They also produce a decrease in sinoatrial node activity and conduction through the atrioventricular node, thus slowing the heart rate and increasing diastolic filling time. Although not a diuretic, digitalis promotes urine output by improving cardiac output and renal blood flow. The *ACE inhibitors*, which prevent the conversion of angiotensin I to angiotensin II, have been effectively used in the treatment of heart failure. In heart failure, renin activity frequently is elevated because of decreased renal blood flow. The net result is an increase in angiotensin II, which causes vasoconstriction and increased aldosterone production with a subsequent increase in salt and water retention by the kidney. The ACE inhibitors reduce both mechanisms to decrease the workload of the heart.

β-adrenergic–blocking agents are used to decrease left ventricular dysfunction associated with activation of the sympathetic nervous system.[13,15] Chronic elevation of norepinephrine levels has been shown to cause cardiac muscle cell death and progressive left ventricular dysfunction, and is associated with poor prognosis in heart failure. The β-adrenergic–blocking agents also decrease the risk of serious cardiac dysrhythmias in the person with heart failure.

Acute Pulmonary Edema

Acute pulmonary edema is the most dramatic symptom of left heart failure. It is a life-threatening condition in which capillary fluid moves into the alveoli. The accumulated fluid in the alveoli and respiratory airways causes lung stiffness, makes lung expansion more difficult, and impairs the gas exchange function of the lung. With the decreased ability of the lungs to oxygenate the blood, the hemoglobin leaves the pulmonary circulation without being fully oxygenated.

Manifestations

Acute pulmonary edema usually is a terrifying experience. The person usually is seen sitting and gasping for air, in obvious apprehension. The pulse is rapid, the skin is moist and cool, and the lips and nail beds are cyanotic. As the lung edema worsens and oxygen supply to the brain drops, confusion and stupor appear. Dyspnea and air hunger are accompanied by a cough productive of frothy and often blood-tinged sputum—the effect of air mixing with serum albumin and red blood cells that have moved into the alveoli. The movement of air through the alveolar fluid produces fine crepitant sounds called *crackles*, which can be heard through a stethoscope placed on the chest. As fluid moves into the larger airways, the breathing becomes louder. The crackles heard earlier become louder and coarser. In the terminal stage the breathing pattern is called the *death rattle*. Persons with severe pulmonary edema literally drown in their own secretions.

Treatment

Treatment of acute pulmonary edema is directed toward reducing the fluid volume in the pulmonary circulation. This can be accomplished by reducing the amount of blood that the right heart delivers to the lungs or by improving the work performance of the left heart. Several measures can decrease the blood volume in the pulmonary circulation; the seriousness of the pulmonary edema determines which are used. One of the simplest measures to relieve orthopnea is assumption of the seated position, in which gravity causes blood to be redistributed to the lower extremities. For many persons, sitting up or standing is almost instinctive and may be sufficient to relieve the symptoms associated with mild accumulation of fluid.

Measures to improve left heart performance focus on decreasing the preload by reducing the filling pressure of the left ventricle and on reducing the afterload against which the left heart must pump. This can be accomplished through the use of diuretics, vasodilator drugs, treatment of arrhythmias that impair cardiac function, and improvement of the contractile properties of the left ventricle with digitalis. Rapid digitalization can be accomplished with intravenous administration of the drug.

Oxygen therapy increases the oxygen content of the blood and helps relieve anxiety. Positive-pressure breathing, which is administered through a specially designed mask, increases the intra-alveolar pressure, opposes the capillary filtration pressure in the pulmonary capillaries, and sometimes is used as a temporary measure to decrease the amount of fluid moving into the alveoli. However, in the most severe cases, endotracheal intubation and mechanical ventilation may be necessary. Although its mechanisms of action are unclear, morphine sulfate usually is the drug of choice in acute pulmonary edema. Morphine relieves anxiety and depresses the pulmonary reflexes that cause spasm of the pulmonary vessels. It also increases venous pooling by vasodilatation.

Cardiogenic Shock

Cardiogenic shock refers to the pronounced failure of the heart as a pump. Cardiogenic shock can occur relatively quickly because of the damage to the heart that occurs during myocardial infarction; ineffective pumping caused by cardiac dysrhythmias; mechanical defects that may occur as a complication of myocardial infarction, such as ventricular septal defect; ventricular aneurysm; acute disruption of valvular function; or problems associated with open heart surgery. Cardiogenic shock also may ensue as an end-stage condition of coronary artery disease or cardiomyopathy.

The most common cause of cardiogenic shock is myocardial infarction. Most patients who die of cardiogenic shock have lost at least 40% of the contracting muscle of the left ventricle because of a recent infarct or a combination of recent and old infarcts.[16] Cardiogenic shock can follow other types of shock associated with inadequate coronary blood flow.

In all cases of cardiogenic shock, there is failure to eject blood from the heart, hypotension, and inadequate cardiac output. Increased systemic vascular resistance often contributes to the deterioration of cardiac function by increasing afterload or the resistance to ventricular systole. The filling pressure, or preload of the heart, also is increased as blood returning to the heart is added to blood that previously was returned but not pumped forward, resulting in an increase in end-systolic ventricular volume. Increased resistance to ventricular systole (*i.e.*, afterload) combined with the decreased myocardial contractility causes the increased end-systolic ventricular volume and increased preload, which further complicate cardiac status.

Manifestations

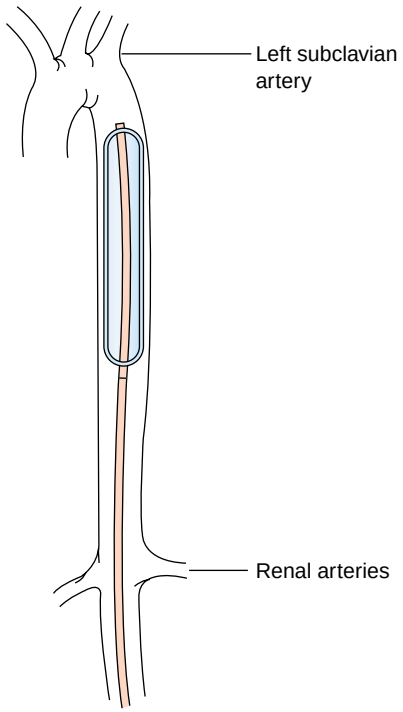
The signs and symptoms of cardiogenic shock are consistent with those of extreme heart failure. The lips, nail beds, and skin are cyanotic because of stagnation of blood flow and increased extraction of oxygen from the hemoglobin as it passes through the capillary bed. The CVP and PCWP rise as a result of volume overload caused by the pumping failure of the heart.

Treatment

Treatment of cardiogenic shock requires a precarious balance between improving cardiac output, reducing the workload and oxygen needs of the myocardium, and preserving coronary perfusion. Fluid volume must be regulated within a level that maintains the filling pressure (*i.e.*, venous return) of the heart and maximum use of the Frank-Starling mechanism without causing pulmonary congestion.

Pharmacologic treatment includes the use of vasodilator drugs such as nitroprusside and nitroglycerin that cause arterial and venous dilatation. These drugs produce venous and arterial dilation, thus decreasing the venous return to the heart and the arterial resistance against which the left heart must pump. Catecholamines increase cardiac contractility but must be used with caution because they also produce vasoconstriction and increase cardiac workload by increasing the afterload.

The *intra-aortic balloon pump* may be used for persons who do not experience response to medical treatment. It provides a means of increasing aortic diastolic pressure and enhancing coronary and peripheral blood flow without increasing systolic pressure and the afterload, against which the left ventricle must pump.[16] The device, which pumps in synchrony with the heart, consists of a balloon that is inserted through a catheter into the descending aorta (Fig. 18-7). The balloon is filled with helium and is timed to inflate during ventricular diastole and deflate just before ventricular systole. Diastolic inflation creates a pressure wave in the ascending aorta that increases coronary artery flow and a less intense wave in the lower aorta that enhances organ perfusion. The sudden balloon deflation at the onset of systole lowers the resistance to ejection of blood from the left ventricle, thereby increasing the heart's pumping efficiency and decreasing myocardial oxygen consumption.



■ **FIGURE 18-7** ■ Aortic balloon pump. (Hudak C.M., Gallo B.M. [1994]. *Critical care nursing* [6th ed.]. Philadelphia: J.B. Lippincott)

Mechanical Support of the Failing Heart and Heart Transplantation

Refractory heart failure reflects deterioration in cardiac function that is unresponsive to medical or surgical interventions. With improved methods of treatment, more people are reaching a point where a cure is unachievable and death is imminent without mechanical support or heart transplantation.

Since the early 1960s, significant progress has been made in improving the efficacy of *ventricular assist devices* (VADs), which are mechanical pumps used to support ventricular function. VADs are used to decrease the workload of the myocardium while maintaining cardiac output and systemic arterial pressure.[17] This decreases the workload on the ventricle and allows it to rest and recover.

Heart transplantation remains the treatment of choice for many persons with end-stage cardiac failure. The number of successful heart transplantations has been steadily climbing, with more than 2800 procedures performed per year. Patients with heart transplants who are treated with triple-immunosuppressant therapy have a 5-year survival rate of 70% to 80%.[18] Despite the overall success of heart transplantation, donor availability and complications associated with infection, rejection, and immunosuppression drug therapy remain problems.

In summary, heart failure occurs when the heart fails to pump sufficient blood to meet the metabolic needs of body tissues. The physiology of heart failure reflects an interplay between a decrease in cardiac output that accompanies impaired function of the failing heart and the compensatory mechanisms designed to preserve the cardiac reserve. Compensatory mechanisms that contribute to maintenance of the cardiac reserve include the Frank-Starling mechanism, sympathetic nervous system responses, the renin-angiotensin-aldosterone mechanism, and myocardial hypertrophy. In the failing heart, early decreases in cardiac function may go unnoticed because these compensatory mechanisms maintain the cardiac output. This is called *compensated heart failure*. Unfortunately, the mechanisms were not intended for long-term use, and in severe and prolonged heart failure, the compensatory mechanisms no longer are effective and further impair cardiac function.

Heart failure may be described as high-output or low-output failure, systolic or diastolic failure, and right-sided or left-sided failure. With high-output failure, the function of the heart may be supernormal but inadequate because of excessive metabolic needs, and low-output failure is caused by disorders that impair the pumping ability of the heart. With systolic dysfunction, there is impaired ejection of blood from the heart during systole; with diastolic dysfunction, there is impaired filling of the heart during diastole. Right-sided failure

is characterized by congestion in the peripheral circulation, and left-sided failure by congestion in the pulmonary circulation.

The manifestations of heart failure include edema, nocturia, fatigue and impaired exercise tolerance, cyanosis, signs of increased sympathetic nervous system activity, and impaired gastrointestinal function and malnutrition. In right-sided failure, there is dependent edema of the lower parts of the body, engorgement of the liver, and ascites. In left-sided failure, shortness of breath and chronic, nonproductive cough are common.

Acute pulmonary edema is a life-threatening condition in which the accumulation of fluid in the interstitium of the lung and alveoli interferes with lung expansion and gas exchange. It is characterized by extreme breathlessness, crackles, frothy sputum, cyanosis, and signs of hypoxemia. In cardiogenic shock, there is failure to eject blood from the heart, hypotension, inadequate cardiac output, and impaired perfusion of peripheral tissues. Mechanical support devices, including the intra-aortic balloon pump (for acute failure) and the VAD, sustain life in persons with severe heart failure. Heart transplantation remains the treatment of choice for many persons with end-stage heart failure.

CIRCULATORY FAILURE (SHOCK)

The functions of the circulatory system are to perfuse body tissues and supply them with oxygen. Whereas heart failure results from impaired ability of the heart as a pump, circulatory shock results from a failure of the circulatory system to supply the peripheral tissues and organs of the body with an adequate blood supply. As with heart failure, circulatory shock is not a specific disease but can occur in the course of many life-threatening traumatic or disease states. In situations of severe cardiovascular compromise, signs of heart failure and vascular compromise may coexist. Although circulatory shock produces hypotension, it should not be equated with a drop in blood pressure. Hypotension often is a late sign and indicates a failure of compensatory mechanisms.

Adequate perfusion of body tissues depends on the pumping ability of the heart, a vascular system that transports blood to the cells and back to the heart, sufficient blood to fill the vascular system, and tissues that are able to use and extract oxygen and nutrients from the blood. As with heart failure, circulatory shock produces compensatory physiologic responses that eventually decompensate into various shock states if the condition is not properly treated in a timely manner.

Types of Shock

Circulatory shock is used to describe a critical decrease in tissue perfusion caused by a loss or redistribution of intravascular fluid. It can be classified as hypovolemic, obstructive, or distributive. These three main types of shock are summarized in Chart 18-1 and depicted in Figure 18-8. Cardiogenic shock, which results from failure of the heart as a pump, was discussed earlier in the chapter.

KEY CONCEPTS

CIRCULATORY SHOCK

- Circulatory shock represents the inability of the circulation to adequately perfuse the tissues of the body.
- It can result from a loss of fluid from the vascular compartment, an increase in the size of the vascular compartment that interferes with the distribution of blood, or obstruction of flow through the vascular compartment.
- The manifestations of shock reflect both the impaired perfusion of body tissues and the body's attempt to maintain tissue perfusion through conservation of water by the kidney, translocation of fluid from the extracellular to the intravascular compartment, and activation of sympathetic nervous system mechanisms that increase heart rate and divert blood from less to more essential body tissues.

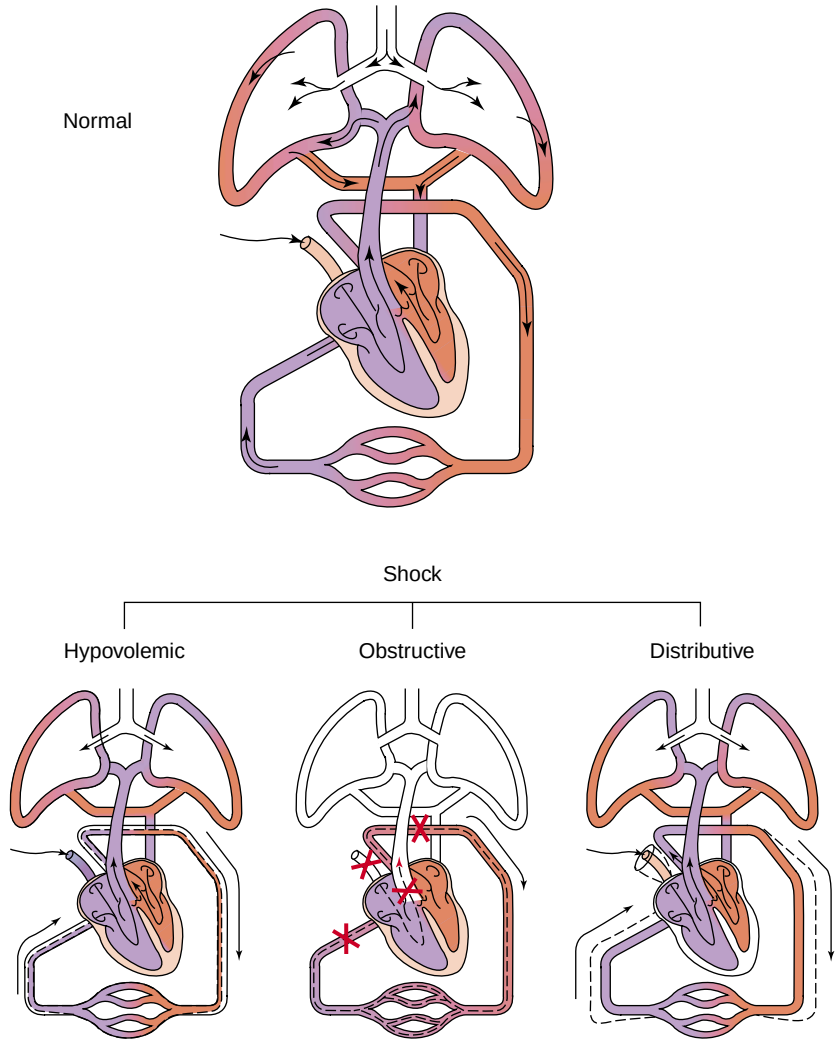
Hypovolemic Shock

Hypovolemic shock is characterized by diminished blood volume such that there is inadequate filling of the vascular compartment (see Fig. 18-9). It occurs when there is an acute loss of 15% to 20% of the circulating blood volume. The decrease may be caused by an external loss of whole blood (*e.g.*, hemorrhage), plasma (*e.g.*, severe burns), or extracellular fluid (*e.g.*, gastrointestinal fluids lost in vomiting or diarrhea). Hypovolemic shock also can result from an internal hemorrhage or from third-space losses, when extracellular fluid is shifted from the vascular compartment to the interstitial space or compartment.

Hypovolemic shock has been the most widely studied type of shock and usually serves as a prototype in discussions of the manifestations of shock. Approximately 10% of the total

CHART 18-1 Classification of Circulatory Shock

Hypovolemic
Loss of whole blood
Loss of plasma
Loss of extracellular fluid
Obstructive
Inability of the heart to fill properly (cardiac tamponade)
Obstruction to outflow from the heart (pulmonary embolus, cardiac myxoma, pneumothorax, or dissecting aneurysm)
Distributive
Loss of sympathetic vasomotor tone
Presence of vasodilating substances in the blood (anaphylactic shock)
Presence of inflammatory mediators (septic shock)



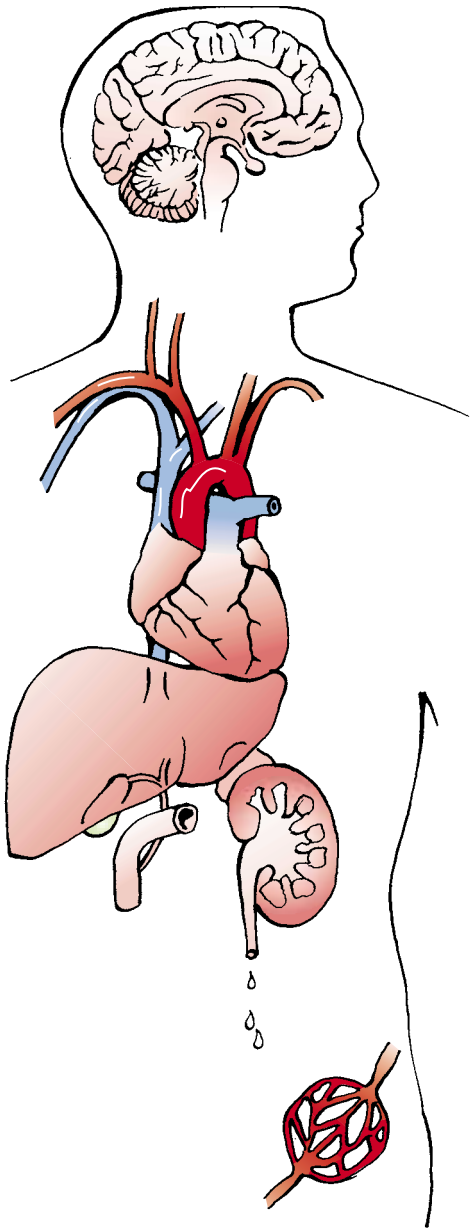
■ **FIGURE 18-8** ■ Types of shock.

blood volume can be removed without changing the cardiac output or arterial pressure.[19] The average blood donor loses a pint of blood without experiencing adverse effects. As increasing amounts of blood (10% to 25%) are removed, the cardiac output falls, but the arterial pressure is maintained because of sympathetic-mediated increases in heart rate and vasoconstriction. Blood pressure is the product of cardiac output and peripheral vascular resistance (BP = CO × PVR); an increase in peripheral vascular resistance maintains blood pressure in the presence of decreased cardiac output for a short period. Cardiac output and tissue perfusion decrease before signs of hypotension occur. The arterial pressure falls to zero when approximately 35% to 45% of the total blood volume has been removed.[19]

Manifestations. The signs and symptoms of hypovolemic shock depend on shock stage and are closely related to low peripheral blood flow and excessive sympathetic stimulation. They include thirst, an increase in heart rate, cool and clammy skin, a decrease in arterial blood pressure, a decrease in urine output, and changes in mentation (Fig. 18-9). Laboratory tests of hemoglobin and hematocrit provide information regarding the severity of blood loss or hemoconcentration caused by dehydration. Serum lactate and arterial pH provide information about the severity of acidosis.

An increase in heart rate is often an early sign of shock. As shock progresses, the pulse becomes weak and thready, indicating vasoconstriction and a reduction in filling of the vascular compartment, and the respirations become rapid and deep. Arterial blood pressure is decreased in moderate to severe shock. However, controversy exists regarding the value of blood pressure measurements in the early diagnosis and management of shock. This is because compensatory mechanisms tend to preserve blood pressure until shock is relatively far advanced. Furthermore, an adequate arterial pressure does not ensure adequate perfusion and oxygenation of vital organs at the cellular level. This does not imply that blood pressure should not be closely monitored in patients at risk for development of shock, but it does indicate the need for other assessment measures by which shock may be detected at an earlier stage.

Decreased intravascular volume results in decreased venous return to the heart and a decrease in CVP. When shock becomes severe, the peripheral veins collapse, making it difficult to insert peripheral venous lines. Sympathetic stimulation also leads



Brain
- Thirst
- Increased production and release ADH

Cardiovascular system
- Increased heart rate
- Increased force of cardiac contraction
- Increased systemic vascular resistance
- Decreased blood flow to the
  kidney
  gastrointestinal tract
  skin
  skeletal muscles
- Constriction of the veins

Adrenal gland
- Increased production and release of aldosterone by the adrenal cortex
- Increased production and release of the catecholamines (epinephrine and norepinephrine) by the adrenal medulla

Liver
- Constriction of veins and sinusoids with mobilization of blood stored in the liver

Kidney
- Increased retention of sodium and water
- Decreased urine output

Capillary bed
- Increased reabsorption of water into the capillary from the interstitial spaces due to constriction of the arterioles with a resultant decrease in capillary pressure

■ **FIGURE 18-9** ■ Compensatory mechanisms in hypovolemic shock.

to intense vasoconstriction of the skin vessels and activation of the sweat glands. As a result, the skin is cool and moist. When shock is caused by hemorrhage, the loss of red blood cells leaves the skin and mucous membranes looking pale.

Urine output decreases very quickly in hypovolemic and other forms of shock. Compensatory mechanisms decrease renal blood flow as a means of diverting blood flow to the heart and brain. Oliguria of 20 mL/hour or less indicates severe shock and inadequate renal perfusion. Continuous measurement of urine output is essential for assessing the circulatory status of the person in shock.

Restlessness and apprehension are common behaviors in early shock. As the shock progresses and blood flow to the brain decreases, restlessness is replaced by apathy and stupor. If shock is unchecked, the apathy progresses to coma. Coma caused by blood loss alone and not related to head injury or other factors is an unfavorable sign.

Treatment. The treatment of hypovolemic shock is directed toward correcting or controlling the underlying cause and improving tissue perfusion. Persons who have sustained blood loss are commonly placed in supine position with the legs elevated to maximize cerebral blood flow. Oxygen is administered to persons with signs of hypoxemia. Because subcutaneous administration is unpredictable, pain medications usually are administered intravenously. Frequent measurements of heart rate and cardiac rhythm, blood pressure, and urine flow are used to assess the severity of circulatory compromise and to monitor treatment.

In hypovolemic shock, the goal of treatment is to restore vascular volume. This can be accomplished through intravenous administration of fluids and blood. The crystalloids (*e.g.*, isotonic saline and glucose solutions) are readily available for emergencies and mass casualties. They often are effective, at least temporarily, when given in adequate doses. Blood or

blood products (packed or frozen red cells) are administered based on hematocrit and hemodynamic findings. Fluids and blood are best administered based on volume indicators such as CVP and PCWP.

Vasoactive drugs (*e.g.*, adrenergic agents) are agents capable of constricting or dilating blood vessels. As a general rule, the adrenergic drugs are not used as a primary form of therapy in hypovolemic shock. Simple blood pressure elevation produced by vasopressor drugs has little effect on the underlying cause of shock and in many cases may be detrimental. These agents are given only when hypotension persists after volume deficits have been corrected.

Dopamine, which induces more favorable actions than many of the other adrenergic drugs, may be used in the treatment of severe and prolonged shock. Dopamine, when given in low doses, is thought to increase blood flow to the kidneys, liver, and other abdominal organs while maintaining vasoconstriction in less vital structures, such as the skin and skeletal muscles. In severe shock, higher doses may be needed to maintain blood pressure. After dopamine administration exceeds this low-dose range, it has vasoconstrictive effects on blood flow to the kidneys and abdominal organs that are similar to those of epinephrine.

Obstructive Shock

The term *obstructive shock* is used to describe circulatory shock that results from mechanical obstruction of the flow of blood through the central circulation (great veins, heart, or lungs; see Fig. 18-8). Obstructive shock may be caused by a number of conditions, including dissecting aortic aneurysm, cardiac tamponade, pneumothorax, atrial myxoma, or evisceration of abdominal contents into the thoracic cavity because of a ruptured hemidiaphragm. The most common cause of obstructive shock is pulmonary embolism.

The primary physiologic results of obstructive shock are elevated right heart pressure and impaired venous return to the heart. The signs of right heart failure are seen, including elevation of central venous pressure and jugular vein distention. Treatment modalities focus on correcting the cause of the disorder, frequently with surgical interventions such as pulmonary embolectomy, pericardiocentesis (*i.e.*, removal of fluid from the pericardial sac) for cardiac tamponade, or the insertion of a chest tube for correction of a tension pneumothorax or hemothorax. In select cases of pulmonary embolus, thrombolytic drugs may be used to dissolve the clots causing the obstruction.

Distributive Shock

Distributive shock is characterized by loss of blood vessel tone, enlargement of the vascular compartment, and displacement of the vascular volume away from the heart and central circulation. With distributive shock, the capacity of the vascular compartment expands to the extent that a normal volume of blood does not fill the circulatory system (see Fig. 18-8). Loss of vessel tone has two main causes: a decrease in the sympathetic control of vasomotor tone and the presence of vasodilator substances in the blood. Venous return is decreased in distributive shock, which leads to a diminished cardiac output but not a decrease in total blood volume; this type of shock is also referred to as *normovolemic shock*. There are two shock states that share the basic circulatory pattern of distributive shock: neurogenic shock and anaphylactic shock. Septic shock shares many of the features of distributive shock.

Neurogenic Shock. Neurogenic shock describes shock caused by decreased sympathetic control of blood vessel tone caused by a defect in the vasomotor center in the brain stem or the sympathetic outflow to the blood vessels. Output from the vasomotor center can be interrupted by brain injury, the depressant action of drugs, general anesthesia, hypoxia, or lack of glucose (*e.g.*, insulin reaction). Fainting attributable to emotional causes is a transient form of neurogenic shock. Spinal anesthesia or spinal cord injury above the midthoracic region can interrupt the transmission of outflow from the vasomotor center. The term *spinal shock* is used to describe the neurogenic shock that occurs in persons with spinal cord injury. Many general anesthetic agents can cause a neurogenic shock-like reaction, especially during induction, because of interference with sympathetic nervous system function. In contrast to hypovolemic shock, the heart rate in neurogenic shock often is slower than normal, and the skin is dry and warm. This type of distributive shock is rare and usually transitory.

Anaphylactic Shock. Anaphylactic shock is characterized by massive vasodilatation, pooling of blood in the peripheral blood vessels, and increased capillary permeability.[20] This type of shock, which is a manifestation of systemic anaphylaxis, is caused by an immune-mediated reaction in which vasodilator substances such as histamine are released into the blood (see Chapter 10). These substances cause dilatation of arterioles and venules along with a marked increase in capillary permeability. The vascular response in anaphylactic shock is often accompanied by bronchospasm, contraction of gastrointestinal and uterine smooth muscle, and urticaria or angioedema (swelling of the face and throat).

Among the most common causes of anaphylactic shock are reactions to drugs, such as penicillin; foods, such as nuts and shellfish; and insect venoms. The most common cause is stings from insects of the order Hymenoptera (*i.e.*, bees, wasps, and fire ants). Latex allergy has caused life-threatening anaphylaxis in a growing segment of the population (see Chapter 10).[21] Health care workers and others who are exposed to latex are developing latex sensitivities that range from mild urticaria, contact dermatitis, and mild respiratory distress to anaphylactic shock.[20] Children with spina bifida also are at extreme risk for this increasingly serious allergy.

The onset of anaphylaxis depends on the sensitivity of the person and the rate and quantity of antigen exposure. Anaphylactic shock often develops suddenly; death can occur within a matter of minutes unless appropriate medical intervention is promptly instituted. Signs and symptoms of impending anaphylactic shock include abdominal cramps; apprehension; burning and warm sensation of the skin, itching, urticaria (*i.e.*, hives); coughing, choking, wheezing, chest tightness, and difficulty in breathing. After blood begins to pool peripherally, there is a precipitous drop in blood pressure, and the pulse becomes so weak that it is difficult to detect. Life-threatening airway obstruction may ensue as a result of laryngeal edema or bronchial spasm.

Treatment includes immediate discontinuance of the inciting agent or institution of measures to decrease its absorption (*e.g.*, application of ice); close monitoring of cardiovascular and respiratory function; and maintenance of adequate

respiratory gas exchange, cardiac output, and tissue perfusion. Epinephrine constricts the blood vessels and relaxes the smooth muscle in the bronchioles; it usually is the first drug to be given to a patient believed to be experiencing an anaphylactic reaction. Other treatment measures include the administration of oxygen, antihistaminic drugs, and corticosteroids. Resuscitation measures may be required.

The prevention of anaphylactic shock is preferable to treatment. Once a person has been sensitized to an antigen, the risk of repeated anaphylactic reactions with subsequent exposure is high. Persons with known hypersensitivities should carry some form of medical identification to alert medical personnel if they become unconscious or unable to relate this information. Persons who are risk for anaphylaxis should be provided with emergency medications (*e.g.*, epinephrine autoinjector) and instructed in procedures to follow in case they are inadvertently exposed to the offending antigen.

Sepsis and Septic Shock

Septic shock is associated with severe infection and the systemic response to infection. It is associated most frequently with gram-negative bacteremia, although it can be caused by gram-positive bacilli and other microorganisms such as fungi, which carry an even greater risk of mortality.[22] Unlike other types of shock, septic shock commonly is associated with pathologic complications, such as acute respiratory distress syndrome, disseminated intravascular coagulation, and multiple organ dysfunction syndrome.

Septic shock has become the most common type of distributive shock. There are approximately 400,000 to 500,000 septic episodes each year in the United States. The growing incidence has been attributed to an increased awareness of the diagnosis, increased numbers of immunocompromised patients, increased use of invasive procedures, increased number of resistant organisms, and an increased number of elderly patients with critical illnesses.[23] Despite advances in treatment methods, the mortality rate is approximately 40%.[24]

Septic shock has been described in the context of the *systemic inflammatory response*.[25] Although usually associated with infection, the systemic inflammatory response syndrome can be initiated by noninfectious disorders such as acute trauma and pancreatitis.

Mechanisms. The mechanisms of septic shock are thought to be related to mediators of the inflammatory response.[26,27] Although the immune system and the inflammatory response are designed to overcome infection, the dysregulated release of inflammatory mediators such as tumor necrosis factor (TNF) and the interleukins (see Chapter 10) may initiate the potentially fatal sepsis syndrome. These inflammatory mediators, which are released in the presence of bacterial toxins, promote endothelial cell–leukocyte adhesion, release of cell-damaging proteases and prostaglandins, and activation of the blood coagulation cascade. The prostaglandins participate in the generation of fever, tachycardia, ventilation-perfusion abnormalities, and lactic acidosis that occur with septic shock.[28]

In addition to inducing the release of inflammatory mediators, endotoxins may themselves induce tissue damage by directly activating pathways such as the coagulation cascade, the complement cascade, vessel injury, or release of vasodilating prostaglandins.[27] Thus, the sepsis syndrome represents the complex consequences of microbial products that produce profound dysregulation of the inflammatory response.

Manifestations. Septic shock typically manifests with fever, vasodilatation, and a warm, flushed skin. Mild hyperventilation, respiratory alkalosis, and abrupt changes in personality and behavior caused by reduction in cerebral blood flow may be the earliest signs and symptoms of septic shock. These manifestations, which are thought to be a primary response to the bacteremia, commonly precede the usual signs and symptoms of sepsis by several hours or days.

Unlike other forms of shock (*i.e.*, cardiogenic, hypovolemic, and obstructive) that are characterized by a compensatory increase in systemic vascular resistance, septic shock often presents with hypovolemia because of arterial and venous dilatation and leakage of plasma into the interstitial spaces. Aggressive treatment of the hypovolemia in septic shock leads to a decrease in systemic vascular resistance and increased cardiac output and tachycardia. With the development of refined resuscitation methods and better hemodynamic monitoring systems, approximately 90% of patients in septic shock convert to this hyperdynamic response with high cardiac output and low systemic vascular resistance.[27] However, as the condition progresses, cardiac function is depressed; the heart becomes dilated, and the ejection fraction decreases.

Treatment. The treatment of septic shock focuses on the causative agent and support of the circulation. The administration of antibiotics specific to the infectious agent is essential. The cardiovascular status of the patient must be supported to maintain oxygen delivery to the cells. Swift and aggressive fluid administration is needed to compensate for third spacing, and equally aggressive use of vasopressor agents is needed to counteract the vasodilation caused by endotoxins.

Complications of Shock

Wiggers, a noted circulatory physiologist, stated, "Shock not only stops the machine, but it wrecks the machinery."[29] Many body systems are wrecked by severe shock. Some of the major complications of severe shock are acute respiratory distress syndrome, acute renal failure, gastrointestinal ulceration, disseminated intravascular coagulation, and multiple organ dysfunction syndrome.

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is a potentially lethal form of respiratory failure that can follow severe shock (see Chapter 21). The symptoms of ARDS usually do not develop until 24 to 48 hours after the initial trauma; in some instances, they occur much later. The respiratory rate and effort of breathing increase, and arterial blood gas analysis demonstrates the presence of profound hypoxemia with hypercapnia.

The exact cause of ARDS is unknown. Neutrophils are thought to play a key role in the pathogenesis of ARDS. A cytokine-mediated activation and accumulation of neutrophils in the pulmonary vasculature and subsequent endothelial injury is thought to cause leaking of fluid and plasma proteins into the interstitium and alveolar spaces.[30,31] The fluid leakage impairs gas exchange and makes the lung stiffer and more difficult to inflate. Abnormalities in the production, composition, and function of surfactant may contribute to alveolar collapse and gas exchange abnormalities.[31]

Interventions for ARDS focus on increasing the oxygen concentration in the inspired air and supporting ventilation mechanically to optimize gas exchange while avoiding oxygen toxicity and preventing further lung injury.[30,31] Despite the delivery of high levels of oxygen using high-pressure mechanical ventilatory support and positive end-expiratory pressure, many persons with ARDS remain hypoxic, often with a fatal outcome.

Acute Renal Failure

The renal tubules are particularly vulnerable to ischemia, and acute renal failure is one important late cause of death in severe shock. Sepsis and trauma account for most cases of acute renal failure (see Chapter 24). The endotoxins implicated in septic shock are powerful vasoconstrictors that are capable of activating the sympathetic nervous system and causing intravascular clotting. They have been shown to trigger all the separate physiologic mechanisms that contribute to the onset of acute renal failure. The degree of renal damage is related to the severity and duration of shock. The normal kidney is able to tolerate severe ischemia for 15 to 20 minutes. The renal lesion most frequently seen after severe shock is acute tubular necrosis. Acute tubular necrosis usually is reversible, although return to normal renal function may require weeks or months. Continuous monitoring of urine output during shock provides a means of assessing renal blood flow. Frequent monitoring of serum creatinine and blood urea nitrogen levels also provides valuable information regarding renal status.

Gastrointestinal Complications

The gastrointestinal tract is particularly vulnerable to ischemia because of the changes in distribution of blood flow to its mucosal surface. In shock, there is widespread constriction of blood vessels that supply the gastrointestinal tract, causing a redistribution of blood flow that severely diminishes mucosal perfusion. Superficial mucosal lesions of the stomach and duodenum can develop within hours of severe trauma, sepsis, or burn.

Bleeding is a common symptom of gastrointestinal ulceration caused by shock. Hemorrhage has its onset usually within 2 to 10 days after the original insult and often begins without warning. Poor perfusion in the gastrointestinal tract has been credited with allowing intestinal bacteria to enter the bloodstream, thereby contributing to the development of sepsis and shock.

Histamine type 2 receptor antagonists, proton pump inhibitors, or mucosal protective agents may be given prophylactically to prevent gastrointestinal ulcerations caused by shock. Nasogastric tubes, when attached to intermittent suction, also help to diminish the accumulation of hydrogen ions in the stomach.

Disseminated Intravascular Coagulation

Disseminated intravascular coagulation (DIC) is characterized by widespread activation of the coagulation system with resultant formation of fibrin clots and thrombotic occlusion of small and midsize vessels (see Chapter 12). The systemic formation of fibrin results from increased generation of thrombin, the simultaneous suppression of physiologic anticoagulation mechanisms, and the delayed removal of fibrin as a consequence of impaired fibrinolysis. Clinically overt DIC is reported to occur in as many as 30% to 50% of persons with sepsis and septic shock.[32] As with other systemic inflammatory responses, the derangement of coagulation and fibrinolysis is thought to be mediated by inflammatory mediators.

The contribution of DIC to morbidity and mortality in sepsis depends on the underlying clinical condition and the intensity of the coagulation disorder. Depletion of the platelets and coagulation factors increases the risk of bleeding. Deposition of fibrin in the vasculature of organs contributes to ischemic damage and organ failure.

The management of sepsis-induced DIC focuses on treatment of the underlying disorder and measures to interrupt the coagulation process. Anticoagulation therapy and administration of platelets and plasma may be used.

Multiple Organ Dysfunction Syndrome

Multiple organ dysfunction syndrome (MODS) represents the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention. As the name implies, MODS commonly affects multiple organ systems, including the kidneys, lungs, liver, brain, and heart. MODS is a particularly life-threatening complication of shock, especially septic shock. It has been reported as the most frequent cause of death in the noncoronary intensive care unit. Mortality rates vary from 30% to 100%, depending on the number of organs involved.[33] Mortality rates increase with an increased number of organs failing. A high mortality rate is associated with failure of the brain, liver, kidney, and lung. The pathogenesis of MODS is not clearly understood, so current management is primarily supportive. Major risk factors for the development of MODS are sepsis, shock, prolonged periods of hypotension, hepatic dysfunction, trauma, infarcted bowel, advanced age, and alcohol abuse.[33] Interventions for multiple organ failure are focused on support of the affected organs.

In summary, circulatory shock is an acute emergency in which body tissues are deprived of oxygen and cellular nutrients or are unable to use these materials in their metabolic processes. Circulatory shock may develop because there is not enough blood in the circulatory system (*i.e.*, hypovolemic shock), blood flow or venous return is obstructed (*i.e.*, obstructive shock), or there is excessive vasodilation and pooling of blood (*i.e.*, distributive shock). Two types of shock share the circulatory features of distributive shock: neurogenic shock and anaphylactic shock. Septic shock, which shares many of the features of distribution shock, is associated with a severe, overwhelming infection and has a mortality rate of approximately 40%.

The manifestations of circulatory shock are related to low peripheral blood flow and excessive sympathetic stimulation. The low peripheral blood flow produces thirst, changes in skin temperature, a decrease in blood pressure, an increase in heart rate, decreased venous pressure, decreased urine output, and changes in the sensorium. Signs and symptoms, such as changes in skin temperature (*i.e.*, increased in septic shock and decreased in hypovolemic and other forms of shock), may differ with the type of shock. The intense vasoconstriction that serves to maintain blood flow to the heart and brain causes a decrease in tissue perfusion, impaired cellular metabolism, liberation of lactic acid, and, eventually, cell death. Whether the shock will be irreversible or the patient will survive is determined largely by changes that occur at the cellular level.

The complications of shock result from the deprivation of blood flow to vital organs or systems, such as the lungs, kidneys, gastrointestinal tract, and blood coagulation system. ARDS is characterized by changes in the permeability of the alveolar-capillary membrane with the development of interstitial edema and severe hypoxia that does not respond to oxygen therapy. The renal tubules are particularly vulnerable to ischemia, and acute renal failure is an important complication of shock. Gastrointestinal ischemia may lead to gastrointestinal bleeding and increased permeability to the intestinal bacteria, which cause further sepsis and shock. DIC is characterized by formation of small clots in the circulation. MODS, perhaps the most ominous complication of shock, rapidly depletes the body's ability to compensate and recover from a shock state.

HEART FAILURE IN CHILDREN AND THE ELDERLY

Heart Failure in Infants and Children

As in adults, heart failure in infants and children results from the inability of the heart to maintain the cardiac output required to sustain metabolic demands.[34,35] Congenital heart defects are the most common cause of CHF during childhood. Surgical correction of congenital heart defects may cause heart failure as a result of intraoperative manipulation of the heart and resection of heart tissue, with subsequent alterations in pressure, flow, and resistance relations. Usually, the heart failure that results is acute and resolves after the effects of the surgical procedure have subsided. Chronic congestive failure occasionally is observed in children with severe chronic anemia, inflammatory heart disease, end-stage congenital heart disease, or cardiomyopathy. Inflammatory heart disorders (*e.g.*, myocarditis, rheumatic fever, bacterial endocarditis, Kawasaki's disease), cardiomyopathy, and congenital heart disorders are discussed in Chapter 17.

Manifestations

Many of the signs and symptoms of heart failure in infants and children are similar to those in adults. They include fatigue, effort intolerance, cough, anorexia, and abdominal pain. A subtle sign of cardiorespiratory distress in infants and children is a change in disposition or responsiveness, including irritability or lethargy. Sympathetic stimulation produces peripheral vasoconstriction and diaphoresis. Decreased renal blood flow often results in a urine output of less than 0.5 to 1.0 mL/kg/hour, despite adequate fluid intake. When right ventricular function is impaired, systemic venous congestion develops. Hepatomegaly caused by liver congestion often is one of the first signs of systemic venous congestion in infants and children. However, dependent edema or ascites rarely is seen unless the CVP is extremely high. Because of their short, fat necks, jugular venous distention is difficult to detect in infants; it is not a reliable sign until the child is of school age or older.

Most commonly, children experience interstitial edema, rather than alveolar pulmonary edema. This reduces lung compliance and increases the work of breathing, causing tachypnea and increased respiratory effort. Older children display use of accessory muscles (*i.e.*, scapular and sternocleidomastoid). Head bobbing and nasal flaring may be observed in infants. Signs of respiratory distress often are the first and most noticeable indication of heart failure in infants and young children. Pulmonary congestion may be mistaken for bronchiolitis or lower respiratory tract infection. The infant or young child with respiratory distress often grunts with expiration. This grunting effort (essentially, exhaling against a closed glottis) is an instinctive effort to increase end-expiratory pressures and prevent collapse of small airways and the development of atelectasis. Respiratory crackles are uncommon in infants and usually suggest development of a respiratory tract infection. Wheezes may be heard, particularly if there is a large left-to-right shunt.

Infants with heart failure often have increased respiratory problems during feeding.[34] The history is one of prolonged feeding with excessive respiratory effort and fatigue. Weight gain is slow because of high energy requirements and low calorie intake. Other frequently occurring manifestations of heart failure in infants are excessive sweating (caused by increased sympathetic tone), particularly over the head and neck, and repeated lower respiratory tract infections. Peripheral perfusion usually is poor, with cool extremities; tachycardia is common (resting heart rate >150 beats per minute); and respiratory rate is increased (resting rate >50 breaths per minute).

Diagnosis and Treatment

Diagnosis of congestive failure in infants and children is based on symptomatology, chest radiographic films, ECG findings, echocardiographic techniques to assess cardiac structures and ventricular function (*i.e.*, end-systolic and end-diastolic diameters), arterial blood gases to determine intracardiac shunting and ventilation-perfusion inequalities, and other laboratory studies to determine anemia and electrolyte imbalances.

Treatment of congestive failure in infants and children includes measures aimed at improving cardiac function and eliminating excess intravascular fluid. Oxygen delivery must be supported and oxygen demands controlled or minimized. When possible, the cause of the disorder is corrected (*e.g.*, medical treatment of sepsis and anemia, surgical correction of congenital heart defects). With congenital anomalies that are amenable to surgery, medical treatment often is needed for a time before surgery and usually is continued during the immediate postoperative period. For many children, only medical management can be provided.

Medical management of heart failure in infants and children is similar to that in the adult, although it is tailored to the special developmental needs of the child. Inotropic agents such as digitalis often are used to increase cardiac contractility. Diuretics may be given to reduce preload and vasodilating drugs used to manipulate the afterload. Drug doses must be carefully tailored to control for the child's weight and conditions such as reduced renal function. Daily weighing and accurate measurement of intake and output are imperative during acute episodes of failure.

Most children feel better in the semiupright position. An infant seat is useful for infants with chronic CHF. Activity restrictions usually are designed to allow children to be as active as possible within the limitations of their heart disease. Infants with congestive failure often have problems feeding. Small, frequent feedings usually are more successful than larger, less fre-

quent feedings. Severely ill infants may lack sufficient strength to suck and may need to be tube fed.

Heart Failure in the Elderly

Heart failure is one of the most common causes of disability in the elderly and is the most common hospital discharge diagnosis for the elderly. More than 75% of patients with heart failure are older than 65 years.[36] Among the factors that have contributed to the increased numbers of older people with heart failure are the improved therapies for ischemic and hypertensive heart disease. Thus, persons who would have died of acute myocardial disease 20 years ago are now surviving, but with residual left ventricular dysfunction. Similarly, improved blood pressure control has led to a 60% decline in stroke mortality rates, yet these same people remain at risk for CHF as a complication of hypertension. In addition, advances in treatment of other diseases have contributed indirectly to the rising prevalence of heart failure in the older population.

Coronary heart disease, hypertension, and valvular heart disease (particularly aortic stenosis and mitral regurgitation) are common causes of heart failure in older adults.[36,37] Although the pathophysiology of heart failure is similar in younger and older persons, elderly persons tend to experience cardiac failure when confronted with stresses that would not produce failure in younger persons. There are four principal changes associated with cardiovascular aging that impair the ability to respond to stress.[36] First, reduced responsiveness to β-adrenergic stimulation limits the heart's capacity maximally to increase heart rate and contractility. A second major effect of aging is increased vascular stiffness, which results in an increased resistance to left ventricular ejection (afterload) and contributes to the development of systolic hypertension in the elderly. Third, in addition to increased vascular stiffness, the heart itself becomes stiffer and less compliant with age. The changes in diastolic stiffness result in important alterations in diastolic filling and atrial function. A reduction in ventricular filling not only affects cardiac output, but also produces an elevation in diastolic pressure that is transmitted back to the left atrium, where it stretches the muscle wall and predisposes to atrial ectopic beats and atrial fibrillation. The fourth major effect of cardiovascular aging is altered myocardial metabolism at the level of the mitochondria. Although older mitochondria may be able to generate sufficient adenosine triphosphate to meet the normal energy needs of the heart, they may not be able to respond under stress.

Manifestations

The manifestations of heart failure in the elderly often are masked by other disease conditions. Nocturia is an early symptom but may be caused by other conditions such as prostatic hypertrophy. Dyspnea on exertion may result from lung disease, lack of exercise, and deconditioning. Lower extremity edema commonly is caused by venous insufficiency.

Among the acute manifestations of heart failure in the elderly are increasing lethargy and confusion, probably the result of impaired cerebral perfusion. Activity intolerance is common. Instead of dyspnea, the prominent sign may be restlessness. Impaired perfusion of the gastrointestinal tract is a common cause of anorexia and profound loss of lean body mass. Loss of lean body mass may be masked by edema.

The elderly also maintain a precarious balance between the managed symptom state and acute symptom exacerbation. During the managed symptom state, they are relatively symptom free while adhering to their treatment regimen. Acute symptom exacerbation, often requiring emergency medical treatment, can be precipitated by seemingly minor conditions such as poor compliance with sodium restriction, infection, or stress. Failure promptly to seek medical care is a common cause of progressive acceleration of symptoms.

Diagnosis and Treatment

The diagnosis of heart failure in the elderly is based on the history, physical examination, chest radiograph, and ECG findings. However, the presenting symptoms of heart failure often are difficult to evaluate.

Treatment of heart failure in the elderly involves many of the same methods as in younger persons. Activities are restricted to a level that is commensurate with the cardiac reserve. Seldom is bed rest recommended or advised. Bed rest causes rapid deconditioning of skeletal muscles and increases the risk of complications, such as orthostatic hypotension and thromboemboli. Instead, carefully prescribed exercise programs can help to maintain activity tolerance. Even walking around a room usually is preferable to continuous bed rest. Sodium restriction usually is indicated.

Age- and disease-related changes increase the likelihood of adverse drug reactions and drug interactions. Drug dosages and the number of drugs that are prescribed should be kept to a minimum. Compliance with drug regimens often is difficult; the simpler the regimen, the more likely it is that the older person will comply. In general, the treatment plan for elderly persons with heart failure must be put in the context of the person's overall needs. An improvement in the quality of life may take precedence over increasing the length of survival.

In summary, the mechanisms of heart failure in children and the elderly are similar to those in adults. However, the causes and manifestations may differ because of age. In children, CHF is seen most commonly during infancy and immediately after heart surgery. It can be caused by congenital and acquired heart defects and is characterized by fatigue, effort intolerance, cough, anorexia, abdominal pain, and impaired growth. Treatment of CHF in children includes correction of the underlying cause when possible. For congenital anomalies that are amenable to surgery, medical treatment often is needed for a time before surgery and usually is continued in the immediate postoperative period. For many children, only medical management can be provided.

In the elderly, age-related changes in cardiovascular functioning contribute to CHF but are not in themselves sufficient to cause heart failure. The manifestations of congestive failure often are different and superimposed on other disease conditions; therefore, CHF often is more difficult to diagnose in the elderly than in younger persons. Because the elderly are more susceptible to adverse drug reactions and have more problems with compliance, the number of drugs that are prescribed is kept to a minimum, and the drug regimen is kept as simple as possible.

REVIEW QUESTIONS

- Explain the effect of decreased cardiac reserve on symptom development in heart failure.
- Explain how increased sympathetic activity, fluid retention, the Frank-Starling mechanism, and myocardial hypertrophy function as compensatory mechanisms in heart failure.
- Differentiate high-output versus low-output heart failure, systolic versus diastolic heart failure, and right-sided versus left-sided heart failure.
- Relate the effect of left ventricular failure to the development of and manifestations of pulmonary edema.
- Describe the pathophysiology of cardiogenic shock.
- Describe the compensatory mechanisms that are activated in circulatory shock.
- List the chief characteristics of hypovolemic shock, cardiogenic shock, obstructive shock, and distributive shock.
- Characterize changes in thirst, skin blood flow, pulse rate, urine output, and sensorium that are indicative of shock.
- Describe the complications of shock as they relate to the lung, kidney, gastrointestinal tract, and blood clotting.
- Define multiple organ dysfunction syndrome and cite its significance in shock.
- Describe the manifestations of heart failure in infants and children.
- Explain how the aging process affects heart failure in the elderly.

connection

Visit the Connection site at connection.lww.com/go/porth for links to chapter-related resources on the Internet.

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